# 9 Unique Aspects of Dairy Cattle Nutrition

## TRANSITION COWS AND NONLACTATING COWS

### *Nutritional and Physiologic Status of the Transition Cow*

Fetal dry weight increases exponentially during gestation (House and Bell, 1993; National Research Council, 1996). Bell et al. (1995) indicated linear or nonlinear regression models were more suitable than exponential models for describing increases in fetal dry weight, fresh weight, and crude protein (CP) and energy accretion during the final trimester of pregnancy. They speculated that exponential models might be more appropriate when describing fetal growth for the entire gestation (i.e., including periods when fetal size is very small). Because conceptus growth approaches linearity during the final stages of gestation, exponential models developed from data obtained throughout pregnancy may overestimate growth during late gestation. Fetal sex does not influence growth rates (Ferrell et al., 1982; House and Bell, 1993). Fetal tissue accounts for 45 percent of the uterine dry weight at day 190 of pregnancy and 80 percent at day 270 of pregnancy (Bell et al., 1995).

The dry period, in particular the transition period, is characterized by dramatic changes in endocrine status. These changes prepare the cow for parturition and lactogenesis. Plasma insulin decreases and growth hormone increases as the cow progresses from late gestation to early lactation, with acute surges in plasma concentrations of both hormones at parturition (Kunz et al., 1985). Plasma thyroxine (T4) concentrations gradually increase during late gestation, decrease approximately 50 percent at calving, and then begin to increase (Kunz et al., 1985). Similar but less pronounced changes occur with 3, 5, 3′-triiodothyronine (T3). Estrogen, primarily estrone of placental origin, increases in plasma during late gestation but decreases immediately at calving (Chew et al., 1979). Progesterone concentrations during the dry period are elevated for maintenance of pregnancy but decline rapidly, approximately 2 days before calving (Chew et al., 1979). Glucocorticoid and prolactin concentrations increase on the day of calving and return to near prepartum concentrations the following day (Edgerton and Hafs, 1973).

Changes in endocrine status and decreases in dry matter intake (DMI) during late gestation influence metabolism and lead to mobilization of fat from adipose tissue and glycogen from the liver. Plasma nonesterified fatty acids (NEFA) increase two-fold or more between 2 to 3 weeks prepartum and 2 to 3 days prepartum, at which time the concentration increases dramatically until completion of parturition (Bertics et al., 1992; Vazquez-Anon, 1994; Grum et al., 1996). How much of the initial increase in plasma NEFA can be accounted for by changing endocrine status compared with energy restriction resulting from decreased DMI is not known. Force feeding cows during the prefresh transition period reduced the magnitude of NEFA increase, but did not completely eliminate it (Bertics et al., 1992). These observations indicate at least part of the prepartum increase in plasma NEFA is hormonally induced. The rapid rise in NEFA on the day of calving is presumably due to the stress of calving. Plasma NEFA concentrations decrease rapidly after calving, but concentrations remain higher than they were before calving.

Plasma glucose concentrations remain stable or increase slightly during the prefresh transition period, increase dramatically at calving, and then decrease immediately postpartum (Kunz et al., 1985; Vazquez-Anon et al., 1994). The transient increase at calving may result from increased glucagon and glucocorticoid concentrations that promote depletion of hepatic glycogen stores. Although the demand for glucose by mammary tissue for lactose synthesis continues after calving, hepatic glycogen stores begin to replete and are increased by day 14 postpartum (Vazquez-Anon et al., 1994). This probably reflects an increase in gluconeogenic capacity to support lactation.

Blood calcium decreases during the last few days prior to calving due to the loss of calcium for the formation of colostrum (Goff and Horst, 1997b). Plasma Ca concentrations are controlled by the coordinated actions of parathyroid hormone and 1, 25-dihydroxyvitamin $D_3$. These hormones act on the intestine, kidney, and bone to increase blood calcium during the periparturient period. Adaptation

of the intestine, kidney, and bone to higher demands for calcium takes several days so that blood calcium typically does not return to normal concentrations until several days postpartum (Goff and Horst, 1997b).

As cows initiate and terminate the dry period, there are changes in rumen dynamics. These alterations are nutritionally induced rather than physiologically induced. Changing from a diet that is high in concentrate to a diet that is high in fiber causes alterations in the microbial population and characteristics of the rumen epithelium. High concentrate diets favor starch utilizing bacteria that enhance propionate and lactate production; high fiber diets favor cellulolytic bacteria and methane production and discriminate against bacteria that produce propionate and utilize lactate. End products of fermentation influence papillae growth in the rumen (Dirksen et al., 1985). Papillae are responsible for the absorption of volatile fatty acids. Increasing grain in the diet and propionate concentration in the rumen favors elongation of papillae; diets high in fiber cause the papillae to shorten. As much as 50 percent of the absorptive area in the rumen may be lost during the first 7 weeks of the dry period and elongation of papillae after reintroduction of concentrate takes several weeks (Dirksen et al., 1985). Consequently, sudden introduction of grain immediately postcalving has several deleterious consequences. Lactate production increases prior to the re-establishment of lactate utilizing bacteria. Lactate is more potent in reducing ruminal pH than other volatile fatty acids and volatile fatty acids are absorbed at a faster rate when pH is low (Goff and Horst, 1997b). Rumen papillae will not have had sufficient time to elongate, therefore, volatile fatty acid absorption is limited.

During the transition period, the immunologic status of the cow is compromised. Neutrophil and lymphocyte function is depressed and plasma concentrations of other components of the immune system are decreased (Goff and Horst, 1997b). It is not known why immune function is suppressed but it may be related to the nutritional and physiologic status of the cow. Estrogen and glucocorticoids are immunosuppressive agents and they increase in plasma as parturition approaches (Goff and Horst, 1997b). Intake of vitamin A and E and other nutrients essential for immune function may be decreased as DMI is reduced during the periparturient period.

### *Nutrient Requirements for Pregnancy*

Dry cows require nutrients for maintenance, growth of the conceptus, and perhaps growth of the dam. Estimation of the nutrient requirements for pregnancy by the factorial method requires knowledge of the rates of nutrient accretion in conceptus tissues (fetus, placenta, fetal fluids, and uterus) and the efficiency with which dietary nutrients are utilized for conceptus growth. There are limited data for dairy cattle.

Estimates of CP, energy, and most mineral requirements for gestation during the last two months of pregnancy are from House and Bell (1993) and Bell et al. (1995). Rates of growth and chemical composition were measured in multiparous Holstein cows that were serially slaughtered from 190 to 270 days of pregnancy. Requirements derived from these studies and equations used for the model are discussed in chapters 2 (energy), 5 (protein), and 6 (minerals).

Other estimates for energy and crude protein requirements are available, but they were obtained from beef cattle, dairy breeds other than Holsteins, or from research conducted more than 25 years ago. However, estimates from Bell et al. (1995) do not vary greatly from previous estimates and thus are supportive of requirements published in *Nutrient Requirements of Dairy Cattle* (National Research Council, 1989). Additionally, by using the data from Bell et al. (1995) energy, protein, and mineral requirements for pregnancy were all derived from the same study.

A quadratic regression equation best described protein and energy accretion in the gravid uterus. Estimates of CP and energy requirements to support pregnancy were derived from cows with a mean body weight of 714 kg that carried a single fetus. They are a function of day of gestation, but an adjustment to accommodate differences in calf birth weight was added to the equations derived from Bell et al. (1995). Crude protein requirements for gestation were obtained by assuming an efficiency of 0.33 for conversion of metabolizable protein (MP) to conceptus protein and efficiency of 0.7 for conversion of dietary CP to MP (Bell et al., 1995). The efficiency of conversion of MP to conceptus protein has been reduced from 0.5 used in the previous edition (National Research Council, 1989). The efficiency of conversion of metabolizable energy to conceptus net energy (NE) was assumed to be 0.14 (Ferrell et al., 1976). The low efficiency most likely reflects the high cost of maintaining the fetus.

### *Nutrient Intake*

Intake of nutrients is a function of DMI and nutrient density of the diet. Dry matter intake during the final 21 days of gestation was described (Hayirli et al., 1998) by an exponential function: $y = a + p \times e^{k \times t}$ where y = DMI as a percentage of body weight, a = the asymptotic intercept at time = $-\infty$ (minus infinity), p = the magnitude of intake depression (kg) from the asymptotic intercept until parturition, and $e^{k \times t}$ describes the shape of the curve. Time (t) is expressed as: days pregnant − 280. Following evaluation of the model (mean square predicted error = 0.06 percent $BW^2$, mean bias = 0.01 percent BW when plotting mean daily observed DMI versus mean daily pre-

dicted DMI), the original data set and the data set used for evaluation were combined to generate the following prediction equations for DMI during the final 21 days of gestation:

$$\text{Heifers: DMI (\% of BW)} = 1.71 - 0.69e^{0.35t} \quad (9\text{-}1)$$

$$\text{Cows: DMI (\% of BW)} = 1.97 - 0.75e^{0.16t} \quad (9\text{-}2)$$

These equations were from a data set that included 172 heifers and 527 cows used in 16 experiment treatments that were conducted at 8 universities and involved 49 treatments.

Factors that influence prepartum DMI are not well established. Zamet et al. (1979a) reported lower prepartum DMI for cows diagnosed with fat cow syndrome compared with "normal" cows that did not have postpartum complications. Hayirli et al. (1998) indicated that over conditioned cows experience a gradual decline in DMI during the prefresh transition period whereas thin cows maintain DMI longer prior to experiencing a more abrupt decrease in DMI shortly before calving. However, a relationship between body condition and prepartum DMI does not imply cause and effect. Categorization of cows on the basis of body condition may also categorize cows into groups that have many genetic, physiologic, and biochemical differences.

Ration composition and nutrient content may influence prepartum DMI. Increasing energy (Coppock et al., 1972; Hernandez-Urdaneta et al., 1976; Minor et al., 1998) or energy and protein (VandeHaar et al., 1999) content of the diet during the prefresh transition period resulted in higher dry matter (DM) and energy intake. In contrast, replacement heifers fed 35 percent concentrate during the final 5 months before first calving had lower DMI (but similar energy intake) during the final 10 days prepartum than did cows fed 6 percent concentrate during the same period (Grummer et al., 1995).

The blood concentrations of many hormones increase or decrease dramatically at parturition and may be potent modifiers of DMI. For example, plasma estrogen of placental origin (specifically estrone) increases in blood as parturition approaches. Exogenous estrogen administration inhibits DMI (Grummer et al., 1990). Reduced DMI during estrus and late pregnancy may reflect greater endogenous estrogen production.

Development of metabolic disorders during the transition period may cause a reduction in DMI. Cows with hypocalcemia have lower prepartum DMI (Goff and Horst, 1997b). Hypocalcemia may cause loss of muscle tone that could adversely affect rumen function, intestinal peristalsis, and passage rate of digesta. Slower passage rates may have a negative effect on DMI.

### *Energy and Protein Density for Dry Cow Diets*

Table 6-5 of *Nutrient Requirements of Dairy Cattle* (National Research Council, 1989) listed one set of nutrient density recommendations for dry, pregnant cows. In the current edition, separate nutrient density guidelines have been developed for far-off dry cows and prefresh transition cows (Chapter 14). This gives greater recognition to DMI depression prior to calving and the unique physiologic and nutritional changes that are associated with late pregnancy, parturition, and lactogenesis. Formulation of a unique diet for prefresh transition cows should reduce the risk of metabolic disorders during early lactation and improve lactation performance.

Nutrient density guidelines for dairy cattle can be obtained by dividing nutrient requirements as determined by the factorial method by predicted DMI. While this approach is appropriate for most classes of cattle, it is problematic for prefresh transition dairy cows because DMI and nutrient requirements are changing relatively rapidly during late gestation. Clearly it is not practical to constantly reformulate diets on a daily basis as cows progress through the prefresh transition period. Additionally, animal physiology at parturition, microbial ecology of the rumen, and pharmacologic effects of nutrients also must be considered when deriving nutrient density recommendations for transition cows. Unique considerations for feeding protein and energy are described below; discussion of adjustments for other nutrients can be found in appropriate sections within this chapter (e.g., selenium/retained placenta, calcium/milk fever).

#### PROTEIN

Results obtained by dividing CP requirements for maintenance, growth (heifers only), and gestation (Bell et al., 1995; data in this edition) by predicted DMI are shown by the solid lines in Figure 9-1. Using this approach, it appears that CP content could be 12 percent or slightly less for mature cows during all but the last few days of the dry period. The previous edition established a minimum of 12 percent CP for diets of dry cows (National Research Council, 1989). Justification for establishing a minimum of 12 percent was absent. Presumably this was based on a minimum amount of CP believed to be necessary to optimize some aspect of ruminal function (e.g., microbial protein synthesis or fiber digestion) (Sahlu et al., 1995). In this revision, it has been established that prefresh transition diets should not be formulated to contain less than 12 percent CP. Feeding a diet containing 12 percent CP provides a margin of safety in the event that DMI would be lower for low protein diets. Chew et al. (1984) fed approximately 9 or 11 percent CP during the entire dry period and observed higher prepartum DMI and higher milk yields when feeding the higher protein diet. Feeding diets with 12 percent CP at predicted DMI is insufficient to meet protein requirements for heifers during the transition period (Figure 9-1). Heifers have lower DMI as a

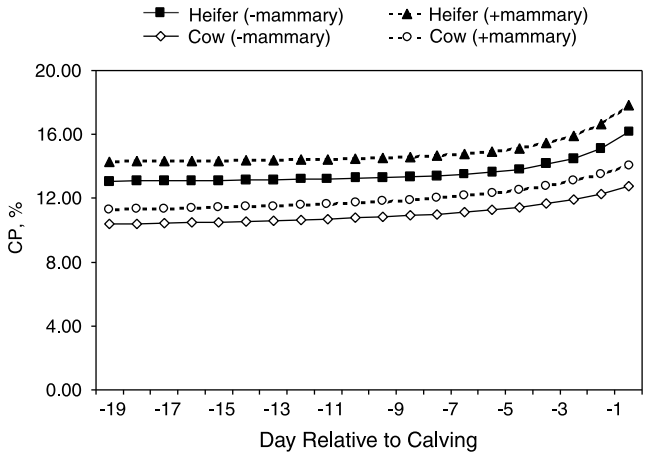


FIGURE 9-1 Dietary concentrations of crude protein needed in diets fed to transition cows to meet requirements. Values were calculated assuming dry matter intakes as predicted by the exponential model described in the text. Solid lines represent calculations using estimates of CP requirements for maintenance, growth, and gestation (from this edition) for a 740 kg mature cow or a 615 kg replacement heifer. Dotted lines represent calculations using estimates of requirements for maintenance, growth, gestation (from this edition), and mammary growth (130 g/d, see text). Body condition score = 3.3, calf birth weight = 45 kg, and heifer growth rate = 300 g/d (without conceptus). Diet consisted of 35 percent corn silage, normal; 34 percent grass silage, C-3, mid-maturity; 10 percent corn grain, ground high moisture; 8 percent soybean meal, solvent, 48 percent CP and 13 percent beet, sugar pulp.

percentage of body weight and have additional nutrient requirements for growth. A preliminary report (Santos et al., 1999a,b) indicated that primiparous but not multiparous cows have improved lactation performance when the CP in prepartum diets is increased from 12.7 to 14.7 percent by the addition of animal proteins.

Crude protein requirements for mammary growth were not included in the model. Insufficient data for mammary parenchymal growth rates, mammary composition, and efficiency of conversion of MP to net protein during late gestation were available to accurately predict requirements for mammary growth. However, as outlined by VandeHaar and Donkin (1999), if mammary parenchymal mass increases by 460 g/d during the transition period (Capuco et al., 1997), mammary parenchymal tissue is 10 percent crude protein (Ferrell et al., 1976; Swanson and Poffenbarger, 1979), and efficiencies of conversion of dietary CP to MP and MP to tissue net protein are 0.7 and 0.5 (National Research Council, 1996), then additional CP for mammary growth would be approximately 130 g/d. This would increase the dietary CP needed to meet requirements by approximately one percentage unit (dotted lines, Figure 9-1). Additional research is needed to determine protein and amino acid requirements for mammary growth.

Several research trials have been conducted to examine the effects of dietary CP during the prefresh transition period on health and productivity of postpartum dairy cows. Increasing dietary CP beyond 12 percent during the dry period by addition of feeds that are high in ruminally undegradable protein improved reproductive performance of first lactation cows (Van Saun et al., 1993) and reduced the incidence of ketosis in multiparous cows (Van Saun and Sniffen, 1995). Increasing dietary CP by 2 to 4 percentage units above 12 to 13 percent CP during the prefresh transition period has reduced postpartum feed intake (Crawley and Kilmer, 1995; Van Saun et al., 1995; Greenfield et al., 1998; Hartwell et al., 1999; Putnam et al., 1999) or milk yield (Crawley and Kilmer, 1995; Greenfield et al., 1988). Most studies have shown that milk yield is not influenced by protein content of prepartum diets (Van Saun et al., 1993, Van Saun and Sniffen, 1995; Wu et al., 1997; Putnam and Varga, 1998; Huyler et al., 1999; Putnam et al., 1999; VandeHaar et al., 1999). Although not observed in the majority of studies, milk protein yield (Moorby et al., 1996) and percentage (Van Saun et al., 1993; Moorby et al., 1996) have been increased when feeding additional ruminally undegradable protein prepartum. Cows fed diets containing 10.5, 12.6, or 14.5 percent CP were all in positive nitrogen balance during the prefresh transition period and had similar lactation performance when fed identical diets postpartum (Putnam and Varga, 1998). Strategic supplementation of limiting amino acids may prove to be more successful than increasing total CP or ruminally undegradable protein; however, amino acid requirements for pregnancy have not been defined. A preliminary report (Chalupa et al., 1999) did not indicate a benefit of feeding ruminally protected amino acids during the prefresh transition period; milk and protein yields were increased when supplementation occurred during the postpartum or prepartum and postpartum period.

Although some positive results have been noted when increasing CP beyond 12 percent by feeding additional ruminally undegradable protein, the results have been inconsistent and sometimes negative (e.g., reduced feed intake). The capacity of the cow to detoxify ammonia may be limited during the periparturient period (Strang et al., 1998). Feeding excess protein may be detrimental to the environment. At this time, there is insufficient evidence to support feeding diets with more than 12 percent CP to mature cows during the prefresh transition period. Therefore, the recommendation of 12 percent CP for dry cow diets that was made in the last edition (National Research Council, 1989) has been retained for mature cows (Table 14-11). Heifers may benefit from feeding higher amounts of CP. According to Figure 9-1, average CP density needed in prefresh transition diets to meet requirements at predicted feed intakes would be 14.2 percent if an adjustment is made for mammary growth. Therefore, it is recom-

mended that heifers be fed diets containing 15 percent CP during the prefresh transition period (Table 14-10). Further research is required to more clearly define protein and amino acid requirements during the prefresh transition period.

### ENERGY

The recommended energy density for diets fed to dry cows was 1.25 Mcal $NE_L$/kg DM in Table 6-5 of *Nutrient Requirements of Dairy Cattle* (National Research Council, 1989). Assuming DMI as predicted above, 1.25 Mcal $NE_L$/kg DM appears adequate for meeting the energy requirements of cows during the far-off dry period but becomes inadequate during the final one to two weeks of the prefresh transition period depending on whether an adjustment has been made for mammary growth (Figure 9-2). Heifers have lower DMI and additional energy requirements for growth, therefore, 1.25 Mcal $NE_L$/kg DM is inadequate during the entire prefresh transition period.

The recommendation for energy density in diets fed to prefresh transition cows and heifers is 1.62 Mcal $NE_L$/kg DM (Tables 14-10, 14-11). At predicted dry matter intakes, 1.62 Mcal $NE_L$/kg DM will not provide sufficient energy to meet requirements of heifers during a significant portion of the prefresh transition period and possibly of mature cows during the final few days prior to calving. However, it is recommended not to feed diets with greater than 1.62 Mcal $NE_L$/kg DM (Tables 14-10, 14-11) because feeding more energy dense diets may increase intake of rapidly fermentable carbohydrate too quickly and adversely affect ruminal fermentation and DMI. Feeding diets with 1.62 Mcal $NE_L$/kg DM will probably provide more energy than required for maintenance and gestation for the majority of the prefresh transition period for cows in the 2nd or greater gestation. However, there are several reasons why feeding diets that high in energy could be beneficial. Increasing energy density by increasing nonfiber carbohydrate will allow ruminal microorganisms to adapt to the high concentrate diets that will be fed postpartum. Greater volatile fatty acid production in the rumen will stimulate papillae growth and increase the capacity for acid to be absorbed from the rumen when additional grain is fed postpartum (Dirksen et al., 1985). Increased propionate formation may trigger an insulin response, which can act to reduce fatty acid mobilization from adipose tissue and lipid-related metabolic disorders (Grummer, 1993; Grummer, 1995). Finally, energy requirements for mammary growth have not been described and were not considered when determining total energy requirements for prefresh transition cows. Feeding diets with 1.62 Mcal $NE_L$/kg DM would probably accommodate energy requirements for maintenance, pregnancy, and mammary growth in mature cows (VandeHaar et al., 1999) except for the final few days prior to calving.

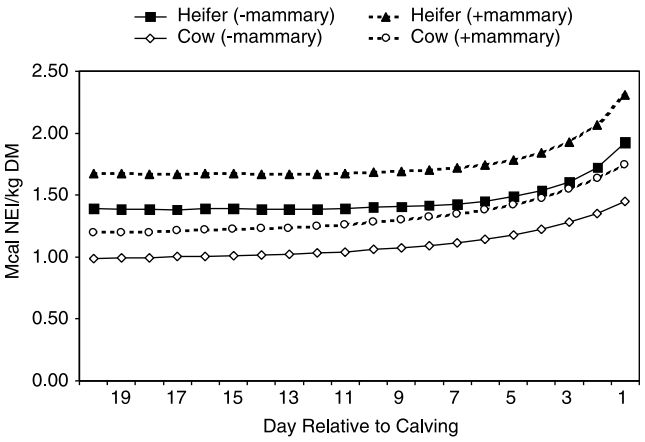


FIGURE 9-2 Dietary concentrations of $NE_L$ needed in diets fed to transition cows to meet requirements. Values were calculated assuming dry matter intakes as predicted by the exponential model described in the text. Solid lines represent calculations using estimates of $NE_L$ requirements for maintenance, growth, and gestation (from this edition) for a 740 kg mature cow or a 615 kg replacement heifer. Dotted lines represent calculations using estimates of requirements for maintenance, growth, gestation (from this edition), and mammary growth (3Mcal/d, VandeHaar et al., 1999). Body condition score = 3.3, calf birth weight = 45 kg, and heifer growth rate = 300 g/d (without conceptus). Diet consisted of 35 percent corn silage, normal; 34 percent grass silage, C-3, mid-maturity; 10 percent corn grain, ground high moisture; 8 percent soybean meal, solvent, 48 percent CP and 13 percent beet, sugar pulp.

## ETIOLOGY AND NUTRITIONAL PREVENTION OF METABOLIC DISORDERS

### *Fatty Liver and Ketosis*

Fatty liver and ketosis are most likely to occur during periods when blood NEFA concentrations are elevated. The most dramatic elevation occurs at calving when plasma concentrations often exceed 1000 μeq/L (Bertics et al., 1992; Vazquez-Anon et al., 1994; Grum et al., 1996). Uptake of NEFA by liver is proportional to NEFA concentrations in blood (Bell, 1979). Extensive reviews on regulation of hepatic lipid metabolism and its relation to fatty liver and ketosis have been published recently (Emery et al., 1992; Grummer, 1993; Bauchart et al., 1996; Drackley, 1999; Hocquette and Bauchart, 1999) and will not be detailed here. Briefly, nonesterified fatty acids taken up by the liver can either be esterified or oxidized in the mitochondria or peroxisomes (Drackley, 1999). The primary esterification product is triglyceride. Triglyceride can either be exported as part of a very low density lipoprotein

or be stored. In ruminants, export of triglyceride occurs at a very slow rate relative to many other species (Kleppe et al., 1988; Pullen et al., 1990). Therefore, under conditions of elevated hepatic NEFA uptake (e.g., low blood glucose and insulin) fatty acid esterification and triglyceride accumulation occurs. The cause for the slow rate of triglyceride export from the liver of ruminants is not known. Complete oxidation of NEFA leads to the formation of $CO_2$; incomplete oxidation yields ketones, primarily acetoacetate and beta-hydroxybutyrate. Ketone formation is also favored when blood glucose and insulin concentrations are low, partially because of greater fatty acid mobilization from adipose tissue. Low insulin probably enhances fatty acid oxidation by decreasing hepatocyte malonyl-CoA concentrations and sensitivity of carnitine palmitoyltransferase-1 to malonyl-CoA (Emery et al., 1992). Carnitine palmitoyltransferase-1 is responsible for translocating fatty acids from the cytosol to the mitochondria for oxidation and is inhibited by malonyl-CoA. Propionate is antiketogenic. The antiketogenic properties of propionate are likely due to indirect effects as an insulin secretegogue as well as direct effects on hepatic metabolism (Grummer, 1993). Ketonemia is common at calving during the sudden surge in NEFA, when energy requirements for milk production far exceed energy intake, and as a secondary disorder to others that may cause DMI depression and elevated NEFA.

Elevated liver triglyceride concentration is common in cows immediately after parturition suggesting that measures to prevent fatty liver take place during the prefresh transition period (Grummer, 1993). Fatty liver can be a secondary complication to any disorder that causes a cow to experience negative energy balance. Because of the slow rate of triglyceride export as lipoprotein, once fatty liver has developed, it will persist for an extended period of time. Depletion usually commences when the cow reaches positive energy balance and may take several weeks until completion. Ketosis usually occurs 2 to 4 weeks postpartum; reasons for the lag period between fatty liver and ketosis are not known. However, cows with elevated liver triglyceride and depressed glycogen are most susceptible to ketosis, and fatty liver preceded ketosis when ketosis was experimentally induced (Veenhuizen et al., 1991). Fatty acid oxidation and ketogenesis are likely the major routes of depletion of excess fat from the liver. Ketones may inhibit fatty acid mobilization from adipose tissue and ultimately reduce hepatic fatty acid uptake and triglyceride accumulation (Emery et al., 1992).

Reducing severity and duration of negative energy balance is crucial in the prevention of fatty liver and ketosis. The critical time for the prevention of fatty liver is approximately one week prior to calving through one week after parturition (Grummer, 1993). This is when the cow is most susceptible to development of fatty liver, which is an indicator of ketosis. Maximizing DMI during the week prior to and after calving may be achieved by avoiding overconditioned cattle, rapid diet changes, unpalatable feeds, periparturient diseases, and environmental stress. Effects of body condition score on health and productivity are variable; however, extremely thin or overconditioned cows should be avoided. Thin cows (body condition score $\leq 3$) can be fed additional energy during the dry period to replenish condition without causing fatty liver because the liver is not a lipid depot during positive energy balance. Over conditioned cattle (body condition score $\geq 4$) should not be feed restricted as this will promote fat mobilization from adipose tissue and elevate blood NEFA and liver triglyceride.

Compounds to decrease fatty acid mobilization from adipose tissue or increase lipoprotein export from the liver have been suggested for prevention of fatty liver and ketosis. Feeding 3 to 12 g niacin per day may reduce blood ketones (Dufva et al., 1983) but a beneficial effect on liver triglyceride concentration has not been observed (Skaar et al., 1989; Minor et al., 1998). Glucose or compounds that can be converted to glucose may decrease blood ketones following intravenous administration (Hamada et al., 1982). The response is presumably mediated via insulin, which suppresses fatty acid mobilization from adipose tissue. Propylene glycol is a glucose precursor that can be given as an oral drench to reduce blood nonesterified fatty acids and the severity of fatty liver at calving (Studer et al., 1993) or blood ketones postcalving (Sauer et al., 1973). Salts of propionic acid are also a glucose precursor and may be effective in lowering blood ketones when fed (Schultz, 1958). There is insufficient evidence to support the use of compounds that are known to be lipotropic agents in nonruminants (e.g., choline, inositol, and methionine) to prevent or treat fatty liver or ketosis (Grummer, 1993).

### *Udder Edema*

Udder edema is a periparturient disorder characterized by excessive accumulation of fluids in the intercellular tissue spaces of the mammary gland. In severe cases, edema and congestion occur in the udder and umbilical area, and may be prominent in the vulva and brisket. Typically the incidence and severity are greater in pregnant heifers than in cows (Zamet et al., 1979; Erb and Grohn, 1988), and tend to be more severe in older than younger heifers (Hays and Albright, 1966). Udder edema can be a major discomfort to the animal and causes management problems such as difficulty with milking machine attachment, increased risk of teat and udder injury, and mastitis. Severe udder edema may reduce milk production and cause a pendulous udder (Dentine and McDaniel, 1984).

The exact cause(s) of udder edema is unknown, more likely it is a multi-factorial condition. Restriction or stasis

of venous and lymph flow from the udder in late pregnancy due to fetal pressure in the pelvic cavity, or increased blood flow to the udder without the concomitant increase in flow from the udder, causing increased venous pressure may be contributing factors (Vestweber and Al-Ani, 1983; Al-Ani and Vestweber, 1986). Changes in amounts and relative proportions of steroid hormones during late pregnancy may be involved, but are not well understood (Mavlen et al., 1983; Miller et al., 1993). Reduced concentrations of proteins and especially globulins in blood, suggesting an increase in vascular permeability as animals approach calving, were associated with greater incidences of udder edema (Vestweber and Al-Ani, 1984). Other potential causes such as inheritance and dietary factors have been associated with the condition (Al-Ani and Vestweber, 1986). The remaining discussion focuses on possible contributing nutritional factors.

### HIGH CONCENTRATE (GRAIN) FEEDING PREPARTUM

Many early studies showed no effects of concentrate feeding prepartum on udder edema regardless of parity (Fountaine et al., 1949; Greenhalgh and Gardner, 1958; Schmidt and Schultz, 1959). However, Hathaway et al. (1957) and Hemken et al. (1960) reported increased severity of edema in cows fed greater amounts of concentrate before parturition. Emery et al. (1969) found increased udder edema in pregnant heifers fed 7 to 8 kg of concentrate/head per day compared with no concentrate during the last 3 weeks of gestation. Udder edema was not found in multiparous cows. Greenhalgh and Gardner (1958) observed no increase in the severity of udder edema in heifers fed 4 kg of concentrate/head per day. Effects of prepartum concentrate feeding on udder edema in multiparous cows are less well documented. In one study, cows fed diets composed primarily of corn and alfalfa silages (88 percent of diet, dry basis) plus 12 percent high moisture corn, or 53.5 percent silages plus 46.5 percent high moisture corn had more edema and mastitis than cows fed an all hay diet for 30 days prepartum (Johnson and Otterby, 1981). Overall, the degree of influence of concentrate feeding on udder edema is unclear and a biologic mechanism(s) has not been elucidated. The possibility of influence of other nutrients (e.g., minerals) present in some concentrate mixes should not be overlooked.

Obese cows may be more predisposed to udder edema (Vigue, 1963). Different concentrations of dietary protein, fed for the last 60 days of gestation did not affect incidence of udder edema, but the severity was greater in heifers than in cows (Wise et al., 1946).

### MINERALS

It was suggested that increased edema observed in heifers in the study of Emery et al. (1969) resulted from 1 percent trace mineralized salt in the grain mix rather than increased concentrate feeding. Excessive intakes of sodium and potassium were implicated as causative agents in udder edema (Randall et al., 1974; Conway et al., 1977; Sanders and Sanders, 1981; Jones et al., 1984). Restriction of sodium chloride and water intakes reduced the severity and incidence of udder edema in pregnant heifers (Hemken et al., 1969). Lower incidence and severity of udder edema were found when diets contained no supplemental salts of sodium or potassium (Randall et al., 1974). In a field study of two commercial dairy herds, potassium fertilization to improve alfalfa production was implicated as the cause of increased udder edema (Sanders and Sanders, 1981). Cows consumed about 450 g of potassium/head per day. In an earlier controlled study, consumption of 454 g of a combination of sodium and potassium chlorides increased the incidence and severity of udder edema (Randall et al., 1974). In a second study, the incidence and severity of udder edema were compared in pregnant heifers fed a grain mix containing 1 percent sodium chloride versus a grain mix with 4 percent supplemental potassium chloride plus 1 percent sodium chloride for 20 days with ad libitum intake of alfalfa hay. The mix with potassium chloride had no influence on the severity of udder edema (Randall et al., 1974). Chronic udder edema also was associated with anemia and hypomagnasemia (Hicks and Pauli, 1976).

Overall, evidence supports the idea that excessive intake of the chloride salts of sodium or potassium increases the severity of udder edema, especially in late pregnant heifers. Intake of these salts typically can be controlled in the peripartum period. Evaluation of other salts of sodium (e.g., sodium bicarbonate) as they might affect the severity of udder edema was not reported. However, Nestor et al. (1988) reported that the severity of udder edema was greater when pregnant heifers were fed additional potassium bicarbonate (0 versus 272 g/head per day) or sodium chloride (23 versus 136 g/head per day) separately, but not when both salts were fed together. Utilizing forages and other feeds that contain low basal concentrations of potassium and sodium would be prudent if udder edema is prevalent.

Tucker et al. (1992) and Lema et al. (1992) studied the effects of calcium chloride, a so-called anionic salt with diuretic properties, on incidence and severity of udder edema. Calcium chloride was used to reduce the cation-anion difference of the prepartum diet of primiparous and multiparous cows. In one study, udder edema was not reduced by supplementation of calcium chloride in the prepartum period, but edema tended to regress more quickly in the early postpartum period, especially in primiparous cows compared with multiparous cows. In a second study, pregnant heifers were fed similar basal diets supplemented with either calcium chloride (1.5 percent, dry basis) or calcium carbonate (2.17 percent) for 3 weeks

prepartum. Calcium chloride reduced udder edema most during the first week of feeding. The effect was less but still evident the last 2 weeks before calving. Onset and development of edema were more gradual in heifers fed calcium chloride prepartum. When animals were fed the same calcium chloride supplemented diet after parturition (without prepartum feeding of calcium chloride), udder edema was greater at 2 weeks postpartum for heifers fed calcium chloride versus calcium carbonate fed prepartum.

#### OXIDATIVE STRESS

Oxidative stress of mammary tissues resulting from reactive oxygen metabolites may play a role in udder edema (Mueller et al., 1989a; Miller et al., 1993; Mueller et al., 1998). Excessive reactive oxygen metabolites (e.g., superoxide and hydrogen peroxide) generated from increased metabolic activity, or for example, excessive exposure to aflatoxins, can initiate abnormal oxidative reactions causing peroxidation of lipids; damage to proteins, polysaccharides, and DNA; degeneration of integrity of cell walls and contents; and tissue damage. Reactive oxygen molecules by themselves are not reactive enough to cause peroxidative chain reactions, but conversion to even more reactive free radicals can be triggered by transition elements such as iron (a pro-oxidant). Release of catalytic iron occurs under conditions of stress, trauma, or nutritional imbalance. Zinc may protect against the catalytic action of iron.

Sources of endogenous molecules (e.g., transferrin, lactoferrin, ceruloplasmin, serum albumin, antioxidant enzymes, and glutathione) and exogenous antioxidants (e.g., β-carotene and α-tocopherol) are important to reduce excessive oxidation. Presumably the diet must supply adequate α-tocopherol (vitamin E) as a chain-breaking antioxidant, copper, zinc, and manganese for superoxide dismutase, selenium for glutathione peroxidase, zinc to displace catalytic iron, and magnesium and zinc to stabilize membranes and maintain cellular integrity.

Mueller et al. (1989b) evaluated the effectiveness of supplemental vitamin E to reduce severity of udder edema in pregnant heifers. Udder edema during the first week after calving was less in heifers supplemented for 6 weeks before calving with 1000 IU vitamin E/head per day versus none. In another study, late pregnant heifers were fed factorial combinations of vitamin E [0 or 1000 IU/head per day], zinc [0 or 800 mg/head per day (about 90 mg/kg)], and iron [0 or 12 g/head per day (about 1300 mg/kg)]. When effects were compared regardless of dietary iron concentration, supplemental vitamin E reduced severity of udder edema, but zinc did not. However, when iron was excessive, vitamin E was ineffective in reducing the severity of udder edema, but zinc was somewhat effective. It is believed that vitamin E and zinc may complement each other in antioxidant function.

Nutritional defense against oxidative stress likely is supplied by supplementation of dietary antioxidants fed to meet nutrient requirements (Mueller et al., 1998). More research evaluating effects of oversupply of pro-oxidants in the diet and (or) supplementation of antioxidants in excess of nutrient requirements would be helpful to understand the effects of oxidative stress on udder edema and potential for its prevention.

### *Milk Fever*

#### OCCURRENCE

Milk fever affects about 6 percent of the dairy cows in the United States each year, according to the 1996 National Animal Health Monitoring Survey (USDA, 1996). In these cows the calcium homeostatic mechanisms, which normally maintain blood calcium concentration between 9 and 10 mg/dl, fail and the lactational drain of calcium causes blood calcium concentration to fall below 5 mg/dl. This hypocalcemia impairs muscle and nerve function to such a degree that the cow is unable to rise. Intravenous calcium treatments are used to keep the cow with milk fever alive long enough for intestinal and bone calcium homeostatic mechanisms to adapt. Although milk fever is relatively easy to treat, cows that have had milk fever are more susceptible to other disorders such as mastitis (especially coliform), displaced abomasum, retained placenta, and ketosis (Curtis et al., 1983). Though milk fever affects only a small percentage of cows, nearly all cows experience some decrease in blood calcium (hypocalcemia) during the first days after calving, while their intestines and bones adapt to the calcium demands of lactation. This sub-clinical hypocalcemia contributes to inappetance in the fresh cow and predisposes the cow to develop other diseases such as ketosis, retained placenta, displacement of the abomasum, and mastitis. Efforts made to raise the concentration of calcium in the blood of the fresh cow can benefit milk production even in herds that do not seem to have a milk fever problem (Beede et al., 1991).

#### ETIOLOGY AND PATHOGENESIS

Milk fever is characterized by and the result of severe hypocalcemia (Oetzel and Goff, 1998). Hypophosphatemia (see phosphorus section in chapter 6) and hypomagnesemia also can be present and can be complicating factors in some cases. The degree of hypocalcemia experienced will depend on the amount of calcium leaving the extracellular calcium pool and the rate at which the calcium homeostasis system can replace that calcium loss. The adaptation to the onset of lactation during the critical first days of lactation is accomplished by release of parathyroid hormone (PTH), which reduces urinary calcium losses, stimulates bone cal-

cium resorption, and increases 1,25-dihydroxyvitamin D synthesis to enhance active intestinal transport of calcium. All three must be operational if hypocalcemia is to be minimized. Milk fever risk factors reduce the efficiency of one or more of these homeostatic mechanisms.

An important determinant of the risk for milk fever is the acid-base status of the cow at the time of parturition (Craige, 1947; Ender et al., 1971). Metabolic alkalosis impairs the physiologic activity of PTH so that bone resorption and production of 1,25-dihydroxyvitamin D are impaired reducing the ability to successfully adjust to the calcium demands of lactation (Block, 1984; Block, 1994; Gaynor et al., 1989; Goff et al., 1991; Phillippo et al., 1994). Evidence suggests that metabolic alkalosis induces conformational changes in the PTH receptor, which prevents tight binding of PTH to its receptor. Cows fed diets that are relatively high in potassium or sodium are in a relative state of metabolic alkalosis, which increases the likelihood that they will not successfully adapt to the calcium demands of lactation and will develop milk fever. The parathyroid glands recognize the onset of hypocalcemia and secrete adequate PTH. However, the tissues respond poorly to the PTH, leading to inadequate osteoclastic bone resorption and renal 1,25-dihydroxyvitamin D production (Goff et al., 1991; Phillippo et al., 1994). This is particularly evident in cows that have been treated for milk fever and require further treatments due to reappearance (relapse) of milk fever signs. These cows have very high blood PTH concentrations but produce little 1,25-dihydroxyvitamin D at parturition. Full recovery from milk fever occurs only after the cow has responded to the PTH by producing 1,25-dihydroxyvitamin D. Production of 1,25-dihydroxyvitamin D can be delayed for 24 to 48 hours in some cows (Goff et al., 1989).

## MILK FEVER RISK FACTORS

*Age* Heifers almost never develop milk fever. The risk of a cow developing milk fever increases with age. Heifers generally produce less colostrum than older cows, which may reduce the calcium stress they experience at calving. More importantly, the bones of heifers are still growing. Growing bones have large numbers of osteoclasts present, which can respond to parathyroid hormone more readily than the bones of mature cows. Aged cows have fewer intestinal vitamin D receptors (Horst et al., 1990).

*Breed* The Jersey and, to a lesser extent, the Swedish Red and White and Norwegian Red breeds are well known to have a higher incidence of milk fever. The reasons remain unclear. Colostrum and milk of Jersey cows have a higher content of calcium than that produced by Holsteins, which may place a relatively large calcium stress on the Jersey cows. In one study, Jersey cows had significantly fewer intestinal receptors for 1,25-dihydroxyvitamin D than did Holsteins (Goff et al., 1995). Fewer receptors may impair the ability of Jersey cows to maintain calcium homeostasis.

## NUTRITIONAL CONSIDERATIONS

*Dietary Cation-Anion Difference.* Because metabolic alkalosis is an important factor in the etiology of milk fever it is important to prevent metabolic alkalosis. The reason the cow's blood is alkaline is because of high dietary cations, especially potassium. Cations are minerals with a positive charge and include potassium, sodium, calcium, and magnesium. If the cations in the feed are absorbed into the blood they cause the blood to become more alkaline. If dietary cations are not absorbed they do not affect blood pH (Stewart, 1983). Nearly all of the potassium and sodium in the diet is absorbed by cows, making these two elements very powerful alkalinizing cations. Calcium and magnesium are poorly absorbed from the diet of the dry cow so these cations are not strong alkalinizing agents. Dry cow diets that are high in potassium, sodium, or both alkalinize the cow's blood and increase the susceptibility for milk fever. Addition of potassium or sodium to the prepartal ration of dairy cows will increase the incidence of milk fever. Adding calcium (from 0.5 to 1.5 percent) to practical prepartal diets does not increase the incidence of milk fever (Goff and Horst, 1997a).

*Hypomagnesemia.* A second common cause of hypocalcemia and milk fever in the periparturient cow is hypomagnesemia (van de Braak et al., 1987; Allen and Davies, 1981; Barber et al., 1983; Sansom et al., 1983). Low magnesium in blood can reduce PTH secretion from the parathyroid glands; and can alter the responsiveness of tissues to PTH by inducing conformational changes in the PTH receptor and G-stimulatory protein complex (Rude et al., 1985; Rude et al., 1978; Littledike et al., 1983). Cows fed adequate dietary magnesium in the prepartal ration will be slightly hypermagnesemic the day after parturition. Blood magnesium concentrations below 2.0 mg/dl within 24 h after calving suggest inadequate dietary magnesium absorption (Goff, 1998b).

## PREVENTION OF MILK FEVER

*Adjustment of Dietary Cation-Anion Difference (DCAD)* Equations to describe DCAD include $(Na^+ + K^+) - (Cl^- + S^{-2})$ (Ender et al., 1971), $(Na^+ + K^+ - Cl^-)$ (Mongin, 1981), and $(Na^+ + K^+ + 0.15\ Ca^{+2} + 0.15\ Mg^{+2}) - (Cl^- + 0.6\ S^{-2} + 0.5\ P^{-3})$ (Goff et al., 1997). The last equation assigns coefficients to the major dietary cations and anions based on their acidifying or alkalinizing potential. To achieve a low DCAD prepartal ration to prevent

hypocalcemia, the following adjustments are recommended:

*Reduce Dietary Sodium and Potassium* Removing potassium from the ration can present a problem as alfalfa, other legumes, and many grasses accumulate potassium within their tissues to concentrations that are well above that required for optimal growth of the plant if soil potassium is high. Corn, a warm season grass, is less likely to accumulate potassium and corn silage is often a practical feedstuff to use to reduce DCAD (Beede, 1992). Other agronomic options to reduce dietary potassium have recently been reviewed (Horst et al., 1997; Thomas, 1999).

*Add Anions to Induce Mild (Compensated) Metabolic Acidosis* Landmark studies (Ender et al., 1971; Ender and Dishington, 1967; Block, 1984) demonstrated that addition of anions to the prepartal diet could prevent milk fever. Ammonium, calcium, and magnesium salts of chloride and sulfate have been successfully used as acidifying anion sources. Chloride salts are more acidogenic than sulfate salts (Goff et al., 1997; Oetzel, 1991; Tucker et al., 1991). Hydrochloric acid also has been successfully utilized as a source of anions for prevention of milk fever and is the most potent of the anion sources available (Ender and Dishington, 1967; Goff and Horst, 1998). Monitoring urine pH of cows during the week before parturition has proven an effective means of assessing effectiveness of anion addition to the prepartal ration. In Holstein cows effective anion addition reduces urine pH to between 6.2 and 6.8 (Gaynor et al., 1989; Jardon, 1995; Oetzel and Goff, 1998). Using the equation favored by most nutritionists, $(Na^+ + K^+) - (Cl^- + S^{-2})$ it is common to attempt to bring DCAD below zero mEq/kg diet to achieve proper acidification of the cow. These targets are not well defined and anions should be added in small increments to the dry cow ration until the proper urine pH is achieved. Urine pH can be assessed as quickly as 48 to 72 hours after a DCAD adjustment.

Feeding 0.35 to 0.40 percent magnesium in prepartal rations prevents a decline in the concentration of magnesium in the blood at parturition. These levels ensure that there is adequate magnesium in the rumen to utilize the passive absorption mechanism for magnesium across the rumen wall and not be reliant on active transport of magnesium across the rumen wall, a process that may be inhibited by dietary potassium (Oetzel and Goff, 1998). Because there is no readily labile body store of magnesium, the daily intake of dietary magnesium must supply needs. These higher levels are needed to accommodate the decline in DMI occurring in the periparturient period (Goff, 1998b; Horst and Goff, 1997). Phosphorus requirements are met by feeding 40 to 50 g of phosphorus/cow/day. Less than 25 g/cow/day may lead to hypophosphatemia and the downer cow syndrome (Julien et al., 1977; Goff, 1998a; Cox, 1998). More than 80 g of phosphorus/cow/day may induce milk fever (Barton et al., 1987).

The optimal prepartal dietary calcium concentration is not well defined. In one study, the incidence of milk fever was not different in cows fed 0.5 or 1.5 percent calcium in diets (Goff and Horst, 1997a). Other studies have successfully utilized diets providing more than 150 g of calcium/cow/day along with anionic salts to prevent hypocalcemia (Oetzel, 1988; Beede et al., 1991). Very high concentrations of dietary calcium (>1.0 percent calcium) may reduce DMI and animal performance (Miller, 1983).

*Very Low Calcium Diets to Prevent Milk Fever* Diets providing less than 15 g calcium/cow/day and fed for at least 10 days before calving will reduce the incidence of milk fever (Goings et al., 1974; Boda, 1954). This concentration of calcium places the cow in negative calcium balance, stimulating parathyroid hormone secretion prior to calving. This activates bone osteoclasts stimulating bone calcium resorption and activates renal tubules to resorb urinary calcium and begin producing 1,25-dihydroxyvitamin D prior to calving (Green et al., 1981). Thus at the onset of lactation these homeostatic mechanisms for calcium are active, preventing a severe decline in the concentration of calcium in the plasma of cows. In the United States, it is nearly impossible to formulate this type of diet. Diets consisting of as little as 35 to 45 g of calcium/day will meet the calcium requirement of cows and will not stimulate the parathyroid glands adequately and will not effectively prevent milk fever.

*Oral Calcium Drenches at Calving* Oral administration of calcium at calving reduces the incidence of milk fever but carries a slight risk of inducing aspiration pneumonia (Jonsson and Pehrson, 1970; Hallgren, 1955; Oetzel, 1993; Goff et al., 1996), and can be labor intensive.

*Exogenous Vitamin D and Parathyroid Hormone* Earlier literature often recommended feeding or injecting massive doses (up to 10 million units) of vitamin D 10 to 14 days prior to calving to prevent milk fever (Hibbs and Pounden, 1955; Littledike and Horst, 1980). This will increase intestinal absorption of calcium and can help prevent milk fever. Unfortunately, the dose of vitamin D that effectively prevents milk fever is very close to the level that causes irreversible metastatic calcification of soft tissues. Lower doses may actually induce milk fever because the high levels of 25-OH D and 1,25-dihydroxyvitamin D suppress PTH secretion and renal synthesis of endogenous 1,25-dihydroxyvitamin D (Littledike and Horst, 1980).

Treatment with 1,25-dihydroxyvitamin D and its analogues or parathyroid hormone prior to calving can be effective but the effective dose is close to the toxic dose

and problems with timing of administration, withdrawal from treatment, and expense have not made these treatments practical (Bar et al., 1985; Goff and Horst, 1990; Goff et al., 1986).

### *Grass Tetany*

Hypomagnesemic tetany is most often associated with cows in early lactation (milk production removes 0.15 g magnesium from the blood for each liter of milk produced) grazing lush pastures high in potassium and nitrogen and low in magnesium and sodium (Littledike et al., 1983). This is the most common situation and it is often referred to as Grass Tetany, Spring Tetany, Grass Staggers, or Lactation Tetany. The clinical signs in affected cows will depend on the severity of the hypomagnesemia. The disease will progess more rapidly and tends to be more severe if accompanied by hypocalcemia, which is often the case. Dairy cows are usually affected 1 to 3 weeks into lactation especially if they are on pasture. Moderate hypomagnesemia (between 0.5 and 0.75 mmol/L or 1.1 and 1.8 mg/dl) is associated with reduced DMI, nervousness, and reduced production of milk fat and total milk. This can be a chronic problem in some dairy herds that often goes unnoticed. It also can predispose these animals to milk fever (Goff, 1998).

Despite the importance of magnesium there is no hormonal mechanism concerned principally and directly with magnesium homeostasis. Factors affecting magnesium transport across the rumen epithelium have been discussed in the section on magnesium requirements.

#### PREVENTION

If hypomagnesemic tetany has occurred in one cow in a herd, steps should be taken immediately to increase intake of magnesium to prevent further losses. Getting an additional 10 to 15 g of magnesium into each pregnant cow and 30 g of magnesium into each lactating dairy cow each day will usually prevent further hypomagnesemic tetany cases. The problem with prevention is getting the extra magnesium into the animal (Goff, 1998b).

Most magnesium salts are unpalatable. Magnesium oxide is the most palatable, most concentrated, least expensive, and, unfortunately, least soluble source of magnesium. Magnesium is readily acceptable in grain concentrates. Including 60 g of magnesium oxide in just 0.5 to 1 kg of grain will be effective. However the expense of the grain and the problems associated with feeding concentrates to pastured cattle often make this option difficult to implement (Goff, 1998b).

Feeding ionophores (monensin, lasalocid) can improve activity of the sodium-linked magnesium transport system in the rumen, increasing magnesium absorption efficiency about 10 percent. However, ionophores are not approved for use in many of the animals they could benefit. Rumen boluses that release ionophores for up to 150 days have been developed to make delivery of ionophores to animals at pasture practical.

Pasture foliage can be dusted with magnesium oxide (500 g of magnesium oxide/cow or 50 kg magnesium oxide/hectare or 50 lb/acre) weekly during the period when cows are tetany prone. Adding 2.5–5 g/L or 10 to 20 lb/500 gal magnesium sulfate $7H_2O$ (epsom salts) or magnesium chloride $6H_2O$ to the drinking water can be an economic means of supplementing magnesium if cows have access to no other water supply as the addition of the salts can reduce palatability. Unfortunately cows grazing lush high moisture pasture rarely drink enough water to make this method effective on tetany prone pastures. Molasses licks and mineral blocks containing magnesium oxide and salt can help supply magnesium to animals at pasture if made readily available and if the animals learn to use the licks prior to parturition. A problem with many of these methods is that some cows in the herd may not voluntarily consume enough of the magnesium supplement and on some tetanogenic pastures cows that do not receive supplementation are often found dead (Goff, 1998b).

Intraruminal magnesium releasing boluses and bullets have been developed, which remain in the reticulum and release low levels of magnesium (1 to 1.5 g) each day for periods of up to 90 days. A 100 g magnesium alloy rumen "bullet" that is 86 percent magnesium has been developed and releases about 1 g of magnesium/day. Some producers administer 2 to 4 bullets per cow. These devices do not supply enough magnesium to raise magnesium in the blood subsantially, though there may be situations where they prove successful despite the low supplementation achieved.

### *Retained Placenta and Metritis*

Retained placenta (retained fetal membranes) is defined as failure of the fetal membranes to be expelled within 12 to 24 hours after parturition. Metritis, an inflammation or infection of the uterus, is often associated with retained placenta. In path analysis, retained placenta was associated directly with increased days to first service and risk of metritis when compared with cows that expelled their placentas within 24 hours. Also, retained placenta was associated indirectly with the greater occurrence of cystic ovaries, lower milk yield, and greater culling; all were mediated through metritis (Erb et al., 1985).

Multiple physiologic and nutritional factors have been associated with or implicated as causes of retained placenta and metritis (Maas, 1982; Miller et al., 1993; Goff and Horst, 1997b). Dystocia in heifers increased the risk of retained placenta and metritis by 3 to 4 times (Erb et al.,

1985). Other predisposing or associated factors include: twinning; various stressors; short dry periods; exposure to toxins such as mycotoxins or nitrates; heredity; milk fever; abnormally low prostaglandin $F_2$ concentrations in placentomes, caruncles, and cotyledons; and, other atypical peripartum profiles of steroid, pituitary, and adrenal hormones in blood (Pelissier, 1976; Chew et al., 1977; Leidl et al., 1980; Maas, 1982). Immunosuppression in the peripartum period has been implicated as a possible contributing factor (Goff and Horst, 1997b). Several dietary essential nutrients are involved in immune function. However, the exact mechanisms of action and nutrient needs to promote periparturient immunocompetence have not been elucidated fully.

### NUTRITIONAL FACTORS

Nutritional causes of retained placenta are due primarily to the diet fed the last 6 to 8 weeks before calving. During this time, dietary deficiencies or imbalances of energy; protein; phosphorus; calcium; selenium; iodine; vitamins A, D, and E; and excesses of dietary energy, protein, and calcium all have been associated with or implicated as causes of retained placenta and metritis (Maas, 1982; Weaver, 1987; Goff and Horst, 1997b).

*Energy and Protein* Extreme deficiency of dietary energy, protein or both can result in retained placenta because cows are weak, and coupled with the stress of parturition lack strength to expel the placenta (Mass, 1982). Cows fed diets for the entire dry period low in dietary crude protein (8 percent) had a higher incidence (50 percent) of retained placenta compared with cows fed 15 percent crude protein (20 percent incidence) (Julien et al., 1976a). Fat cow syndrome (hepatic lipidosis), resulting from excessive energy intake prepartum, also frequently is associated with increased incidences of retained placenta and metritis (Morrow, 1976).

*Phosphorus* The rate of retained placenta was associated with imbalances in calcium and phosphorus metabolism (Noorsdsy et al., 1973; Pelissier, 1976). However, Julien et al. (1977) found no influence of prepartum dietary phosphorus content (0.30 versus 0.70 percent, dry basis) on rate of retention of placentas, and the correlation between phosphorus intake and incidence of retained placenta was low. Dietary concentrations of calcium were similar among treatments, and rate of calcium intake was not correlated with retained placenta.

*Calcium* The association between hypocalcemia (either clinical or subclinical) in the peripartum period and retained placenta has been known for some time (Pelissier, 1976; Greunert, 1980). Excess of calcium and phosphorus in the diet, and a deficiency of vitamin $D_3$ all affect calcium metabolism in the periparturient period and can result in hypocalcemia. Path analyses showed that multiparous cows having milk fever were 2 times more likely to have retained placenta and metritis than cows without milk fever (Erb et al., 1985). In another study, multiparous cows having milk fever were 4 times more likely to have placental retention (Curtis et al., 1985). Hypocalcemia results in loss of muscle tone in the uterus, which may contribute to the increased incidence of retained placenta (Goff and Horst, 1997b). Decreasing the dietary cation-anion difference of the prepartum diet by supplementation of ammonium sulfate and ammonium chloride reduced hypocalcemia and reduced the incidence of retained placenta (Oetzel et al., 1988). Other details about prevention of hypocalcemia are discussed in the milk fever and transition cow sections of this publication. Harrison et al. (1984) reported that apparent absorption of selenium from natural feeds by nonlactating pregnant cows was lower with low (0.4 percent) or high (1.4 percent) dietary calcium, and maximal with about 0.8 percent calcium, dry basis; calcium intake ranged from about 30 to 200 g/cow per day. Even though large amounts of dietary calcium reduced selenium absorption, feeding 1.32 percent calcium with supplemental anions plus 3 mg of selenium/cow per day by oral bolus to pregnant Holstein cows for 14 to 21 days before calving did not negatively affect peripartum selenium status of cows or newborn calves compared with that of cows receiving 1.08 percent dietary calcium, no supplemental anions, and 3 mg of oral selenium (Gant et al., 1998).

*Selenium and Vitamin E* An excess of highly reactive oxygen metabolites (e.g., peroxides and superoxide) can cause peroxidative damage of cell membranes and other cellular components, and interfere with normal metabolic function, including normal steroidogenesis (Miller et al., 1993). Nutrient antioxidants (e.g., selenium and vitamin E) are needed to reduce peroxidation. Cows with retained placenta had lower total antioxidants in blood plasma during the 2 weeks before calving than cows without retained placenta (Miller et al., 1993). Supplementation of diets with antioxidants to meet requirements is crucial especially during the periparturient period (Weiss et al., 1990), when blood $\alpha$-tocopherol (vitamin E) concentrations are the lowest of the entire lactation cycle (Goff and Stabel, 1990; Weiss et al., 1990).

However, supplementation with selenium and vitamin E reduced the incidence of retained placenta and improved reproduction of dairy cows in some (Trinder et al., 1969, 1973; Julien et al., 1976b,c; Harrison et al., 1984; Mueller et al., 1988, 1989b,c; Thomas et al., 1990), but not all (Gwazdauskas et al., 1979; Schnigoethe et al., 1982; Ishak et al., 1983; Kappel et al., 1984; Hidiroglou et al., 1987; Stowe et al., 1988) comparisons. In cows fed selenium-

deficient diets (0.05 to 0.07 mg/kg), supplemental selenium substantially reduced the incidence of retained placenta (Julien et al., 1976 b,c). Administration of selenium and vitamin E in combination was more effective in reducing retained placenta than either antioxidant alone. Trinder et al. (1973) reported in a summary of three experiments involving 171 parturitions that injection 28 days before expected calving of 15 mg of selenium as potassium selenate alone was slightly less effective (10 percent incidence) for reducing retained placenta than the combination of selenium (15 mg) plus vitamin E (680 IU) (2 percent incidence). The incidence rate was 39 percent with no selenium or vitamin E injection. Basal diets contained between 0.025 and 0.047 mg/kg of selenium, dry basis. Segerson et al. (1981) found that the incidence of retained placenta was reduced by a combination of selenium and vitamin E injection in the prepartum period of cows marginally deficient in blood serum selenium concentrations (14.9 versus 25.4 percent incidences with and without selenium and vitamin E), but not in cows either adequate or very deficient in selenium. With prepartum diets having basal concentrations of 0.035 to 0.109 mg/kg of selenium, one injection of selenium (2.3 to 23 mg) alone about 3 weeks before expecting calving was as effective in reducing retained placenta as a selenium-vitamin E combination (Eger et al., 1985). Low doses of selenium (2.3 to 4.6 mg given 3 weeks before calving) tended to be more effective than higher doses. In some studies, 50 mg of selenium plus 680 IU of vitamin E were injected intramuscularly 3 weeks before expected calving of Holstein cows (Segerson et al., 1981; Julien et al., 1976 b, c). This amount of selenium was adequate to reduce retained placenta as long as adequate vitamin E also was administered; however, given alone neither nutrient was very effective for reducing retained placenta or metritis.

Retained placenta and days to conception were not reduced by 1000 IU of vitamin E/cow per day given orally in cows fed diets with less than 0.06 mg/kg of selenium, unless cows also were injected intramuscularly with 0.1 mg of selenium/kg live weight 3 weeks before expected calving (Harrison et al., 1984). When the diet contained at least 0.12 mg/kg of selenium, 1000 IU of dietary vitamin E/cow per day reduced the incidence of retained placenta compared with cows not receiving supplemental vitamin E (Mueller et al., 1988, 1989b,c; Thomas et al., 1990; Brzezinska-Slebodzinska and Miller, 1992). Miller et al. (1993) noted that preventive (e.g., selenium) and chain breaking antioxidants (e.g., vitamin E and β-carotene) work in concert, and effectiveness of the total antioxidant system is impaired if one or more of the antioxidant components is inadequate, and the supplemented antioxidant may be less effective if another antioxidant is limiting.

Schingoethe et al. (1982) and Ishak et al. (1983) found no reduction in incidence of retained placenta by intramuscular injection of selenium, vitamin E, or a combination of the two given in the feed, if diets already contained 0.1 to 2.0 mg/kg of selenium. Data are not available on the effect of supplementing more than 0.3 mg selenium/kg of feed (dry matter basis) and (or) more than 1000 IU of vitamin E per day on the incidence of retained placenta. The legal upper limit for selenium supplementation of complete diets for cattle is 0.3 mg/kg (Food and Drug Administration, 1997).

*Vitamin A and β-Carotene* Avitaminosis was shown to increase the incidence of retained placenta (Ronning et al., 1953; Roberts, 1961; Nicholson and Cunningham, 1965). Ronning et al., (1953) demonstrated a decrease in the incidence of retained placenta with increased intake of carotene. Dairy cows fed 600 mg/cow per day of β-carotene for 4 weeks before calving had reduced incidence of retained placenta compared with cows fed an equivalent amount of pre-formed vitamin A (240,000 IU/day) (Michal et al., 1990).

*Iodine* Controlled studies on the effects of iodine deficiency on the incidence of retained placenta and metritis are lacking. Some case studies indicate that iodine deficiency was associated with retained placenta (Moberg, 1959; McDonald et al., 1961; Hemken and Vandersall, 1967). However, in a later field study with 1,572 cows in an iodine-deficient area of Finland, retained placenta was not reduced with supplementary iodine (Moberg, 1961). An association between retained placenta and goiter in calves was reported (Maas, 1982).

### *Displacement of the Abomasum*

Displacement of the abomasum is a disease of increasing importance as milk production increases in the United States. A survey of high producing herds found that on average 3.3 percent of cows developed displaced abomasum (Jordan and Fourdraine, 1993). The transition period from 3 weeks before calving until 4 weeks postpartum is the major risk period for development of displaced abomasum. About 85 percent of cases involve displacement to the left side of the cow.

#### ABOMASAL PHYSIOLOGY

In the nonpregnant cow, the abomasum occupies the ventral portion of the abdomen, very nearly on the midline, with the pylorus extending to the right side of the cow caudal to the omasum. As pregnancy progresses, the growing uterus occupies an increasing amount of the abdominal cavity. The uterus begins to slide under the caudal aspects of the rumen, reducing rumen volume by one third at the end of gestation. This also forces the abomasum forward

and slightly to the left side of the cow, although the pylorus continues to extend across the abdomen to the right side of the cow (Habel, 1981). After calving, the uterus retracts back toward the pelvic inlet which, under normal conditions, allows the abomasum to return to its original position. During left displacement of the abomasum, the pyloric end of the abomasum slides completely under the rumen to the left side of the cow. Three factors are believed to be responsible for allowing the abomasum to move to the left side of the cow. First, the rumen must fail to take up the void left by the retracting uterus. If the rumen moved into its normal position on the left ventral floor of the abdomen, the abomasum would not be able to slide under it. Second, the omentum attached to the abomasum must have been stretched to permit movement of the abomasum to the left side. These two factors constitute opportunity for displacement. A third factor necessary to cause abomasal displacement is abomasal atony. Normally, gases produced in the abomasum (from fermentation of feedstuffs or $CO_2$ released when bicarbonate from the rumen meets the HCl of the abomasum) are expelled back into the rumen as a result of abomasal contractions. It is felt that these contractions are impaired in cows developing left displacement of the abomasum (Goff and Horst, 1997b; Breukink, 1991; Hull and Wass, 1973). The cause of abomasal atony is less clear.

A decline in the concentration of calcium in plasma around parturition linearly decreases abomasal contractility, which is suspected to lead to atony and distension of the abomasum (Massey et al., 1993; Hull and Wass, 1973; Curtis et al., 1983). At a concentration of 5 mg/dl calcium in plasma, abomasal motility was reduced by 70 percent and strength of contractions is reduced by 50 percent as compared to when plasma calcium is normal (9 to 10 mg/dl). At a concentration of 7.5 mg/dl calcium in plasma, the motility and strength of abomasal contractions were reduced by 30 percent and 25 percent, respectively (Daniel, 1983). Clinical signs of milk fever (down cows) often are not seen until calcium is about 4 mg/dl. In a recent study of plasma calcium concentrations in periparturient Holstein and Jersey cows, 10 to 50 percent of cows remained subclinically hypocalcemic (plasma calcium $<7.5$ mg/dl) up to 10 days after calving, depending on herd efforts to combat milk fever (Goff et al., 1986). Oetzel (1996) reported that administration of oral calcium chloride at calving to reduce subclinical hypocalcemia resulted in a significant decrease in the incidence of displacement of the abomasum.

Decreasing the forage to concentrate ratio of the diet fed in late gestation and early lactation will increase the incidence of displaced abomasum (Coppock et al., 1972). Volatile fatty acids within the abomasum have been demonstrated to reduce abomasal contractility (Breukink, 1991); however, ruminal VFA concentrations are not highly correlated with the concentration of VFA in the abomasum (Breukink and de Ruyter, 1976). A high grain, low forage diet can promote the appearance of VFA in the abomasum by reducing the depth of the ruminal mat or raft (made up primarily of the long fibers of forages). Physical reduction of forage particle length by chopping forages too finely prior to ensiling or overzealous use of mixer wagons also can contribute to loss of rumen raft (Shaver, 1997). The ruminal raft captures grain particles so that they are fermented at the top of the ruminal fliud. The VFA produced at the top of the ruminal fliud are generally absorbed from the rumen with little VFA entering the abomasum. In cows with an inadequate ruminal raft, grain particles fall to the ventral portion of the rumen and reticulum where they are fermented or pass on to the abomasum (where they can then be fermented to some extent). The VFA produced in the ventral rumen can pass through the rumenoreticular orifice to enter the abomasum before the rumen can absorb them. A thick ruminal raft is generally present during the dry period when cows are fed a high forage diet, but the depth of the ruminal raft is rapidly reduced in early lactation; especially if the cow experiences a pronounced decline in DMI. Since the ruminal raft also stimulates regurgitation of the cud and mastication, the release of saliva, which promotes rumen buffering, is decreased when cows are fed a higher grain ration. Also, early in lactation, the underdeveloped ruminal papillae allow more of the VFA produced in the ventral rumen to escape the rumen than would a highly absorptive ruminal mucosa typical of later lactation (Dirksen, 1985).

Cows dried off with high body condition scores are at increased risk of left displaced abomasum as a result of poor DMI around parturition (Cameron et al., 1998).

The amount of effective fiber determines the consistency and depth of the rumen raft and stimulates rumen contractility. Guidelines are poorly defined, but readers are referred to Chapter 4 of this report and recent reviews that discuss methods for evaluating the amount of effective fiber needed in the diet (Armentano and Pereira, 1997; Firkins, 1997; Grant, 1997; Allen, 1997; Mertens, 1997).

TMR that are easily sorted by cows may affect the ratio of forage to concentrate of total feed consumed by individual cows and will contribute to displaced abomasum (Shaver, 1997). When a TMR is not fed, grain intake after calving should be increased slowly (0.2 to 0.25 kg/day) until peak grain intake is achieved. Grain fed to cows should be divided into at least 3 meals per day (Shaver, 1997).

### *Rumen Acidosis and Laminitis*

Acids produced during fermentation of grains generally keep rumen pH slightly below neutral or 7.0. How far below neutral is dependent on the rate of production and total amount of acid produced, the rate of absorption of

acids out of the rumen, and the amount of salivary secretion released to neutralize the acids. High forage diets produce acids only slowly and stimulate release of large amounts of saliva as they stimulate mastication. Rumen pH tends to be higher on forage diets. Rumen acidosis is associated with the feeding of diets with higher amounts of grain in them and the acidosis commonly occurs in the first month of lactation (Nocek, 1997). Upon dry-off, the cow is fed a high forage ration that is less energy dense and higher in neutral detergent fiber than the lactation ration. This affects rumen function in two ways. The bacterial population shifts away from the lactate producers (bacteria such as *Streptococcus bovis*, and the lactobacilli) as a result of the decrease in readily fermentable starches in the diet (Yokoyama and Johnson, 1988) Therefore, the population of those bacteria (primarily *Megasphaera elsdenii* and *Selenomonas ruminantium*) capable of converting lactate to acetate, propionate, or long chain fatty acids useful to the cow declines. Another effect of the lower energy diet of the early dry period is a reduction in the papillae length and volatile fatty acid (VFA) absorptive capacity of the ruminal mucosa. As much as 50 percent of the absorptive area may be lost during the first 7 weeks of the dry period (Dirksen et al., 1985). If the fresh cow is now abruptly switched to a high energy lactation diet, she is at risk of developing rumen acidosis because the lactate producers will respond rapidly to the higher starch diets and produce high amounts of lactate. The lactate converting bacterial population responds only slowly to a change in diet, requiring 3 to 4 wk to reach levels that will effectively prevent lactate from building up in the rumen. Lactate is a stronger acid ($pK_a$ = 3.86) than propionate ($pK_a$ = 4.87), acetate ($pK_a$ = 4.76), or butyrate ($pK_a$ = 4.82), so that its presence has a slightly larger effect on rumen pH than the VFA especially as the rumen pH falls below 6.0. Also, lactate and the other VFA are absorbed by rumen epithelium when in the undissociated acid state only. As the pH of the rumen decreases more of the VFA exists in the undissociated acid state. Because the $pK_a$ of lactate is lower than the VFA, it is absorbed more slowly than acetate, propionate, or butyrate from the rumen (Merchen, 1988).

Normally, only a small amount of lactate is produced within the rumen and it is all in the L-lactate form. An early hypothesis on the etiology of the "grain overload" syndrome attached a great deal of significance to the observation that under conditions of grain engorgement, D-lactate is produced in very high amounts by the lactobacilli. The theory contended that D-lactate was not absorbed from the rumen as well as L-lactate, and also was metabolized only slowly by body tissues once it was absorbed. However D-lactate is absorbed from the rumen (Huntington and Britton, 1979) and metabolized by tissues (Harmon et al., 1983) at the same rate as L-lactate.

Much of the research conducted on the etiology of rumen acidosis was performed using the feedlot steer as a model. In this model it appears that lactic acid production is an important aspect of the development of rumen acidosis. However recent research with dairy cattle suggests that rumen acidosis observed in early and mid-lactation cows is more closely related to total VFA production within the rumen and build-up of VFA. High rumen fluid lactic acid concentrations may not play as prominent a role in dairy "rumen acidosis" as it does in feedlot cattle (Oetzel et al., 1999).

The lactic acid, and the endotoxins and histamine released as the rumen flora die, are absorbed systemically, and affect the microvasculature of the growing hoof wall, which can then result in clinical laminitis (Radostits et al., 1994) Metabolic acidosis will follow rumen acidosis if the amount of organic acid absorbed into the blood exceeds the ability of the liver and other tissues to metabolize these anions.

The scientific name for laminitis is pododermatitis aseptic diffusa, which is an inflammation of the dermal layers inside the foot. Incidence rate of laminitis from surveys ranged from 5.5 to 30 percent (Nocek, 1997). Laminitis has a multifaceted etiology and is thought to be associated with several, largely independent factors. Nutritional management has been identified as a key component in the development of laminitis, particularly the feeding of diets high in fermentable carbohydrates, which can result in an acidotic state. Manson and Leaver (1988) compared diets containing 60:40 or 40:60 concentrate to silage fed to cows during weeks 3 to 26 of lactation. The 60:40 diet increased the number and incidences of clinical lameness, and decreased hoof hardness. Other nutritional factors including adequate effective fiber to stimulate salivation and presence of mycotoxins have been suggested to predispose animals to the development of laminitis (Vermunt and Greenough, 1994). Metabolic and digestive disorders can predispose the cow to laminitis. Supplementary biotin may improve hoof strength and provide greater resistance to laminitis (Midla et al., 1998).

Infectious diseases, such as mastitis, metritis, and foot rot, can cause specific endotoxic insults (Maclean, 1971). Factors related to the environment, such as hard surfaces, lack of or little use of bedding, and lack of or excessive exercise on undesirable surfaces, can separately or in combination predispose animals to mechanical damage (Bergsten, 1994). Bergsten (1995) found a higher prevalence of solar hemorrhages in cows kept in tie stalls with concrete floors as compared with cows housed in tie stalls that had been fitted with rubber mats.

Other factors such as excess body condition, heavy body weight, and poor feet and leg structure (Greenough, 1991), can increase the weight load and stress on feet, exacerbat-

ing the internal mechanical damage that is associated with laminitis (Nocek, 1997).

### *Milk Fat Depression*

Diet has a significant impact on both the yield and concentration of fat in milk of the dairy cow. Fat is the milk component on which diet has the greatest influence (Sutton, 1989), principally by reducing fat content. Requirements for NDF and effective fiber are driven in part, by the need to maintain milk fat (Erdman, 1988). Several dietary factors are known to depress milk fat including high levels of concentrate, finely chopped forages, and diets containing high amounts of polyunsaturated fatty acids such as those contained in either marine or vegetable oils. Under extreme dietary circumstances, milk fat concentration can be reduced by as much 50 to 60 percent from normal levels.

#### PREVIOUS THEORIES

The influence of diet and the dietary factors that alter milk fat has been known for a long time. Van Soest (1994) cited work of Boussingault in 1845 that documented low milk fat in cows fed a low fiber and high starch diet of beets. Petersen (1932) demonstrated that feeding cod liver oil markedly reduced milk fat. Shaw and Ensor (1959) confirmed the response to feeding cod liver oil and also found that inclusion of vegetable oils containing high levels of polyunsaturated fatty acids (PUFA) in the diet, also depressed milk fat content. Powell (1939) in a series of experiments showed that feeding high grain diets or feeding diets with ground or pelleted hay reduced milk fat and he concluded that the factors, which caused the fat depression, were related to changes in rumen fermentation. Finally, Emery and Brown (1961) demonstrated the importance of rumen pH in milk fat depression where the addition of dietary buffers partially corrected the reduction in milk fat content in cows fed high concentrate diets.

Most previously proposed mechanisms by which diet influences milk fat concentration have been related to changes in rumen volatile fatty acids (VFA) and the subsequent changes in metabolism associated with altered rumen VFA (Van Soest, 1994). Where milk fat depression is caused by feeding either high grain diets or diets where the forage particle size is too small, the molar percentage of acetate declines and propionate increases. Tyznick and Allen (1951) suggested that because of the reduced rumen acetate concentrations, the reduction in milk fat was caused by a deficiency in acetate, a precursor for de novo fatty acid synthesis in the mammary gland.

Because of the acetate deficiency theory, rumen acetate: propionate molar ratios have frequently been used as an indicator of fermentation changes associated with fat depressing diets. The importance of rumen VFA patterns was reinforced by the work of Emery and Brown (1961) and Emery et al. (1964). Based on the observation that rumen pH was reduced when cows were fed low forage diets, it was correctly hypothesized that addition of buffer to the diet would increase rumen pH and milk fat production. These experiments also showed increased rumen acetate: propionate ratios with buffer addition that reinforced the concept that changes in rumen VFA patterns were the cause of milk fat depression. However, Bauman and Davis (1970) using radioisotopes to measure actual VFA production in the rumen, showed that acetate production did not decrease with fat depressing diets, but rather, propionate production increased causing decreased acetate: propionate molar ratios. This indicated that acetate did not become deficient during milk fat depression.

Other theories related to changes in rumen VFA patterns included a deficiency in beta-hydroxy butyrate (Van Soest and Allen, 1959), increased uptake of acetate by adipose caused by elevated blood insulin (McClymont and Valence, 1962) and vitamin $B_{12}$ deficiency (Frobish and Davis, 1977). Intramuscular injections of vitamin $B_{12}$ had no effect on milk fat percentage in cows that were fed fat depressing diets (Croom et al., 1981). McGuire et al. (1995) using a hyperinsulinemic-euglycemic clamp where circulating insulin levels were increased 5-fold while blood glucose was held constant, failed to show any effect of insulin on milk fat synthesis.

#### ROLE OF TRANS FATTY ACIDS

Research conducted during the last 10 years strongly suggests that milk fat depression is the result of changes in the rumen biohydrogenation process and not changes in rumen VFA patterns. Rumen biohydrogenation is the process by which polyunsaturated fatty acid (PUFA) present in dietary fat, are hydrogenated (saturated) by rumen bacteria. Under normal conditions, very few unsaturated fatty acids reach the small intestine even when large amounts of PUFA are fed because of rumen biohydrogenation. The predominant PUFA in dairy cow diets are linoleic (C 18:2) and linolenic (C 18:3) acids in plant lipids whereas eicosapentaenoic (C 20:5), docosapentaenoic (C 22:5) and docosahexaenoic (C 22:6) acids are common PUFA in marine oils. The biohydrogenation process is one of the principle reasons that ruminant animals generally have both milk and tissue fatty acids that are highly saturated (Christie, 1981).

The steps in rumen fatty acid biohydrogenation include: hydrolysis of triglycerides to glycerol and free fatty acids; isomerization of PUFA to trans double bond containing dienes such as conjugated linoleic acid (CLA) (cis-9, trans-11 C-18), hydrogenation of CLA to vaccenic acid (trans-11, C-18:1) and finally hydrogenation of vaccenic acid to

stearic acid (C 18:0) (Harfoot and Hazlewood, 1987). Although the steps outlined above are typically thought of as the most common pathways of rumen biohydrogenation, many different CLA and trans 18:1 isomers are created as the result of rumen biohydrogenation. Although trans-11 is the most common, positional isomers ranging from trans-3 to trans-16 have been identified in rumen contents (Katz and Keeney, 1966; Griinari et al., 1998; Piperova et al., 2000). The numbers of individual CLA isomers produced are even greater including both positional and geometric (cis versus trans) variation in the double bonds (Yurawecz et al., 1998; Piperova et al., 2000).

During milk fat depression, trans fatty acids (TFA) in milk are increased. Storry and Rook (1965) first reported increased TFA in milk of cows fed high concentrate restricted forage diets. Teter et al. (1990) found a significant negative correlation between milk TFA content and milk fat percent ($r = -0.53$). Wonsil et al. (1994) found that the change in fat concentration in milk was negatively related to TFA content in milk fat. Postruminal infusion of TFA isomers resulted in milk fat depression (Gaynor et al., 1994; Romo et al., 1996). High concentrate diets result in increased TFA in milk (Kalscheur et al., 1997a; Griinari et al., 1998). The addition of buffers in the diet decreased both duodenal flow of TFA and TFA content in milk fat but increased milk fat percent (Kalscheur et al., 1997a).

The trans double bond in TFA and CLA's in milk fat can originate only from bacterial (rumen) fermentation and not from metabolism of the cow. Milk TFA can increase substantially in cows fed diets that are high in PUFA without depressing milk fat percent if diets contain adequate forage (Griinari, et al., 1998; Kalscheur et al., 1997b). The specific positional isomer of the TFA double bond produced during rumen fermentation is important. Griinari et al. (1998) reported that milk fat depression occurred only when trans-10 (C18:1) isomer was increased compared with normal situations where trans-11 (C18:1) isomer was the most abundant. In this experiment, cows fed high concentrate diets with the addition of vegetable fat had reduced milk fat which corresponded to increased trans-10 (C18:1) whereas, feeding vegetable fat in a high forage diet resulted in increased trans-11 (C18:1) isomer but did not result in milk fat depression.

Conjugated linoleic acid can directly decrease milk fat content. Chouinard et al. (1999) showed that postruminal infusion of 50 to 150 grams per day of CLA mixtures decreased milk fat by more than 50 percent. More recent experiments (Baumgard et al., 2000; Chouinard et al., 1999) using mixtures where the proportion of cis-9, trans-11 CLA versus trans-10, cis-12 CLA are altered showed that trans-10 containing CLA inhibit milk fat synthesis while trans-11 CLA have no effect on milk fat synthesis. The amounts of trans-10 containing CLA required to reduce milk fat are small (10 grams or less) compared to the amounts of TFA reaching the duodenum (50–300 g/day). At present, the amounts of trans-10 containing CLA that reach the duodenum for absorption are not known. The fact that postruminal infusion of either trans-10 containing C18:1 fatty acids or CLA decrease milk fat suggest that both probably play a role in diet induced milk fat depression. Although there is strong evidence that cis-9, trans-11 CLA can be synthesized in the mammary gland from absorbed trans-11 (C18:1) fatty acids (Griinari et al., 2000), the source of trans-10 containing CLA is presumed to be the result of rumen fermentation because a delta-10 desaturase enzyme has not been reported.

The short chain fatty acids ($C<16$) are the fatty acids in milk fat that are decreased most during milk fat depression. This suggests that the mechanism by which trans containing fatty acids reduce overall fat synthesis is by a reduction in de novo fatty acid synthesis. Acetyl CoA carboxylase (ACC) has been demonstrated to be the rate limiting enzyme for fatty acid synthesis in the mammary gland (Mellenberger et al. 1973). Measured enzyme activities along with mRNA for ACC, fatty acid synthase, and stearolyl CoA desaturase in the mammary gland of cows have been reported to be markedly reduced in cows fed fat depressing diets (Loor et al., 1998; Piperova et al., 2000).

Milk fat percent and yield can be altered by diet formulation and ingredient selection. Two factors, dietary PUFA and dietary fiber are important. Dietary polyunsaturated fatty acids are required as substrates for production of CLA and TFA. Inadequate dietary fiber results in low rumen pH. Low rumen pH influences the proportion of trans-10 fatty acids produced as a result of rumen biohydrogenation and potentially inhibits the complete saturation of trans 18:1 fatty acids to stearate. Griinari et al. (1998) found that increased trans-10 fatty acid in milk fat and milk fat depression occurred only when low forage diets (low rumen pH) supplemented with vegetable oil (PUFA source) were fed. The direct effect of rumen pH was shown by Kalscheur et al. (1997a) where addition of buffers to high concentrate diets reduced duodenal TFA flow, decreased milk TFA content, and increased milk fat percent. Diets that cause mean rumen pH to fall below 6.0 appear to be required to cause milk fat depression, and addition of dietary buffers to correct milk fat depression is effective as a means to increase rumen pH and milk fat percent in those circumstances (Erdman, 1988). Addition of high levels of polyunsaturated fatty acids failed to cause milk fat depression in diets with normal amounts of forage (Kalscheur et al., 1997b; Griinari et al., 1998). Sources of dietary PUFA can include those from direct addition of marine or vegetable oils, indirectly from feedstuffs that are relatively high in fat such as fish meal or oilseeds, or to a lesser extent, those naturally contained in cereal grains and forages. Diet formulations that result in adequate amounts of effective fiber needed to maintain adequate rumen pH, and those

that also restrict the amounts of dietary PUFA as potential sources of TFA, should result in normal milk fat percent.

## PERFORMANCE MODIFIERS

### *Mineral Salts and Their Role as Buffers*

Compounds such as sodium bicarbonate, sodium sesquicarbonate, calcium carbonate, or magnesium oxide are added to diets in an effort to reduce digestive upsets or to maintain milk fat percentage when diets high in grain and carbohydrate fermentability or low in effective fiber are fed to lactating dairy cows. Efficacy of dietary buffers for dairy cows is well-documented (Davis, 1979; Emery, 1976; Erdman, 1988; Muller, 1979). Sodium bicarbonate has been shown to increase DMI (Erdman et al., 1982; Staples et al., 1986; Vicini et al., 1988; West et al., 1987), milk yield (Erdman et al., 1980; Kilmer et al., 1981; Rogers et al., 1985; Solorzano et al., 1989; Thomas et al., 1984), and milk fat yield (Rogers et al., 1985; Soloranzo et al., 1989; Staples et al., 1986) in some studies. In other studies (DePeters et al., 1984; Rogers et al., 1985) there was no response to dietary buffers.

Erdman (1988) reported that the relationship between ruminal pH and the percentage of fat in milk from dairy cows fed alfalfa haylage-based rations was not significant. Additionally, relationships between dietary sodium bicarbonate or magnesium oxide and blood or urine pH, blood $pCO_2$, or blood bicarbonate concentrations were not found to be significant (Erdman, 1988). Aslam et al. (1991) indicated there was no difference in ruminal pH or volatile fatty acid concentrations for a period of 6 hours after the addition of sodium bicarbonate to the rumen. Two sodium bicarbonate buffers fed to dairy cows resulted in no differences in ruminal pH or organic acids; however, milk fat percentage was increased for some diets (Xu et al., 1994). Tucker et al. (1994) evaluated the use of 0 or 1 percent sodium sesquicarbonate in lactating dairy cows for 308 days postpartum. The buffer did not affect milk yield or composition the first 56 days of lactation. In midlactation (56 to 252 days postpartum), buffer increased milk protein content only. During 252 to 308 days postpartum, fat and protein contents in milk increased with buffer supplementation. Xin et al. (1989) observed that 0.4 percent MgO increased milk fat content without increasing milk yield. Sodium sesquicarbonate increased milk fat percentage and 4 percent FCM (Cassida et al., 1988). A buffer containing KCl, NaCl, and Mg and Na bicarbonates increased 4 percent FCM without modifying DMI (Soloranzo et al., 1989; Staples et al., 1986).

Staples and Lough (1989) summarized 41 experiments involving supplemental feeding of 0.4 to 1.7 percent sodium bicarbonate to dairy cows consuming diets that contained 57 percent concentrate. When corn silage was the main dietary forage, cows receiving supplemented diets produced an average of 0.8 kg/day more milk with 0.22 percentage units higher milk fat resulting in 1.6 kg/day more 4 percent FCM. When a grass and legume silage- or hay-based diet was fed, results were inconsistent with sodium bicarbonate feeding. Little response in production to sodium bicarbonate was obtained when feeding cottonseed hull-containing diets.

In general it is recommended that buffers will be of greatest benefit to the cow: 1) during early lactation; 2) when large amounts of rapidly fermentable carbohydrates are fed; 3) when cows are fed at infrequent intervals; 4) when fermented forage, primarily corn silage is the major forage source; 5) when concentrates and forages are fed separately; 6) when particle size of the total dietary DM has been reduced to the extent that chewing activity is reduced; 7) when milk fat content is low and when low dry matter intake problems are encountered (Davis and Clark, 1983); and 8) when NDF of the ration is below minimum recommendations (Chapter 4). It is recommended that buffers be fed at 0.6 to 0.8 percent of DMI or 1.2 to 1.6 percent of a concentrate mixture.

Lack of a strong relationship between feeding buffers and metabolic or physiologic variables of acid-base status does not support the idea that these compounds function as ruminal or metabolic buffers. Russell and Chow (1993) concluded that physiologically it was unlikely that dietary buffers could have much of an effect on ruminal fluid pH relative to the predominant effect of transfer of $CO_2$ from blood. They proposed that the mechanism of action of sodium bicarbonate is to increase water consumption, increase dilution of ruminal fluid, and therefore increase the amount of starch that escapes fermentation. Kohn and Dunlap (1998) however demonstrated that when the impact of ruminal bicarbonate under carbon dioxide pressure is considered, pH is predicted to decrease from dilution rather than increase. Dilution would result in an increase in the effective volume of the liquid. Additional liquid in the rumen enables more carbon dioxide to be converted to bicarbonate with the release of more protons, thereby pH would be reduced by dilution with water. These researchers indicate that the effect of sodium bicarbonate on raising ruminal pH may not completely be explained by dilution effects alone.

### *Ionophores*

Ionophores are antibiotics produced by a variety of actinomycetes, most often Streptomyces spp. Ionophores alter the flux of ions across biological membranes. Gram negative bacteria contain a complex outer membrane and are usually unaffected by ionophores. Gram positive bacteria lack the outer membrane and are more sensitive to iono-

phores. Addition of ionophores to the diet decreases the proportion of gram positive bacteria and increases the proportion of gram negative bacteria. As a result, there is a shift in fermentation end-products. Methane production is decreased and the molar proportion of acetate and butyrate are decreased while the molar proportion of propionate is increased. Propionate production may increase 50 to 75 percent depending on the basal diet (Van Maanen et al., 1978) and methane production may be reduced by 30 percent (Schelling, 1984; Mackintosh, et al., 1997). Consequently, the net energy content of feeds is increased when ionophores are fed. Several ionophores have been approved for cattle in the United States. At the time of this writing, ionophores had not been approved for lactating cattle in the United States. Claims for use in nonlactating cattle include increased feed efficiency, prevention of coccidiosis, and increased rate of gain. In countries where ionophores have been approved for lactating cattle (e.g., Australia, Mexico, and Brazil) claims include increased milk production, prevention of ketosis, bloat reduction, and increased milk protein production.

Lasalocid and monensin are approved for prevention and control of coccidiosis. Reduction in the severity of coccidiosis was demonstrated when ionophores were fed to calves that were naturally (Heinrichs and Bush, 1991) or experimentally (Quigley et al., 1997) exposed to coccidia oocysts. Young calves (<4 weeks of age) are at risk of infection, but low DMI of calf starter may preclude consumption of sufficient ionophore to control coccidiosis. Inclusion of ionophore in milk or milk replacer may provide greater control than feeding in calf starters only (Eicher-Pruiett et al., 1992; McMeniman and Elliot, 1995; Quigley et al., 1997). Body weight gains have been improved by the inclusion of ionophores in milk or milk replacer when calves were experimentally exposed to coccidia (Quigley et al., 1997) or when coccidia were not present (Ilan et al., 1981). Eicher-Pruiett et al. (1992) indicated that lasalocid is most effective when delivered at greater than 1 mg/kg body weight.

Almost all of the growth studies with ionophores have been done with beef cattle. Feeding ionophores typically increases the efficiency of feed utilization. In general, when high-concentrate diets were fed, feed intake was decreased and average daily gain was not altered. In contrast when high forage diets were fed, feed intake was not affected but rate of gain was increased (National Research Council, 1996). Very few studies have examined the effects of ionophores on growth of dairy heifers. Feed intake was not significantly reduced by ionophore supplementation (Baile et al., 1982; Meinert et al., 1992; Steen et al., 1992). Average daily gain or efficiency of feed utilization by heifers has been increased by ionophore supplementation, but the differences have not always been significant (Bartley et al., 1979; Baile et al., 1982; Meinert et al., 1992; Chester-Jones et al., 1997). Studies utilizing large numbers of animals will be required to detect quantitative growth responses by dairy heifers to ionophores. Ionophore supplementation may improve reproductive performance of growing heifers. A reduction in days to first estrus (Snyder et al., 1981), days to conception (Baile et al., 1982), or age at first breeding and age at first calving (Meinert et al., 1992) have been reported. Ionophore supplementation had no effects on first lactation milk yield (Baile et al., 1982).

Ionophore effects on ruminal fermentation may influence lactation performance. Increasing propionate production at the expense of acetate, butyrate, and methane will increase energy that is potentially available for milk synthesis. Increased propionate production may enhance glucose synthesis by the animal, which could influence milk production directly by providing a precursor for the synthesis of lactose. Indirect effects of additional glucose production include sparing of amino acids for gluconeogenesis and alteration of hormonal status, which could influence the partitioning of nutrients and milk components. Milk yield is often increased during ionophore supplementation; as much as 3 kg/d when cows received pasture (Moshen et al., 1981; Lean and Wade, 1997; Beckett et al., 1998; Van Der Werf et al., 1998). Pasture fed cows may benefit from bloat reduction. Duffield et al. (1997) indicated an interaction between body condition score and milk production; milk yield responses to ionophore supplementation were greater as body condition score was increased. Milk yield responses of Holsteins may be greater than Jerseys, and Holsteins with high breeding value for milk protein and fat production may be more responsive than those with low breeding value (Van Der Werf et al., 1998). Milk fat percentage is usually decreased by 0.1 percentage units or more (Kennelly and Lien, 1997) and the response in milk protein percentage is variable (Kennelly and Lien, 1997). The effect of ionophore to depress milk fat was alleviated as forage to concentrate ratio was increased (Phipps et al., 1995). Potential mechanisms for depressed milk fat percentage include less acetate and butyrate for fatty acid synthesis, endocrine changes resulting in the partitioning of nutrients away from mammary tissue, or depressed biohydrogenation resulting in greater production of TFA (Fellner et al., 1997).

Effects of ionophores on DMI have been variable; most studies indicate no effects or a decrease in intake (Johnson et al., 1988; Sauer et al., 1989; Weiss and Amiet, 1990). A preliminary report (Symanowski et al., 1999) from a large multi-university trial employing 858 cows and examining 0, 8, 16, and 24 mg/kg dietary monensin indicated that DMI is modestly decreased (< 1 kg/d), solids-corrected milk yield is modestly increased (< 1 kg/d), and efficiency of solids-corrected milk production is increased. The same study indicated that cows fed monensin lost less body condition during early lactation and maintained a higher body

condition score (Wagner et al., 1999). Reductions in DMI and greater body weight gain during mid to late lactation might be expected if cows are in positive energy balance, and ionophores cause an increase in the NE content of the diet. Knowlton et al. (1996a) observed a slight increase in DMI when feeding lasalocid. Ionophores could have a positive influence on DMI if cows are fed high concentrate diets and lactate production in the rumen is decreased (Nagaraja et al., 1981); however, lactate concentration was increased in the study of Knowlton et al. (1996b).

Reproductive performance of lactating cows grazing pasture was not improved by ionophore supplementation in two large field trials (Abe et al., 1994; Lean et al., 1994; Hayes et al., 1996). Phipps et al. (1997b) indicated that reproductive performance was not improved during the first lactation but was improved when cows were fed ionophores for a second lactation.

Feeding ionophores may improve animal health. Increased propionate production and gluconeogenesis may spare amino acid catabolism and reduce fat mobilization from adipose tissue and ketone production by the liver. An increase in plasma glucose, decrease in plasma nonesterified fatty acids, decrease in blood beta-hydroxybutyrate, or combinations of the above have been attributed to ionophore feeding on several occasions (Sauer et al., 1989; Lean and Wade, 1997; Phipps et al., 1997a; Duffield et al., 1998a; Green et al., 1999). Lower nonesterified fatty acids and beta-hydroxybutyrate in blood probably reflect less body condition loss when feeding ionophores (Knowlton et al., 1996a; Erasmus et al., 1997; Wagner et al., 1999). The prevalence and incidence of subclinical ketosis was reduced by 50 percent when monensin was delivered by a sustained-release intraruminal device beginning at 3 weeks precalving (Duffield et al., 1998b). A lower incidence of bloat when feeding ionophores (Lowe et al., 1991) is probably attributed to less gas production. As previously indicated, ionophores may have a role in the prevention of subclinical acidosis by reducing lactate formation in the rumen and stabilizing rumen pH.

### *Direct Fed Microbials*

Direct fed microbials (DFM), traditionally referred to as "probiotics" are live or viable naturally occurring organisms supplemented to animals. Direct fed microbials have generally been supplemented to animals during periods of stress or low DMI with the assumption that establishment of a beneficial microorganism population in the digestive tract will decrease or prevent pathogenic organism establishment. The DFM have been fed continuously to attempt to enhance production performance, alter ruminal fermentation, or improve nutrient utilization. The most common DFM are fungal cultures (*Aspergillus oryzae* and *Saccharomyces cerevisiae*), and the lactic acid bacteria *Lactobacillus* or *Streptococcus*. Other bacterial species such as *Bifidobacterium* spp., *Bacillus* spp., and *Propionibacterium* spp. are found in DFM, but to a lesser extent than lactic acid bacteria. Yoon and Stern (1995) in a review found that multiple modes of action have been proposed in which DFM may elicit responses, but none are clearly understood or well defined. They categorized mode of actions into the following:

- stimulation of desirable microbial growth in the rumen,
- stabilization of rumen pH,
- altered ruminal fermentation pattern and end product production,
- increased nutrient flow postruminally,
- increased nutrient digestibility, and
- alleviation of stress through enhanced immune response.

### *Fungal Cultures*

Production responses to the addition of fungal cultures to diets of lactating dairy cows have been variable. Yoon and Stern (1995) reported significant increases in DMI in 2 of 10 studies and significant increases in milk production in 3 of 11 studies with supplementation of *S. cerevisiae*. In more recent studies, supplementation of *S. cerevisiae* increased DMI and milk production in three studies (Adams et al., 1995; Putman et al., 1997; Wohlt et al., 1998), but not in two others (Robinson, 1997; Kung et al., 1997). *Aspergillus oryzae* increased DMI in 1 of 8 studies and milk production in 6 of 14 studies summarized by Yoon and Stern (1995). In more recent studies with supplementation of *A. oryzae* to lactating cow diets, no increase in milk production was reported in one study (Bertrand and Grimes, 1997) and mixed, but an overall positive increase in milk production was reported in 46 commercial dairy herds (McGilliard and Stallings, 1998).

Stimulation of the growth and activities of both total and certain specific groups of ruminal bacteria have been the most consistent reproducible modes of action for fungal cultures (Yoon and Stern, 1995, 1996; Beharka and Nagaraja, 1998; Newbold et al., 1996). Cellulose digesting and lactic acid utilizing bacteria are the most commonly enhanced ruminal bacteria groups by fungal supplementation (Callaway and Martin, 1997). Why and how fungal cultures increase bacterial numbers is not understood, but one proposed mechanism is that the respiratory activity of yeast protects anaerobic rumen bacteria from damage by oxygen (Newbold et al., 1996).

Dietary composition and forage source are significant factors affecting production responses to fungal cultures. High concentrate diets (60:40 concentrate to forage ratio) resulted in greater milk production response to fungal cul-

ture supplementation than lower concentrate diets (Williams et al., 1991), and ruminal digestion of NDF in alfalfa was increased more than that of NDF in corn silage or other sources of NDF by fungal culture supplementation (Miranda et al., 1996; Adams et al., 1995). Total volatile fatty acid (VFA) production or ratios of VFA are generally not affected by additions of fungal cultures (Yoon and Stern, 1995, 1996; Beharka and Nagaraja, 1998). Passage of essential amino acids or the ratio of microbial to feed nitrogen that passed to the small intestine was not increased with yeast supplementation (Putman et al., 1997) nor was overall total tract digestibility (Yoon and Stern, 1995).

#### LACTOBACILLUS

Considerably less research has been conducted to determine the effects of lactic acid bacteria on production responses or ruminal fermentation changes than with fungal cultures. Supplementation of lactic acid bacteria to diets has primarily been for a "probiotic" effect where ingestion of beneficial organisms colonize the intestinal tract preventing pathogen proliferation, compete with enterotoxin-producing organisms for absorption sites in the intestine, and possibly enhance digestion of nutrients in the small intestine (Yoon and Stern, 1995). In the review by Yoon and Stern (1995), only two studies were found where *Lactobacillus acidophilus* was fed to lactating dairy cattle. In both studies, milk production increased by feeding *L. acidophilus*. Cruywagen et al. (1996) reported supplementing *L. acidophilus* in milk replacer resulted in calves losing less weight the initial two weeks of life, but over a six-week period had no affect on weight gain, feed intake, or diarrhea occurrence. The addition of *L. acidophilus* or *Bifidobacterium animalis* to a milk replacer containing an antibiotic increased growth rate and efficiency of feed utilization by calves during the milk replacer feeding period (first 35 days of life) and the next 21 days postweaning (Abe et al., 1995).

### *Bovine Somatotropin*

Bovine somatotropin (BST) is a naturally-occurring protein hormone produced in the pituitary gland of dairy cattle. It is a major regulator of growth and milk production. This hormone can be produced in commercial quantity using recombinant DNA technology. BST was approved for use in lactating dairy cows by the Food and Drug Administration in November 1993. Because of a 90-day moratorium passed by the U.S. Congress, BST could not be sold for commercial use until February 1994.

Supplementation of BST to growing and lactating animals affects many physiologic processes (Peel and Bauman, 1987; Bauman et al., 1989a; National Research Council, 1994). Metabolic adaptations that partition increased quantities of absorbed nutrients to the required tissue for optimum growth or milk production is the principle effect of BST in growing and lactating dairy cattle. Supplementation of BST to growing or lactating dairy cattle does not affect digestibilities of DM, energy, or protein (Bauman et al., 1989a; Boyd and Bauman, 1989; Chalupa and Galligan, 1989) nor does BST affect energy utilization for maintenance or the partial efficiency of milk synthesis (Tyrrell et al., 1988; Sechen et al., 1989; Kirchgessner et al., 1991). However, the efficiency of overall nutrient utilization for milk production by cows is improved because a smaller proportion of the nutrient intake is needed to fulfill the maintenance requirements.

The effects of BST on milk yield have been reviewed (Peel and Bauman, 1987; Chilliard, 1989; McBride et al., 1988; Chalupa and Galligan, 1989; Peel et al., 1989; Crooker and Otterby, 1991; Hartnell et al., 1991; McGuffey and Williamson, 1991; Bauman, 1992; National Research Council, 1994; Bauman et al., 1999). Increases in milk yield to varying doses of BST (5 to 50 mg/cow/day) range from about 3 to 6 kg of milk/cow/day (National Research Council, 1994). Persistency of lactation also is improved. Supplementation of BST has increased milk yield in all breeds of dairy cattle studied and in animals of different parity and genetic potential (National Research Council, 1994). The magnitude of the increased milk yield will be affected by the quality of management, especially nutrition management (Bauman, 1987).

Nutritional status, diet composition, environment, season, stage of lactation, genetics, and age affect the concentration of fat and protein in milk (Linn, 1988; Sutton, 1989). These factors also affect the composition of milk from cows supplemented with BST. The nutritional status of cows both before and during supplementation of BST determines the effect of BST on the concentration of fat and protein in milk (Peel and Bauman, 1987; McBride et al., 1988; Bauman et al., 1989a; Chalupa and Galligan, 1989; van den Berg, 1991; Dell'Orto and Savoini, 1991; Barbano et al., 1992; Lynch et al., 1992; Laurent et al., 1992). Short-term changes in milk composition when BST is supplemented may occur because of increased milk synthesis and because of increased mobilization of energy and protein from body reserves to meet the increased nutrient demands for synthesis of milk and milk components. However, when BST was supplemented for a complete lactation the concentration of fat and protein in milk was not different for control and BST cows (Bauman et al., 1989b). BST did not affect milk composition during long-term supplementation, because cows, within a few weeks after the start of BST administration, increased nutrient intake to meet requirements for synthesis of milk and milk components and to replenish body reserves (Peel and Bauman, 1987; Chalupa and Galligan, 1989; Chilliard, 1989). High quality feeds and excellent nutrition management are required to

attain maximum response from cows supplemented with BST (Bauman, 1987, 1992).

Nutrition of dairy cows supplemented with BST has been discussed in several papers (Bauman, 1987; Chalupa and Galligan, 1989; Chilliard, 1989; Crooker and Otterby, 1991; Kirchgessner et al., 1991; McGuffey and Wilkinson, 1991; Muller, 1992; Collier et al., 1992; National Research Council, 1994). Nutrient requirements are identical for BST supplemented cows and unsupplemented cows if they are producing the same amount of milk with an identical composition, have the same body size and weight, and are losing or gaining the same body weight. Diet formulation and feeding strategies should be the same for BST supplemented and unsupplemented cows of the same size and weight that are producing the same amount of milk and milk components. Current recommendations are that cows supplemented with BST should be fed and managed like unsupplemented cows at similar levels of production.

## REFERENCES


Abe, F., N. Ishibashi, and S. Shimamura. 1995. Effect of administration of bifidobacteria and lactic acid bacteria to newborn calves and piglets. J. Dairy Sci. 78:2838–2845.

Abe, N., I. J. Lean, A. Rabiee, J. Porter, and C. Graham. 1994. Effects of sodium monensin on reproductive performance of dairy cattle. II. Effects on metabolites in plasma, resumption of ovarian cyclicity and oestrus in lactating cows. Aust. Vet. J. 71:277–282.

Adams, A. L., B. Harris, Jr., H. H. Van Horn, and C. J. Wilcox. 1995. Effects of varying forage types on milk production responses to whole cottonseed, tallow and yeast. J. Dairy Sci. 78:573–581.

Al-Ani, F. K., and J. G. E. Vesteweber. 1986. Udder edema: An updated review. Vet. Bull. 56:763–769.

Allen, M. S. 1997. Relationship between fermentation acid production in the rumen and the requirements for physically effective fiber. J. Dairy. Sci. 80:1447–1462.

Allen, W. M., and D. C. Davies. 1981. Milk fever, hypomagnesaemia, and the 'downer cow' syndrome. Br. Vet. J. 137:435–441.

Anderson, K. L., T. G. Nagaraja, J. L. Morrill, P. G. Reddy, T. B. Avery, and N. V. Anderson. 1988. Performance and ruminal changes of early-weaned calves fed lasalocid. J. Anim. Sci. 66:806–813.

Armentano, L., and M. Pereira. 1997. Measuring the effectiveness of fiber by animal response trials. J. Dairy Sci. 80:1416–1425.

Aslam, M., W. B. Tucker, J. F. Hogue, R. K. Vernon, and G. D. Adams. 1991. Controlled ruminal infusion of sodium bicarbonate. 2. Effects of dietary and infused buffer on ruminal milieu. J. Dairy Sci. 74:3496–3504.

Baile, C. A., C. L. McLauglin, W. V. Chalupa, D. L. Snyder, L. C. Pendlum, and E. L. Potter. 1982. Effects of monensin fed to replacement dairy heifers during the growing and gestation period upon growth, reproduction, and subsequent lactation. J. Dairy Sci. 65:1941–1944.

Bar, A., R. Perlman, and M. Sachs. 1985. Observation on the use of 1-alpha-hydroxyvitamin D3 in the prevention of bovine parturient paresis: The effect of a single injection on plasma 1-alpha-hydroxyvitamin D3, 1,25-dihydroxyvitamin D3, calcium, and hydroxyproline. J. Dairy Sci. 68:1952–1958.

Barbano, D. M., J. M. Lynch, D. E. Bauman, G. F. Hartnell, R. L. Hintz, and M. A. Nemeth. 1992. Effect of a prolonged-release formulation of N-methionyl bovine somatotropin (sometribove) on milk composition. J. Dairy Sci. 75:1775–1793.

Barber, D. M., C. L. Wright, and W. MacLennan. 1983. Hypomagnesaemia in periparturient dairy cows. Vet. Rec. 112:35–36.

Bartley, E. E., E. L. Herod, R. M. Bechtle, D. A. Sapienza, and B. E. Brent. 1979. Effect of monensin or lasalocid, with and without niacin or amicloral, on rumen fermentation and feed efficiency. J. Anim. Sci. 49:1066–1075.

Barton, B. A., N. A. Jorgensen, and H. F. DeLuca. 1987. Impact of prepartum dietary phosphorus intake on calcium homeostasis at parturition. J. Dairy Sci. 70:1186–91.

Bauchart, D., D. Gruffat, and D. Durand. 1996. Lipid absorption and hepatic lipid metabolism. Proc. Nutr. Soc. 55:39–47.

Bauman, D. E. 1987. Bovine somatotropin: The Cornell experience. Pp. 46 in National Invitational Workshop on Bovine Somatotropin. Washington, D.C.: U.S. Department of Agriculture Extension Service.

Bauman, D. E. 1992. Bovine somatotropin: Review of an emerging animal technology. J. Dairy Sci. 75:3432–3451.

Bauman, D. E., B. A. Corl, and L. H. Baumgard. 1998. Trans fatty acids, conjugated linoleic acid and milk fat synthesis. Proceeding 1998 Cornell Nutrition Conference. p. 95.

Bauman, D. E., F. R. Dunshea, Y. R. Biosclair, M. A. McGuire, D. M. Harris, and K. L. Houseknecht. 1989a. Regulation of nutrient partitioning: Homeostasis, homeorhesis and exogenous somatotropin. Pp. 306–323 in Seventh International Conference on Production Diseases in Farm Animals, F. A. Kallfelz, ed. Ithaca, NY: Cornell University.

Bauman, D. E., R. W. Everett, W. H. Weiland, and R. J. Collier. 1999. Production responses to bovine somatotropin in northeast dairy herds. J. Dairy Sci. 82:2564–2573.

Bauman, D. E., D. L. Hard, B. A. Crooker, M. S. Partridge, K. Garrick, L. D. Sandles, H. N. Erb, S. E. Franson, G. F. Hartnell, and R. L. Hintz. 1989b. Long-term evaluation of prolonged-release formulation of N-methionyl bovine somatotropin in lactating dairy cows. J. Dairy Sci. 72:642–651.

Baumgard, L. H., B. A. Corl, D. A. Dwyer, A. Saeb, and D. E. Bauman. 2000. Identification of the conjugated linoleic acid isomer that inhibits milk fat synthesis. Am. J. Physiol. 278:R179–184.

Beckett, S., I. Lean, R. Dyson, W. Tranter, and L. Wade. 1998. Effects of monensin on the reproduction, health, and milk production of dairy cows. J. Dairy Sci. 81:1563–1573.

Beede, D. K. 1992. Dietary cation-anion difference: Preventing milk fever. Feed Management 43:28–31.

Beede, D. K., C. Wang, G. A. Donovan, L. F. Archbald, and W. K. Sanchez. 1991. Dietary cation-anion difference (electrolyte balance) in late pregnancy. Florida Dairy Production Conf. Proc., April 10, 1991. pp. 1–6.

Beharka, A. A., and T. G. Nagaraja. 1998. Effect of *Aspergillus oryzae* extract alone or in combination with antimicrobial compounds on ruminal bacteria. J. Dairy Sci. 81:1591–1598.

Bell, A. W. 1979. Lipid metabolism in the liver and selected tissues and in the whole body of ruminant animals. Prog. Lipid Res. 18:117–164.

Bell, A. W. 1995. Regulation of organic nutrient metabolism during transition from late pregnancy to early lactation. J. Anim. Sci. 73:2804–2819.

Bell, A. W., G. Slepetis, and R. A. Ehrhardt. 1995. Growth and accretion of energy and protein in the gravid uterus during late pregnancy in Holstein cows. J. Dairy Sci. 78:1954–1961.

Bergsten, C. 1994. Hemorrhages of the sole horn of dairy cows as a retrospective indicator of laminitis: An epidemiological study. Acta Vet. Scand. 35:55–66.

Bergsten, C. 1995. Digital disorders in dairy cattle with special reference to laminitis and heel erosion: The influence of housing, management and nutrition. Dissertation. Skara, Sweden.

Bertics, S. J., R. R. Grummer, C. Cadorniga-Valino, and E. E. Stoddard. 1992. Effect of prepartum dry matter intake on liver triglyceride concentration and early lactation. J. Dairy Sci. 75:1914–1922.

Bertrand, J. A., and L. W. Grimes. 1997. Influence of tallow and *Aspergillus oryzae* fermentation extract in dairy cattle rations. J. Dairy Sci. 80:1179–1184.

Block, E. 1984. Manipulating dietary anions and cations for prepartum dairy cows to reduce incidence of milk fever. J. Dairy Sci. 67:2939–2948.

Block, E. 1994. Manipulation of dietary cation-anion difference on nutritionally related production diseases, productivity and metabolic responses of dairy cows. J. Dairy Sci. 77:1437–1450

Boda, J. M., and H. H. Cole. 1954. The influence of dietary calcium and phosphorus on the incidence of milk fever in dairy cattle. J. Dairy Sci. 37:360–372.

Boyd, R. D., and D. E. Bauman. 1989. Mechanisms of action for somatotropin in growth. Pp. 257–293 in Current Concepts of Animal Regulation, D. R. Campion, G. J. Hausman, and R. J. Martin, eds. New York: Plenum.

Breukink, H. J. 1991. Abomasal displacement, etiology, pathogenesis, treatment and prevention. Bovine Practitioner 26:148–153.

Breukink, H. J., and T. de Ruyter. 1976. Abomasal displacement in cattle: Influence of concentrates in the ration on fatty acid concentrations in ruminal, abomasal, and duodenal contents. Am. J. Vet. Res. 37:1181–1184.

Brzezinska-Slebodzinska, E., and J. K. Miller. 1992. Antioxidant status of dairy cows supplemented prepartum with vitamin E and selenium. Fed. Am. Soc. Exp. Biol. J. 6:A1953 (Abstr.).

Callaway, E. S., and S. A. Martin. 1997. Effects of a *Saccharomyces cerevisiae* culture on ruminal bacteria that utilize lactate and digest cellulose. J. Dairy Sci. 80:2035–2044.

Cameron, R. E., 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.

Campling, R. C., M. Freer, and C. C. Balch. 1962. Factors affecting the voluntary intake of food by cows. Brit. J. Nutr. 16:115–124.

Capuco, A. V., R. M. Akers, and J. J. Smith. 1997. Mammary growth in Holstein cows during the dry period: Quantification of nucleic acids and histology. J. Dairy Sci. 80:447–487.

Cassida, K. A., L. D. Muller, and T. F. Sweeney. 1988. Sodium sesquicarbonate for early lactation dairy cows fed corn silage-based diets. J. Dairy Sci. 71:381–387.

Chalupa, W., and D.T. Galligan. 1989. Nutritional implications of somatotropin for lactating cows. J. Dairy Sci. 72:2510–2524.

Chalupa, W., C. J. Sniffen, W. E. Julien, H. Sato, T. Fujieda, T. Ueda, and H. Suzuki. 1999. Lactation response of cows in a commercial dairy to rumen protected methionine and lysine. J. Dairy Sci. 82(Suppl. 1): 121. (Abstr.).

Chester-Jones, H., J. G. Linn, D. M. Ziegler, and M. Engstrom. 1997. Determination of the caloric equivalency of an ionophore in dairy heifer diets. J. Dairy Sci. 80(Suppl. 1):216 (Abstr.).

Chew, B. P., F. R. Murdock, R. E. Riley, and J. K. Hillers. 1984. Influence of prepartum dietary crude protein on growth hormone, insulin, reproduction, and lactation of dairy cows. J. Dairy Sci 67:270–275.

Chew, B. P., H. F. Keller, R. E. Erb, and P. V. Malven. 1977. Periparturient concentrations of prolactin, progesterone, and estrogen in blood plasma of cows retaining or not retaining fetal membranes. J. Anim. Sci. 44:1055–1060.

Chew, B. P., R. E. Erb, J. F. Fessler, C. J. Callahan, and P. V. Malven. 1979. Effects of ovariectomy during pregnancy and of prematurely induced parturition on progesterone, estrogens, and calving traits. J. Dairy Sci. 62:557–566.

Chilliard, Y. 1989. Long-term effects of recombinant bovine somatotropin (rbST) on dairy cow performances: A review. Pp. 61–87 in Use of Somatotropin in Livestock Production, K. Sejrsen, M. Vestergaard, and A. Neimann-Sorensen, eds. New York: Elsevier Applied Science.

Chouinard, P. Y., L. Corneau, A. Sb, and D. E. Bauman. 1999a. Milk yield and composition during abomasal infusion of conjugated linoleic acid. J. Dairy Sci. 82:2737–2745.

Chouinard, P. Y., L. Corneau, D. M. Barbano, I. E. Metzger, and D. E. Bauman. 1999b. Conjugated linoleic acids alter milk fatty acid composition and inhibit milk fat secretion in dairy cows. J. Nutr. 129:1579–1584.

Christie, W. W. 1981. The effects of diet and other factors on the lipid composition of ruminant tissues and milk. *In* Lipid metabolism in ruminant animals. Ed. W. W. Christie, Pergamon Press.

Collier, R. J., J. L. Vicini, C. D. Knight, C. L. McLaughlin, and C. A. Baile. 1992. Impact of somatotropins on nutrient requirements in domestic animals. J. Nutr. 122:855–866.

Conway, J. F., H. H. Olson, and G. C. McGoy. 1977. Effects of sodium chloride supplementation on the incidence and severity of mammary edema and on serum sodium levels in pre-parturient cows and heifer. J. Dairy Sci. 60(Suppl.1):110 (Abstr.).

Coppock, C. E., C. H. Noller, S. A. Wolfe, C. J. Callahan, and J. S. Baker. 1972. Effect of forage-concentrate ratio in complete feeds fed ad libitum on feed intake prepartum and the occurrence of abomasal displacement in dairy cows. J. Dairy Sci. 55:783–789.

Cox, V. S. 1998. Downer cow syndrome. Pp. 215–218 in Current Veterinary Therapy 4: Food Animal Practice, J. L. Howard and R. A. Smith, eds. Philadelphia: W.B. Saunders Co.

Craige, A. H., and I.V. Stoll. 1947. Milk fever (parturient paresis) as a manifestation of alkalosis. Am. J. Vet. Res. 8:168–172.

Crawley, D. D., and L. H. Kilmer. 1995. Effects of level and source of rumen degradable protein fed prepartum on postpartum performance of dairy cattle. J. Dairy Sci. 78(Suppl.1):266 (Abstr.).

Crooker, B. A., and D. E. Otterby. 1991. Management of the dairy herd treated with bovine somatotropin. Food Anim. Prac. 7:417–437.

Croom, W. J., A. H. Rakes, A. C. Linnerud, G. A. Ducharme, and J. M. Elliot. 1981. Vitamin $B_{12}$ administration for milk fat synthesis in lactating dairy cows fed a low fiber diet. J. Dairy Sci. 64:1555–1560.

Cruywagen, C. W., I. Jordaan, and L. Venter. 1996. Effect of *Lactobacillus acidophilus* supplementation of milk replacer on preweaning performance of calves. J. Dairy Sci. 79:483–486.

Curtis, C. R., H. N. Erb, C. J. Sniffen, R. D. Smith, and D. S. Kronfeld. 1985. Path analysis of dry period nutrition, postpartum metabolic and reproductive disorders, and mastitis in Holstein cows. J. Dairy Sci. 68:2347–2360.

Curtis, C. R., H. N. Erb, C. J. Sniffen, R. D. Smith, P. A. Powers, M. C. Smith, M. E. White, R. B. Hillman, and E. J. Pearson. 1983. Association of parturient hypocalcemia with eight periparturient disorders in Holstein cows. JAVMA 183:559–561.

Daniel, R. C. W. 1983. Motility of the rumen and abomasum during hypocalcaemia. Can. J. Comp. Med. 47:276–280.

Davis, C. L. 1979. The use of buffers in the rations of lactating cows. Pp. 51–64 in Regulation of Acid-Base Balance, W. H. Hale and P. Meinhardt, eds. Church and Dwight, Co. Inc.

Davis, C. L., and J. H. Clark. 1983. Response of dairy cattle to buffers. Proc. Buffers, Neutralizers and Electrolytes Symp. West Des Moines, Iowa: National Feed Ingredients Association.

Dell'Orto, V., and G. Savoini. 1991. Recombinant bovine somatotropin (rbST) treatment in dairy cows: Effect on ruminal activity and milk properties. Microbiol. Aliments Nutr. 9:121–132.

Dentine, M. R., and B. T. McDaniel. 1984. Association of subjective udder edema scores and description trait codes for udder types. J. Dairy Sci. 67:208–215.

DePeters, E. J., A. H. Fredeen, D. L. Bath, and N. E. Smith. 1984. Effect of sodium bicarbonate addition to alfalfa hay-based diets on digestibility of dietary fractions and rumen characteristics. J. Dairy Sci. 67:2344–2355.

Drackley, J. K. 1999. Biology of dairy cows during the transition period: the final frontier? J. Dairy Sci. 82:2259–2273.

Dirksen, G. U., H. G. Liebich, and E. Mayer. 1985. Adaptive changes of the ruminal mucosa and their functional and clinical significance. Bovine Pract. 20:116–120.

Duffield, T. F., D. Sandals, K. E. Leslie, K. Lissemore, B. W. McBride, J. H. Lumsden, P. Dick, and R. Bagg. 1998a. Effect of prepartum administration of monensin in a controlled-release capsule on postpartum energy indicators in lactating cows. J. Dairy Sci. 81:2354–2361.

Duffield, T. F., D. Sandals, K. E. Leslie, K. Lissemore, B. W. McBride, J. H. Lumsden, P. Dick, and R. Bagg. 1998b. Efficacy of monensin for the prevention of subclinical ketosis in lactating dairy cows. J. Dairy Sci. 81:2866–2873.

Duffield, T. F., K. E. Leslie, D. Sandals, K. Lissemore, B. McBride, and J. H. Lumsden. 1997. Effect of monensin on cow health and milk production. J. Dairy Sci. 80(Suppl. 1):167 (Abstr.).

Dufva, G. S., E. E. Bartley, A. D. Dayton, and D. O. Riddell. 1983. Effect of niacin supplementation on milk production and ketosis of dairy cattle. J. Dairy Sci. 66:2329–2336.

Edgerton, L. A., and H. D. Hafs. 1973. Serum luteinizing hormone, prolactin, glucocorticoid, and progestin in dairy cows from calving to gestation. J. Dairy Sci. 56:451–458.

Eger, S., D. Drori, I. Kadoori, N. Miller, and H. Schindler. 1985. Effects of selenium and vitamin E on incidence of retained placenta. J. Dairy Sci. 68:2119–2122.

Eicher-Pruiett, S. D., J. L. Morrill, T. G. Nagaraja, J. J. Higgins, N. V. Anderson, and P. G. Reddy. 1992. Response of young dairy calves with lasalocid delivery varied in feed sources. J. Dairy Sci. 75:857–862.

Emery, R. S. 1976. High energy feeds for milk production. Pp. 149–159 in Buffers in Ruminant Physiology and Metabolism, M. S. Weinberg, and A. L. Sheffner, eds. Church and Dwight Co., Inc.

Emery, R. S. and L. D. Brown. 1961. Effect of feeding sodium and potassium bicarbonate on milk fat, rumen pH, and volatile fatty acid production. J. Dairy Sci. 44:1899–1902.

Emery, R. S., L. D. Brown, and J. W. Thomas. 1964. Effect of sodium bicarbonate and calcium carbonates on milk production and composition of milk, blood, and rumen contents of cows fed grain ad libitum with restricted roughage. J. Dairy Sci. 47:1325.

Emery, R. S., H. D. Hafs, D. Armstrong, and W. W. Snyder. 1969. Prepartum grain feeding effects on milk production, mammary edema, and incidence of diseases. J. Dairy Sci. 52:345–351.

Emery, R. S., J. S. Liesman, and T. H. Herdt. 1992. Metabolism of long chain fatty acids by rumimant liver. J. Nutr. 122:832–837.

Ender, F., and I. W. Dishington. 1967. Comparative studies on calcium balance levels in parturient cows fed diets inducing and preventing milk fever. Pp. 557A in XVIII$^{th}$ World Veterinary Congress, Vol. II. Paris.

Ender, F., I. W. Dishington, and A. Helgebostad. 1971. Calcium balance studies in dairy cows under experimental induction and prevention of hypocalcaemic paresis puerperalis. The solution of the aetiology and the prevention of milk fever by dietary means. Z. Tierphysiol. 28:233–256.

Erasmus, L. J., A. Muller, I. Smith, and D. O'Hagan. 1997. Effect of lasalocid on performance of lactating dairy cows. J. Dairy Sci. 80(Suppl. 1):208 (Abstr.).

Erb, H. N., and Y. T. Grohn. 1988. Epidemiology of metabolic disorders in the periparturient dairy cow. J. Dairy Sci. 71:2557–2571.

Erb, H. N., R. D. Smith, P. A. Oltenaw, C. L. Guard, R. B. Hillman, P. A. Powers, M. C. Smith, and M. E. White. 1985. Path model of reproductive disorders and performance, milk fever, mastitis, milk yield, and culling in Holstein cows. J. Dairy Sci. 68:3337–3349.

Erdman, R. A. 1988. Dietary buffering requirements of the lactating dairy cow. J. Dairy Sci. 71:3246–3266.

Erdman, R. A., R. W. Hemken, and L. S. Bull. 1982. Dietary sodium bicarbonate and magnesium oxide for early post partum lactating dairy cows: effects on production, acid-base metabolism, and digestion. J. Dairy Sci. 65:712–731.

Erdman, R. A., R. L. Botts, R. W. Hemken, and L. S. Bull. 1980. Effect of sodium bicarbonate and magnesium oxide on production and physiology in early lactation. J. Dairy Sci. 63:923–930.

Fellner, V., F. D. Sauer, and J. K. G. Kramer. 1997. Effect of nigericin, monensin, and tetronasin on biohydrogenation in continuous flow-through ruminal fermenters. J. Dairy Sci. 80:921–928.

Ferrell, C. L., W. N. Garrett, and N. Hinman. 1976. Growth, development and composition of the udder and gravid uterus of beef heifers during pregnancy. J. Anim. Sci. 42:1477–1489.

Ferrell, C. L., D. B. Laster, and R. L. Prior. 1982. Mineral accretion during prenatal growth of cattle. J. Anim. Sci. 54:618–624.

Ferrell, C. L., W. N. Garrett, N. Hinman, and G. Grichting. 1976. Energy utilization by pregnant and nonpregnant heifers. J. Anim. Sci. 42:937–950.

Firkins, J. L. 1997. Effects of feeding nonforage fiber sources on site of fiber digestion. J. Dairy Sci. 80:1426–1437.

Food and Drug Administration. 1997. Food additives permitted in feed and drinking water of animals; selenium. Federal Register (Aug. 25) 62:44892–44894.

Fountaine, F. C., D. B. Parrish, and F. W. Atkeson. 1949. Comparison of the incidence and severity of mammary edema of cows fed roughages alone or roughages plus grain during the dry period. J. Dairy Sci. 32:721 (Abstr.).

Frobish, R. A., and C. L. Davis. 1977. Theory involving propionate and vitamin $B_{12}$ in the low-fat milk syndrome. J. Dairy Sci. 60:268–273.

Gant, R. G., W. Sanchez, and R. L. Kincaid. 1998. Effects of anionic salts on selenium metabolism in nonlactating, pregnant dry cows. J. Dairy Sci. 81:1637–1642.

Gaynor, P. J., F. J. Mueller, J. K. Miller, N. Ramsey, J. P. Goff, and R. L. Horst. 1989. Parturient hypocalcemia in Jersey cows fed alfalfa haylage-based diets with different cation to anion ratios. J. Dairy Sci. 72:2525–2531.

Gaynor, P. J., R. A. Erdman, B. B. Teter, J. Sampugna, A. V. Capuco, D. R. Waldo and M. Hamosh. 1994. Milk fat yield and composition during abomasal infusion of cis or trans octadecenoates in Holstein cows. J. Dairy Sci. 77:157–165.

Goff, J. P. 1998a. Phosphorus deficiency. Pp. 218–220 in Current Veterinary Therapy 4: Food Animal Practice, J. L. Howard and R. A. Smith, eds. Philadelphia: W.B. Saunders Co.

Goff, J. P. 1998b. Ruminant hypomagnesemic tetanies. Pp. 215–218 in Current Veterinary Therapy 4: Food Animal Practice, J. L. Howard and R. A. Smith, eds. Philadelphia: W.B. Saunders Co.

Goff, J. P. 2000. Pathophysiology of calcium and phosphorus disorders. Vet Clin North Am Food Animal Pract. 16:319–337.

Goff, J. P., and R. L. Horst. 1990. Effect of subcutaneously released 24F-1,25-dihydroxyvitamin D3 on incidence of parturient paresis in dairy cows. J. Dairy Sci 73:406–412.

Goff, J. P., and R. L. Horst. 1997a. Effects of the addition of potassium or sodium, but not calcium, to prepartum rations on milk fever in dairy cows. J. Dairy Sci. 80:176–186.

Goff, J. P., and R. L. Horst. 1997b. Physiological changes at parturition and their relationship to metabolic disorders. J. Dairy Sci. 80:1260–1268.

Goff, J. P., and R. L. Horst. 1998. Use of hydrochloric acid as a source of anions for prevention of milk fever. J. Dairy Sci. 81: 2874–2880.

Goff, J. P., and J. R. Stabel. 1990. Decreased plasma retinal, alpha-tocopherol, and zinc concentration during the periparturient period: effect on milk fever. J. Dairy Sci. 73:3195–3199.

Goff, J. P., E. T. Littledike, and R. L. Horst. 1986. Effect of synthetic bovine parathyroid hormone in dairy cows: Prevention of hypocalcemic parturient paresis. J. Dairy Sci. 69:2278–2289.

Goff, J. P., T. A. Reinhardt, and R. L. Horst. 1989. Recurring hypocalcemia of bovine parturient paresis is associated with failure to produce 1,25-dihydroxyvitamin D. Endocrinol. 125:49–53.

Goff, J. P., R. L. Horst, F. J. Mueller, J. K. Miller, G. A. Kiess, and H. H. Dowlen. 1991. Addition of chloride to a prepartal diet high in cations increases 1,25-dihydroxyvitamin D response to hypocalcemia preventing milk fever. J. Dairy Sci. 74:3863–3871.

Goff, J. P., T. A. Reinhardt, D. C. Beitz, and R. L. Horst. 1995. Breed affects tissue vitamin D receptor concentration in periparturient dairy cows: A milk fever risk factor? J. Dairy Sci. 78(Suppl. 1):184.

Goff, J. P., R. L. Horst, P. W. Jardon, C. Borelli, and J. Wedam. 1996. Field trials of an oral calcium propionate paste as an aid to prevent milk fever in periparturient dairy cows. J. Dairy Sci. 79:378–383.

Goff, J. P., R. Ruiz, and R. L. Horst. 1997. Relative acidiogenic activity of commonly used anionic salts—re-thinking the dietary cation-anion difference equations. J. Dairy Sci. 80(Suppl. 1):169.

Goings, R. L., N. L. Jacobson, D. C. Beitz, E. T. Littledike, and K. D. Wiggers. 1974. Prevention of parturient paresis by a prepartum, calcium-deficient diet. J. Dairy Sci. 57:1184–1188.

Grant, R. J. 1997. Interactions among forages and nonforage fiber sources. J. Dairy Sci. 80:1438–1446.

Green, B. L., B. W. McBride, D. Sandals, K. E. Leslie, R. Bagg, and P. Dick. 1999. The impact of a monensin controlled-release capsule on subclinical ketosis in the transition dairy cow. J. Dairy Sci. 82:333–342.

Green, H. B., R. L. Horst, D. C. Beitz, and E. T. Littledike. 1981. Vitamin D metabolites in plasma of cows fed a prepartum low-calcium diet for prevention of parturient hypocalcemia. J. Dairy Sci. 64:217–226.

Greenfield, R., S. S. Donkin, M. J. Cecava, and T. R. Johnson. 1998. Protein requirements of transition dairy cows. J. Dairy Sci. (Midwest Section Abstr.):79.

Greenhalgh, J. F. D., and K. E. Gardner. 1958. Effects of heavy concentrate feeding before calving upon lactation and mammary gland edema. J. Dairy Sci. 41:822–829.

Greenough, P. R., and J. J. Vermunt. 1991. Evaluation of subclinical laminitis in a dairy herd and observations on associated nutritional and management factors. Vet. Rec. 128:11–17.

Greunert, E. 1980. Etiology of retained bovine placenta. In: Current Therapy in Theriogenology, D. A. Morrow (ed.). W. B. Saunders Co., Philadelphia, pp. 180–186.

Griinari, J. M., B. A. Corl, S. H. Lacy, P. Y. Chouinard, K. V. V. Nurmela, and D. E. Bauman. 2000. Conjugated linoleic acid is synthesized endogenously in lactating dairy cows by 9-desaturase. J. Nutr. 130:2285–2291.

Griinari, J. M., D. A. Dwyer, M. A. McGuire, D. E. Bauman, D. L. Palmquist, and K. V. V. Nurmela. 1998. Trans-octadecaenoic acids and milk fat depression in lactating dairy cows. J. Dairy Sci. 81:1251–1261.

Grohn, Y., L. A. Lindberg, M. L. Bruss, and T. B. Farver. 1983. Fatty infiltration of liver in spontaneously ketotic dairy cows. J. Dairy Sci. 66:2320–2328.

Grum, D. E., J. K. Drackley, R. S. Younker, D. W. LaCount, and J. J. Veenhuizen. 1996. Nutrition during the dry period and hepatic lipid metabolism of periparturient cows. J. Dairy Sci. 79:1850–1864.

Grummer, R. R. 1993. Etiology of lipid-related metabolic disorders in periparturient dairy cows. J. Dairy Sci. 76:3882–3896.

Grummer, R. R. 1995. Impact of changes in organic nutrient metabolism on feeding the transition dairy cow. J. Anim. Sci. 73:2820–2833.

Grummer, R. R., P. C. Hoffman, M. L. Luck, and S. J. Bertics. 1995. Effect of prepartum and postpartum dietary energy on growth and lactation of primiparous cows. J. Dairy Sci. 78:172–180.

Grummer, R. R., S. J. Bertics, D. W. LaCount, J. A. Snow, M. R. Dentine, and R. H. Stauffacher. 1990. Estrogen induction of fatty liver in dairy cattle. J. Dairy Sci. 73:1537–1543.

Gwazdanskas, F. C., T. L. Bibb, T. L. McGilliard, and J. A. Lineweaver. 1979. Effect of selenium -vitamin E injection on time for placenta to pass and on reproductive functions. J. Dairy Sci. 62:978–981.

Habel, R. E. 1981. Stomach. Pp. 230 in Applied Veterinary Anatomy, Robert E. Habel, Ithaca, NY.

Hallgren, W. 1955. Studies on parturient paresis in dairy cows. Nord. Vet. Med. 7:433–463.

Hamada, T., T. Ishii, and S. Taguchi. 1982. Blood changes of spontaneously ketotic cows before and four hours after administration of glucose, xylitol, 1,2-propanediol, or magnesium propionate. J. Dairy Sci. 65:1509–1513.

Harfoot, C. G., and G. P. Hazlewood. 1987. Lipid Metabolism in the rumen. Page 285 in The Rumen Microbial Ecosystem. P. N. Hobson Ed. Elsevier Science Publishers, LTD, Essex, England.

Harmon, D. L., R. A. Britton, and R. L. Prior. 1983. Influence of diet on glucose turnover and rates of gluconeogenesis, oxidation, and turnover of $D_{(-)}$ lactate in the bovine. J. Nutr. 113:1842–1850.

Harrison, J. H., and H. R. Conrad. 1984. Effect of dietary calcium on selenium absorption by the nonlactating dairy cow. J. Dairy Sci. 67:1860–1664.

Harrison, J. H., D. D. Hancock, and H. R. Conrad. 1984. Vitamin E and selenium for reproduction of the dairy cow. J. Dairy Sci. 67:123–132.

Hartnell, G. F., S. E. Franson, D. E. Bauman, H. H. Head, J. T. Huber, R. C. Lamb, K. S. Madsen, W. J. Cole, and R. L. Hintz. 1991. Evaluation of sometribove in a prolonged-release system in lactating dairy cows production responses. J. Dairy Sci. 74:2645–2663.

Hartwell, J. H., M. J. Cecava, B. Miller, and S. S. Donkin. 1999. Rumen protected choline and dietary protein for transition cows. J. Dairy Sci. 82(Suppl. 1):125. (Abstr.).

Hathaway, H. D., W. J. Brakel, W. J. Tyznik, and H. E. Kaeser. 1957. The effect of concentrate intake at calving time on physiological activities with special emphasis on ketosis. J. Dairy Sci. 40:616 (Abstr.).

Hayes, D. P., D. U. Pfeiffer, and N. B. Williamson. 1996. Effect of intraruminal monensin capsules on reproductive performance and milk production of dairy cows fed pasture. J. Dairy Sci. 79:1000–1008.

Hayirli, A., R. R. Grummer, E. Nordheim, P. Crump, D. K. Beede, M. J. VandeHaar, and L. H. Kilmer. 1998. A mathematical model for describing dry matter intake of transition cows. J. Dairy Sci. 81(Suppl. 1): 296 (Abstr.).

Hays, R. L., and J. L. Albright. 1966. Udder edema: Its incidence and severity as affected by certain management practices. Illinois Res. 8:6.

Heinrichs, A. J., and G. J. Bush. 1991. Evaluation of decoquinate or lasalocid against coccidiosis from natural exposure in neonatal dairy calves. J. Dairy Sci. 74:3223–3227.

Hemken, R. W., and J. H. Vandersall. 1967. Feasibility of an all silage forage program. J. Dairy Sci. 50:417–422.

Hemken, R. W., E. Gainer, and R. F. Davis. 1960. Effect of kind and level of concentrates on udder edema. J. Dairy Sci. 43:887-888.

Hemken, R. W., W. H. Choate, and R. D. Plowman. 1969. Salt and water intakes as related to udder edema. J. Anim. Sci. 28:874 (Abstr.).

Hernandez-Urdaneta, A., C. E. Coppock, R. E. McDowell, D. Gianola, and N. E. Smith. 1976. Changes in forage-concentrate ratio of complete feeds for dairy cows. J. Dairy Sci. 59:695–707.

Hibbs, J. W., and W. D. Pounden. 1955. Studies on milk fever in dairy cows. IV. Prevention by short-time, prepartum feeding of massive doses of vitamin D. J. Dairy Sci. 38:65–72.

Hicks, J. D., and J. V. Pauli. 1976. Chronic udder edema: Clinical aspects of the syndrome and its connection with hypomagnasemia and anemia. New Zealand Vet. J. 24:225–228.

Hidiroglou, M., A. J. McAllister, and C. J. Williams. 1987. Prepartum supplementation of selenium and vitamin E to dairy cows: Assessment of selenium status and reproductive performance. J. Dairy Sci. 70:1281–1288.

Hocquette, J. F., and D. Bauchart. 1999. Intestinal absorption, blood transport, and hepatic muscle metabolism of fatty acids in preruminant and ruminant animals. Reprod. Nutr. Dev. 39:27–48.

Horst, R. L., and J. P. Goff. 1997. Milk fever and dietary potassium. Pp. 181–189 in 1997 Cornell Nutrition Conference. Rochester, N.Y.: Cornell University.

Horst, R. L., J. P. Goff, and T. A. Reinhardt. 1990. Advancing age results in reduction of intestinal and bone 1,25-dihydroxyvitamin D receptor. Endocrinol. 126:1053–1057.

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.

House, W. A., and A. W. Bell. 1993. Mineral accretion in the fetus and adnexa during late gestation in Holstein cows. J. Dairy Sci. 76:2999–3010.

Hull, B. L., and W. M. Wass. 1973. Abomasal displacement. 2. Hypocalcemia as a contributing causative factor. Vet. Med. Small Anim. Clin. 68:412–417.

Huntington, G. B., and R. Britton. 1979. Effect of dietary lactic acid content and energy level on rumen lactate metabolism in sheep. J. Anim. Sci. 47:241–246.

Huyler, M. T., R. L. Kincaid, and D. F. Dostal. 1999. Metabolic and yield responses of multiparous Holstein cows to prepartum rumen-undegradable protein. J. Dairy Sci. 82:527–536.

Ilan, D., A. Bne-Asher, Z. Holzer, Z. Nitsan, I. Nir, and D. Levy. 1981. Effect of monensin supplementation on growth, feed digestibility and utilization in young calves. Anim. Prod. 32:125–131.

Ishak, M. A., L. L. Larson, F. G. Owen, S. R. Lowry, and E. D. Erickson. 1983. Effects of selenium, vitamins, and ration fiber on placental retention and performance of dairy cattle. J. Dairy Sci. 66:99–106.

Jardon, P. W. 1995. Using urine pH to monitor anionic salt programs. Compend. Contin. Educ. Pract. Vet. 17:860–862.

Johnson, D. G., and D. E. Otterby. 1981. Influence of dry period diet on early postpartum health, feed intake, milk production, and reproductive efficiency of Holstein cows. J. Dairy Sci. 64:290–295.

Johnson, J. C., P. R. Utley, B. G. Mullinix, Jr., and A. Merrill. 1988. Effects of adding fat and lasalocid to diets of dairy cows. J. Dairy Sci. 71:2151–2165.

Jones, T. O., R. Knight, and R. K. Evans. 1984. Chronic udder edema in milking cows and heifers. Vet. Record 115:218–219.

Jonsson, G., and B. Pehrson. 1970. Trials with prophylactic treatment of parturient paresis. Vet. Rec. 87:575–583.

Jordan, E. R., and R. H. Fourdraine. 1993. Characterization of the management practices of the top milk producing herds in the country. J. Dairy Sci. 76:3247–3256.

Julien, W. E., H. R. Conrad, and D. R. Redman. 1976a. Influence of dietary protein on susceptibility to alert downer syndrome. J. Dairy Sci. 60:210–215.

Julien, W. E., H. R. Conrad, J. E. Jones, and A. L. Moxon. 1976b. Selenium and vitamin E and incidence of retained placenta in parturient dairy cows. J. Dairy Sci. 59:1954–1959.

Julien, W. E., H. R. Conrad, J. E. Jones, and A. L. Moxon. 1976c. Selenium and vitamin E and incidence of retained placenta in parturient dairy cows. II. Prevention in commercial herds with prepartum treatment. J. Dairy Sci. 59:1960–1962.

Julien, W. E., H. R. Conrad, J. W. Hibbs, and W. L. Crist. 1977. Milk fever in dairy cows. VIII. Effect of injected vitamin $D_3$ and calcium and phosphorus intake on incidence. J. Dairy Sci. 60:431–436.

Kalscheur, K. F., B. B. Teter, L. S. Piperova, and R. A. Erdman. 1997a. Effect of dietary forage level and buffer addition on milk trans fatty acids and duodenal trans fatty acid flow in lactating dairy cows. J. Dairy Sci. 80:2104–2114.

Kalscheur, K. F., B. B. Teter, L. S. Piperova, and R. A. Erdman. 1997b. Effect of fat source on trans fatty acid flow and milk fat production in lactating dairy cows. J. Dairy Sci. 80:2115–2126.

Kappel, L. C., R. H. Ingraham, E. D. Morgan, J. M. Dixon, L. Zeringue, D. Wilson, and D. K. Babcock. 1984. Selenium concentrations in feeds and effect of treating pregnant cows with selenium and vitamin E on blood selenium values and reproductive performance. Am. J. Vet. Res. 45:691–694.

Katz, I., and M. Keeney. 1966. Characterization of the octadecaenoic acids in rumen digesta and rumen bacteria. J. Dairy Sci. 49:962.

Kennelly, J. J., and K. A. Lien. 1997. Effect of ionophore supplementation on milk components from lactating cows. Pp. 40-49 in Proceedings of a symposium: Usefulness of ionophores in lactating dairy cattle. Ontario Veterinary College. June 25–26, 1997.

Kilmer, L. H., L. D. Muller, and T. J. Snyder. 1981. Addition of sodium bicarbonate to rations of postpartum dairy cows: physiological and metabolic effects. J. Dairy Sci. 64:2357–2369.

Kirchgessner, M., W. Windisch, W. Schwab, and H. L. Muller. 1991. Energy metabolism of lactating dairy cows treated with prolonged-release bovine somatotropin or energy deficiency. J. Dairy Sci. 74(Suppl. 2):35–43.

Kleppe, B. B., R. J. Aiello, R. R. Grummer, and L. E. Armentano. 1988. Triglyceride accumulation and very low density lipoprotein secretion by rat and goat hepatocytes in vitro. J. Dairy Sci. 71:1813–1822.

Knowlton, K. F., M. S. Allen, and P. S. Erickson. 1996a. Lasalocid and particle size of corn grain for dairy cows in early lactation. 1. Effect on performance, serum metabolites, and nutrient digestibility. J. Dairy Sci. 79:557–564.

Knowlton, K. F., M. S. Allen, and P. S. Erickson. 1996b. Lasalocid and particle size of corn grain for dairy cows in early lactation. 2. Effect on ruminal measurements and feeding behavior. J. Dairy Sci. 79:566–574.

Kohn, R. A., and T. F. Dunlap. 1998. Calculation of the buffering capacity of bicarbonate in the rumen and in vitro. J. Anim. Sci. 76(6):1702–1709.

Kung, L., Jr., E. M. Kreck, R. S. Tung, A. O. Hession, A. C. Sheperd, M. A. Cohen, H. E. Swain and J. A. Z. Leedlet. 1997. Effects of a live yeast culture and enzymes on in vitro ruminal fermentation and milk production of dairy cows. J. Dairy Sci. 80:2045–2051.

Kunz, P. L., J. W. Blum, I. C. Hart, H. Bickel, and J. Landis. 1985. Effects of different energy intakes before and after calving on food intake, performance and blood hormones and metabolites in dairy cows. Anim. Prod. 40:219–231.

Laurent, F., B. Vignon, D. Coomans, J. Wilkinson, and A. Bonnel. 1992. Influence of bovine somatotropin on the composition and manufacturing properties of milk. J. Dairy Sci. 75:2226–2234.

Lean, I. J., M. Curtis, R. Dyson, and B. Lowe. 1994. Effects of sodium monensin on reproductive performance of dairy cattle. I. Effects of conception rates, calving-to-conception intervals, calving-to-heat and milk production in dairy cows. Aust. Vet. J. 71:273–277.

Lean, I. J., and L. Wade. 1997. Effects of monensin on metabolism, production, and health of dairy cattle. Pp. 50–70 in Proceedings of a symposium: Usefulness of ionophores in lactating dairy cattle. Ontario Veterinary College. June 25–26, 1997.

Leidl, W., D. Hegner, and P. Rockel. 1980. Investigations on the $PGF_2$ concentration in maternal and feotal cotyledons of cows with and without retained fetal membranes. Zbl. Vet. Med. A. 27:691–696.

Lema, M., W. B. Tucker, M. Aslam, I. S. Shin, P. Le Ruyet, and G. D. Adams. 1992. Influence of calcium chloride fed prepartum on severity of edema and lactational performance of dairy heifers. J. Dairy Sci. 75:2388–2393.

Linn, J. G. 1988. Factors affecting the composition of milk from dairy cows. Pp. 224–241 in Designing Foods: Animal Product Options in the Marketplace. Washington, D.C.: National Academy Press.

Littledike, E. T., and R. L. Horst. 1980. Problems with vitamin D injections for prevention of milk fever: Toxicity of large doses and increased incidence of small doses. J. Dairy Sci. 63(Suppl. 1):89.

Littledike, E. T., J. A. Stuedemann, S. R. Wilkinson, and R. L. Horst. 1983. Grass tetany syndrome. Pp. 173–195 in Role of Magnesium in Animal Nutrition, J. P. Fontenot, G. E. Bunce, K. E. Webb, V. G. Allen, eds. Blacksburg, VA: Virginia Polytechnic Inst. and State Univ.

Loor, J. J., and J. H. Herbein. 1998. Exogenous conjugated linoleic acid isomers reduce bovine milk fat concentration and yield by inhibiting de novo synthesis. J. Nutr. 128:2411–2419.

Lowe, L. B., G. J. Ball, V. R. Carruthers, R. C. Dobos, G. A. Lynch, P. J. Moate, P. R. Poolse, and S. C. Valentine. 1991. Monensin controlled-release intraruminal capsule for control of bloat in pastured dairy cows. Aust. Vet. J. 68:17–20.

Lynch, J. M., D. M. Barbano, D. E. Bauman, G. F. Hartnell, and M. A. Nemeth. 1992. Effects of prolonged-release formulation of N-methionyl bovine somatotropin (sometribove) on milk fat. J. Dairy Sci. 75:1794–1809.

Maas, J. P. 1982. Prevention of retained fetal membranes in dairy cattle. Compend. Contin. Educ. 4:S519–S527.

Mackintosh, E. D., R. H. Phipps, J. D. Sutton, and J. I. D. Wilkinson. 1997. The effects of monensin and diet composition on methane production using the *in vitro* semi-continuous rumen simulation technique (Rusitec). J. Dairy Sci. 80(Suppl. 1):208 (Abstr.).

Maclean, C. W. 1971. The histopathology of laminitis in dairy cows. J. Comp. Pathol. 81:563–570.

Malven, P. V., R. E. Erb, M. F. D'Amico, T. S. Stewart, and B. P. Chew. 1983. Factors associated with edema of the mammary gland in primigravid dairy heifers. J. Dairy Sci. 66:246–252.

Manson, F. J., and J. D. Leaver. 1988. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. Anim. Prod. 47:185–190.

Massey, C. D., C. Wang, G. A. Donovan, and D. K. Beede. 1993. Hypocalcemia at parturition as a risk factor for left displacement of the abomasum in dairy cows. J. Am. Vet. Med. Assoc. 203:852–853.

McBride, B. W., J. L. Burton, and J.H. Burton. 1988. The influence of bovine growth hormone (somatotropin) on animals and their products. Res. Dev. Agric. 5:1–21.

McClymont, G. L., and S. Valence. 1962. Depression in blood glycerides and milk fat synthesis by glucose infusion. Proc. Nutr. Soc. 21:xli.

McDonald, R. J., G. W. McKay, and J. D. Thompson. 1961. The use of organic iodine in the treatment of repeat breeder cows. Proc. 4th Int. Congr. Anim. Reprod. 3:679–681.

McGilliard, M. L., and C. C. Stallings. 1998. Increase in milk yield of commercial dairy herds fed a microbial and enzyme supplement. J. Dairy Sci. 81:1353–1375.

McGuffey, R. K., and J. I. D. Wilkinson. 1991. Nutritional implications of bovine somatotropin for the lactating dairy cow. J. Dairy Sci. 74(Suppl. 2):63–71.

McGuire, M. A., J. M. Griinari, D. A. Dywer, and D. E. Bauman. 1995. Role of insulin in the regulation of mammary synthesis of fat and protein. J. Dairy Sci. 78:816–824.

McMeniman, N. P., and R. Elliot. 1995. Control of coccidia in young calves using lasalocid. Aust. Vet. J. 72:7–9

Meinert, R. A., C. M. J. Yang, A. J. Heinrichs, and G. A. Varga. 1992. Effect of monensin on growth, reproductive performance, and estimated body composition in Holstein heifers. J. Dairy Sci. 75:257–261.

Mellenberger, R. W., D. E. Bauman, and D. E. Nelson. 1973. Fatty acid and lactose synthesis in cow mammary tissue. Biochem J. 136:741–748.

Merchen, N. R. 1988. Digestion, absorption and excretion in ruminants. In The Ruminant Animal: Digestive Physiology and Nutrition, D. C. Church editor, Waveland Press, Prospect Heights, IL. pp. 176–177.

Mertens, D. R. 1997. Creating a system for meeting the fiber requirements of dairy cows. J. Dairy Sci. 80:1463–1481.

Michal, J. J., B. P. Chew, T. S. Wong, L. R. Heirman, and F. E. Sandaert. 1990. Effects of supplemental β-carotene on blood and mammary phagocyte function in peripartum dairy cows. J. Dairy Sci. 73(Suppl. 1): 149.(Abstr.).

Midla, L. T., K. H. Hoblet, W. P. Weiss, and M. L. Moeschberger. 1998. Supplemental dietary biotin for prevention of lesions associated with aseptic subclinical laminitis (pododermatitis aseptica diffusa) in primiparous cows. Am. J. Vet. Res. 59:733–738.

Miller, J. K., E. Brzezinska-Slebodzinska, and F. C. Madsen. 1993. Oxidative stress, antioxidants, and animal function. J. Dairy Sci. 76:2812–2823.

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.

Miranda, R. L. A., M.G. D. Mendoza, J. R. Barcena-Gama, M. S. S. Gonzalez, R. Ferrara, C. M. E. Ortega, and P.M. A. Cobos. 1996. Effect of *Saccharomyces cerevisiae* or *Aspergillus oryzae* cultures and NDF level on parameters of ruminal fermentation. Anim. Feed Sci. Tech. 63:289–296.

Moberg, R. 1959. Possible influences of iodine deficiency in reproductive performance in cattle with special reference to retained placenta. Proc. 3rd World Congress on Fertility and Sterility, Amsterdam.

Moberg, R. 1961. Possible influences of supplementary iodine, administered by evaporation, on reproductive performances in cattle. Proc. 4th Int. Congr. Anim. Reprod. 3:682–685.

Mongin, P. 1981. Recent advances in dietary anion-cation balance: Applications in poultry. Proc. Nutr. Soc. 40:285–295.

Moorby, J. M., R. J. Dewhrst, and S. Marsden. 1996. Effect of increasing digestible undegraded protein supply to dairy cows in late gestation on the yield and composition of milk during the subsequent lactation. Anim. Sci. 63:201–213.

Morrow, D. A. 1976. Fat cow syndrome. J. Dairy Sci. 59:1625–1629.

Moshen, M. K., F. El-Keraby, and M. S. El-Safty. 1981. Effect of monensin on milk yield, milk composition and reproductive performance of Friesian cows. Agric. Res. Rev. 59:15–27.

Mueller, F. J., J. K. Miller, M. H. Campbell, and F. C. Madsen. 1998. Prevention of udder edema in dairy cows. Proc. Tri-State Dairy Nutr. Conf. pp. 79–95, Fort Wayne, IN.

Mueller, F. J., J. K. Miller, N. Ramsey, R. C. DeLost, and F. C. Madsen. 1989a. Reduced udder edema in heifers fed vitamin E prepartum. J. Dairy Sci. 72:2211 (Abstr.).

Mueller, F. J., J. K. Miller, N. Ramsey, R. C. Delost, and F. C. Madsen. 1989b. Effects of vitamin E and excess iron on placental retention and subsequent milk yield in dairy cows. J. Dairy Sci. 72(Suppl. 1):564 (Abstr.).

Mueller, F. J., J. K. Miller, N. Ramsey, R. C. Delost, F. C. Madsen, and T. D. Mayers. 1988. Effects of vitamin E and excess iron on placental retention in dairy cows. J. Dairy Sci. 71(Suppl. 1):157 (Abstr.).

Muller, L. D. 1979. Buffers in rations for dairy calves and pre- and postpartum dairy cows. Pp. 125–156 in Regulation of Acid-Base Balance, W. H. Hale and P. Meinhardt, eds. Church and Dwight, Co. Inc.

Muller, L. D. 1992. BST and dairy cow performance. Pp. 53–72 in Bovine Somatotropin and Emerging Issues—An Assessment. M.C. Halberg, ed. Westview Press, Boulder, CO.

Nagaraja, T. G., T. B. Avery, E. E. Bartley, S. J. Galitzer, and A. D. Dayton. 1981. Prevention of lactic acidosis in cattle by lasalocid or monensin. J. Anim. Sci. 53:206–215.

National Research Council. 1989. Nutrient requirements of dairy cattle, sixth revised ed. Washington, D.C.: National Academy Press.

National Research Council. 1994. Metabolic modifiers: Effects on the nutrient requirements of food-producing animals. Washington, D.C.: National Academy Press.

National Research Council. 1996. Nutrient Requirements of Beef Cattle, Seventh Revised Ed. Washington, D.C.: National Academy Press.

Nestor, K. E., Jr., R. W. Hemken, and R. J. Harmon. 1988. Influence of sodium chloride and potassium bicarbonate on udder edema and selected blood parameters. J. Dairy Sci. 71:366–372.

Newbold, C. J., R. J. Wallace and F. M. McIntosh. 1996. Mode of action of the yeast *Saccharomyces cerevisiae* as a feed additive for ruminants. Brit. J. Nutr. 76:249–261.

Nicholson, J. W. G., and H. M. Cunningham. 1965. Retained placenta, abortion, and abnormal calves from beef cows fed all barley rations. Can. Vet. J. 6:275–281.

Nocek, J. E. 1997. Bovine acidosis: Implications on laminitis. J. Dairy Sci. 80:1005–1028.

Noorsdsy, J. L., H. W. Leipold, D. L. Carmahan, R. A. Frey, J. Vestweber, M. G. Robl, G. Kennedy, J. R. Dunham, T. E. Chapin, and W. E. Moore. 1973. Metabolic disturbances in the dairy cow influenced by managerial practices: Case reports and epidemiological studies. Rumen Function Conf., Chicago, IL.

Oetzel G. R. 1996. Effect of calcium chloride gel treatment in dairy cows on incidence of periparturient diseases. J Am Vet Med. Assoc. 209:958–961.

Oetzel, G. R. 1993. Effects of prophylactic treatment with a calcium chloride gel on serum calcium concentration at calving, milk fever, and displaced abomasum in Holstein cows. J. Dairy Sci. 76(Suppl 1): 304 (Abstr.).

Oetzel, G. R., and J. P. Goff. 1998. Milk fever (parturient paresis) in cows, ewes, and doe goats. Pp. 215–218 in Current Veterinary Therapy 4: Food Animal Practice, J. L. Howard and R. A. Smith, eds. Philadelphia: W. B. Saunders Co.

Oetzel, G. R., J. D. Olson, C. R. Curtis, and M. J. Fettman. 1988. Ammonium chloride and ammonium sulfate for prevention of parturient paresis in dairy cows. J. Dairy Sci. 71:3302–3309.

Oetzel, G. R., M. J. Fettman, D. W. Hamar, and J. D. Olson. 1991. Screening of anionic salts for palatability, effects on acid-base status and urinary calcium excretion in dairy cows. J. Dairy Sci. 74:965–971.

Oetzel, G. R., K. V. Nordlund, and E. F. Garrett. 1999. Effect of ruminal pH and stage of lactation on ruminal lactate concentrations in dairy cows. J. Dairy Sci. 82(Suppl. 1):38.

Peel, C. J., and D. E. Bauman. 1987. Somatotropin and lactation. J. Dairy Sci. 70:474–486.

Peel, C. J., D. L. Hard, K. S. Madsen, and G. de Kerchove. 1989. Bovine somatotropin: Mechanism of action and experimental results from different world areas. Pp. 9–18 in Meeting the Challenges of New Technology. Proceedings of the Monsanto Technical Symposium, Animal Science Division, St. Louis, MO: Monsanto Agricultural Company.

Pelissier, C. L. 1976. Dairy cattle breeding problems and their consequences. Theriogenology 6:575–583.

Petersen, W. E. 1932. The effects of cod liver oil in the ration upon the quantity and quality of cow's milk. J. Dairy Sci. 15:283.

Phillippo, M., G. W. Reid, and I. M. Nevison. 1994. Parturient hypocalcaemia in dairy cows: Effects of dietary acidity on plasma minerals and calciotrophic hormones. Res. Vet. Sci. 56:303–309.

Phipps, R. H., B. A. Jones, J. I. D. Wilkinson, and M. E. Tarrant. 1995. Effect of monensin on milk production of Friesian dairy cows in the United Kingdom. J. Dairy Sci. 78(Suppl. 1):268 (Abstr.).

Phipps, R. H., B. A. Jones, J. I. D. Wilkinson, and M. E. Tarrant. 1997a. The influence of monensin on milk production of Friesian dairy cows in the United Kingdom. J. Dairy Sci. 80(Suppl. 1):208 (Abstr.).

Phipps, R. H., J. I. D. Wilkinson, A. K. Jones, L. J. Jonker, M. Tarrant, E. D. Mackintosh, and A. M. Cocker. 1997b. A study over two lactations: The effect of monensin on milk production, health, and reproduction in lactating dairy cows. Pp. 26–39 in Proceedings of a symposium: Usefulness of ionophores in lactating dairy cattle. Ontario Veterinary College. June 25–26, 1997.

Piperova, L. S., B. B. Teter, I. Bruckental, J. Sampagna, S. E. Mills, M. P. Yurawecz, J. Fritsche, K. Ku, and R. A. Erdman. 2000. Mammary lipogenic enzyme activity, trans fatty acids and conjugated linoleic acid isomers during milk fat depression in lactating dairy cows. J. Nutr. 130:2568–2574.

Powell, E. B. 1939. Some relations of the roughage intake to composition of milk. J. Dairy Sci. 22:452.

Pullen, D. L., J. S. Liesman, and R. S. Emery. 1990. A species comparison of liver slice synthesis and secretion of triacylglycerol from nonesterified fatty acid in the media. J. Anim. Sci. 68:1395–1399.

Putnam, D. E., and G. A. Varga. 1998. Protein density and its influence on metabolite concentration and nitrogen retention by Holstein cows in late gestation. J. Dairy Sci. 81:1608–1618.

Putnam, D. E., C. G. Schwab, M. T. Socha, N. L. Whitehouse, N. A. Kierstead, and B. D. Garthwaite. 1997. Effect of yeast culture in the diets of early lactation dairy cows on ruminal fermentation and passage of nitrogen fractions and amino acids to the small intestine. J. Dairy Sci. 80:374–384.

Putnam, D. E., G. A. Varga, and H. M. Dann. 1999. Metabolic and production responses to dietary protein and exogenous somatotropin in late gestation dairy cows. J. Dairy Sci. 82:982–995.

Quigley III, J. D., J. J. Drewry, L. M. Murray, and S. J. Ivey. 1997. Effects of lasalocid in milk replacer or calf starter on health and performance of calves challenged with *eimeria* species. J. Dairy Sci. 80:2972–2976.

Radostits, O. M., D. C. Blood, and C. C. Gay. 1994. Page 1618 *in* Veterinary Medicine. Bailliere Tindall, Philadelphia, PA.

Ramanzin, M., L. Bailoni, S. Schiavon, and G. Bittante. 1997. Effect of monensin on milk production and efficiency of dairy cows fed two diets differing in forage to concentrate ratios. J. Dairy Sci. 80:1136–1142.

Randall, W. E., R. W. Hemken, L. S. Bull, and L. W. Douglas. 1974. Effect of dietary sodium and potassium on udder edema in Holstein heifers. J. Dairy Sci. 57:472–475.

Roberts, S. J. 1961. Page 225 *in* Veterinary obstetrics and genital diseases. Edwards Bros., Ann Arbor, MI.

Robinson, P. H. 1997. Effect of yeast culture (*Saccharomyces cerevisiae)* on adaptation of cows to diets postpartum. J. Dairy Sci. 80:1119–1125.

Rogers, J. A., L. D. Muller, C. L. Davis, W. Chalupa, D. S. Kronfeld, L. F. Karcher, and K. R. Cummings. 1985. Response of dairy cows to sodium bicarbonate and limestone in early lactation. J. Dairy Sci. 68:646–660.

Rogers, J. A., L. D. Muller, T. J. Snyder, and T. L. Maddox. 1985. Milk production, nutrient digestion, and rate of digesta passage in dairy cows fed long or chopped alfalfa hay supplemented with sodium bicarbonate. J. Dairy Sci. 68:868–880.

Romo, G. A., D. P. Casper, R. A. Erdman, and B. B. Teter. 1996. Abomasal infusion of cis or trans fatty acid isomers and energy metabolism of lactating dairy cows. J. Dairy Sci. 79:2005–2015.

Ronning, M., E. R. Berousek, A. H. Kuhman, and W. D. Gallap. 1953. The carotene requirements for reproduction in Guernsey cattle. J. Dairy Sci. 36:52–56.

Rude, R. K., J. S. Adams, E. Ryzen, D. B. Endres, H. Niimi, R. L. Horst, J. G. Haddad, Jr., and F. R. Singer. 1985. Low serum concentrations of 1,25-dihydroxyvitamin D in human magnesium deficiency. J. Clin. Endo. Met. 61:933–940.

Rude, R. K., S. B. Oldham, C. F. Sharp, Jr., and F. R. Singer. 1978. Parathyroid hormone secretion in magnesium deficiency. J. Clin. Endocrin. Metab. 47:800–806.

Russell, J. B., and J. M. Chow. 1993. Another theory for the action of ruminal buffer salts: Decreased starch fermentation and propionate production. J. Dairy Sci. 76:826–830.

Sahlu, T., S. P. Hart, T. Le-Trong, Z. Jia, L. Dawson, T. Gipson, and T. H. Teh. 1995. Influence of prepartum protein and energy concentrations for dairy goats during pregnancy and early lactation. J. Dairy Sci. 78:378–387.

Sanders, D. E., and J. A. Sanders. 1981. Chronic udder edema in dairy cows. J. Am. Vet. Med. Assoc. 178:1273–1274.

Sansom, B. F., R. Manston, and M. J. Vagg. 1983. Magnesium and milk fever. Vet. Rec. 112:447–449.

Santos, J. E. P., E. J. DePeters, P. W. Jaron, and J. T. Huber. 1999a. Effect of prepartum crude protein level on performance of multiparous Holstein cows. J. Dairy Sci. 82(Suppl.1):120 (Abstr.).

Santos, J. E. P., E. J. DePeters, P. W. Jaron, and J. T. Huber. 1999b. Effect of prepartum crude protein level on performance of primiparous Holstein cows. J. Dairy Sci. 82(Suppl.1):120 (Abstr.).

Satter, L. D., and L. L. Slyter. 1974. Effect of ammonia concentration on rumen microbial protein production in vitro. Brit. J. Nutr. 32:199–208.

Sauer, F. D., J. D. Erfle, and L. J. Fisher. 1973. Propylene glycol and glycerol as a feed additive for lactating dairy cows: An evaluation of blood metabolite parameters. Can. J. Anim. Sci. 53:265–271.

Sauer, F. D., J. K. G. Kramer, and W. J. Cantwell. 1989. Antiketogenic effects of monensin in early lactation. J. Dairy Sci. 72:436–442.

Schelling, G. T. 1984. Monensin mode of action in the rumen. J. Anim. Sci. 58:1518–1527.

Schingoethe, D. J., C. A. Kirkbride, I. S. Palmer, M. J. Owens, and W. L. Tucker. 1982. Response of cows consuming adequate selenium to vitamin E and selenium supplementation prepartum. J. Dairy Sci. 65:2338–2344.

Schmidt, G. H., and L. H. Schultz. 1959. Effect of three levels of grain feeding during the dry period on the incidence of ketosis, severity of udder edema, and subsequent milk production of dairy cows. J. Dairy Sci. 42:170–179.

Schultz, L. H. 1958. Use of sodium propionate in the prevention of ketosis in dairy cattle. J. Dairy Sci. 41:160.

Sechen, S. J., D. E. Bauman, H. F. Tyrrell, and P. J. Reynolds. 1989. Effect of somatotropin on kinetics of nonesterified fatty acids and partition of energy, carbon, and nitrogen in lactating dairy cows. J. Dairy Sci. 72:59–67.

Segerson, E. C., G. J. Riviere, H. L. Dalton, and M. D. Whitacre. 1981. Retained placenta of Holstein cows treated with selenium and vitamin E. J. Dairy Sci. 64:1833–1836.

Shaver, R. D. 1997. Nutritional risk factors in the etiology of left displaced abomasum in dairy cows: A review. J. Dairy Sci. 80:2449–2453.

Shaw, J. C., and W. L. Ensor. 1959. Effect of feeding cod liver oil and unsaturated fatty acids on ruminal volatile fatty acids and milk fat content. J. Dairy Sci. 42:1238.

Skaar, T. C., R. R. Grummer, M. R. Dentine, and R. H. Stauffacher. 1989. Seasonal effects of pre- and postpartum fat and niacin feeding on lactation performance and lipid metabolism. J. Dairy Sci. 72:2028–2038.

Snyder, D. L., R. K. McGuffey, L. C. Pendlum, and E. L. Potter. 1981. Growth and reproduction of Holstein heifers fed monensin. J. Dairy Sci. 64(Suppl. 1):127 (Abstr.).

Solorzano, L. D., L. E. Armentano, R. S. Emery, and B. R. Schricker. 1989. Effects of Rumen-Mate on lactational performance of Holsteins fed a high grain diet. J. Dairy Sci. 72:1831–1841.

Staples, C. R., S. M. Emanuele, M. Ventura, D. K. Beede, and B. Schricker. 1986. Effects of a new multielement buffer on production, ruminal environment and blood minerals of lactating dairy cows. J. Dairy Sci. 71:1573–1586.

Steen, T. M., J. D. Quigley III, R. N. Heitman, and J. D. Gresham. 1992. Effects of lasalocid and undegradable protein on growth and body composition of Holstein heifers. J. Dairy Sci. 75:2517–2523.

Stewart, P. A. 1983. Modern quantitative acid-base chemistry. Can. J. Physiol. Pharmacol. 61:1444–1461.

Storry, J. E., and J. A. F. Rook. 1965. The effects of a diet low in hay and high in flaked maize on milk-fat secretion and on the concentrations of certain constituents in the blood plasma of the cow. Brit. J. Nutr. 19:101.

Stowe, H. D., J. W. Thomas, T. Johnson, J. V. Marateniuk, D. A. Morrow, and D. E. Ullrey. 1988. Responses of dairy cattle to long-term and short-term supplementation with oral selenium and vitamin E. J. Dairy Sci. 71:1830–1839.

Strang, B. D., S. J. Bertics, R. R. Grummer, and L. E. Armenatno. 1998. Effect of long-chain fatty acids on triglyceride accumulation, gluconeogenesis, and ureagenesis in bovine hepatocytes. J. Dairy Sci. 81:728–739.

Studer, V. A., R. R. Grummer, and S. J. Bertics. 1993. Effect of prepartum propylene glycol administration on periparturient fatty liver in dairy cows. J. Dairy Sci. 76:2931–2939.

Sutton, J. D. 1989. Altering milk composition by feeding. J. Dairy Sci. 72:2801–2814.

Swanson, E. W., and J. I. Poffenbarger. 1979. Mammary gland development of dairy heifers during their first gestation. J. Dairy Sci. 62:702–714.

Symanowski, J. T., H. B. Green, J. R. Wagner, J. I. D. Wilkinson, J. S. Davis, M. R. Himstedt, M. S. Allen, E. Block, J. J. Brennen, H. H. Head, J. J. Kennelly, J. N. Nielsen, J. E. Nocek, J. J. Vand Der List, and L. W. Whitlow. 1999. Milk production and efficiency of cows fed monensin. J. Dairy Sci. 82(Suppl. 1):75 (Abstr.).

Teter, B. B., J. Sampugna, and M. Keeney. 1990. Milk fat depression in C57B1/6J mice consuming partially hydrogenated fat. J. Nutr. 120:818.

Thomas, D. G., J. K. Miller, F. J. Mueller, C. R. Holmes, and F. C. Madsen. 1990. Daily supplementation of dairy cows with 1,000 IU vitamin E for 6 weeks before calving reduced placental retention by almost 60 percent. J. Dairy Sci. 73(Suppl. 1):166 (Abstr.).

Thomas, E. D. 1999. A new look at managing potassium levels in grasses. Hoard's Dairyman. Jan. 29th:53.

Thomas, J. W., R. S. Emery, J. K. Breaux, and J. S. Liesman. 1984. Response of milking cows fed a high concentrate, low roughage diet plus sodium bicarbonate, magnesium oxide, or magnesium hydroxide. J. Dairy Sci. 67:2532–2545.

Trinder, N., C. D. Woodhouse, and C. P. Renton. 1969. The effect of vitamin E and selenium on the incidence of retained placentae in dairy cows. Vet. Rec. 85:550–553.

Trinder, N., R. J. Hall, and C. P. Renton. 1973. The relationship between the intake of selenium and vitamin E on the incidence of retained placentae in dairy cows. Vet. Rec. 93:641–643.

Tucker, W. B., I. S. Shin, J. F. Hougue, G. D. Aslam, M. T. Van Koevering, R. K. Vernon, and K. R. Cummings. 1994. Natural sodium sesquicarbonate fed for an entire lactation: Influence on performance and acid-base status of dairy cows. J. Dairy Sci. 77:3111–3117.

Tucker, W. B., J. F. Hogue, D. F. Waterman, T. S. Swenson, Z. Xin, R. W. Hemken, J. A. Jackson, G. D. Adams, and L. J. Spicer. 1991. Role of sulfur and chloride in the dietary cation-anion balance equation for lactating dairy cattle. J. Anim. Sci. 69:1205–1213.

Tucker, W. B., J. F. Hogue, G. D. Adams, M. Aslam, I. S. Shin, and G. Morgan. 1992. Influence of dietary cation-anion balance during the dry period on the occurrence of parturient paresis in cows fed excess calcium. J. Anim. Sci. 70:1238–1250.

Tyrrell, H. F., A. C. G. Brown, P. J. Reynolds, G. L. Haaland, D. E. Bauman, C. J. Peel, and W. D. Steinhour. 1988. Effect of bovine somatotropin on metabolism of lactating dairy cows: Energy and nitrogen utilization as determined by respiration calorimetry. J. Nutr. 118:1024–1030.

Tyznick W., and N. N. Allen. 1951. The relation of roughage intake to the fat content of the milk and the level of fatty acids in the rumen. J. Dairy Sci. 34:493.

U.S. Department of Agriculture, Animal and Plant Health Inspection Service. 1996. Part I: Reference of 1996 Dairy Management Practices. Fort Collins, CO: Centers for Epidemiology and Animal Health.

Van de Braak, A. E., A. T. van't Klooster, and A. Malestein. 1987. Influence of a deficient supply of magnesium during the dry period on the rate of calcium mobilisation by dairy cows at parturition. Res. Vet. Sci. 42:101–108.

Van den Berg, G. 1991. A review of quality and processing suitability of milk from cows treated with bovine somatotropin. J. Dairy Sci. 74(Suppl. 2):2–11.

Van Der Werf, J. H. J., L. J. Jonker, and J. K. Oldenbroek. 1998. Effect of monensin on milk production by Jersey cows. J. Dairy Sci. 81:427–433.

Van Maanen, R. W., J. H. Herbein, A. D. McGilliard, and J. W. Young. 1978. Effects of monensin on in vivo rumen propionate production and blood glucose kinetics in cattle. J. Nutr. 108:1002–1007.

Van Soest, P. J. 1994. pp. 334–336 In Nutritional Ecology of the Ruminant. Cornell University Press, Ithaca, New York.

VandeHaar, M. J., and S. S. Donkin. 1999. Protein nutrition of dry cows. Proc. Tri-State Dairy Nutr. Conf., M. L. Eastridge, ed. April 20, Ft. Wayne, IN, pages 113–130. The Ohio State University, Columbus.

VandeHaar, M. J., G. Yousif, B. K. Sharma, T. H. Herdt, R. S. Emery, M. S. Allen, and J. S. Liesman. 1999. Energy and protein density of prepartum diets alters fat and protein metabolism of dairy cows in the periparturient period. J. Dairy Sci. 82:1282–1295.

VanSaun, R. J., and C. J. Sniffen. 1995. Effects of undegradable protein fed prepartum on lactation, reproduction, and health in dairy cattle. J. Dairy Sci. 78:265 (Abstr.).

VanSaun, R. J., S. C. Idleman, and C. J. Sniffen. 1993. Effect of undegradable protein amount fed prepartum on postpartum production in first lactation Holstein cows. J. Dairy Sci. 76:236–244.

Vazquez-Anon, M., S. Bertics, M. Luck, R. R. Grummer, and J. Pinheiro. 1994. Peripartum liver triglyceride and plasma metabolites in dairy cows. J. Dairy Sci. 77:1521–1528.

Veenhuizen, J. J., J. K. Drackley, M. J. Richard, T. P. Sanderson, L. D. Miller, and J. W. Young. 1991. Metabolic changes in blood and liver during development and the early treatment of experimental fatty liver and ketosis in cows. J. Dairy Sci. 74:4238–4253.

Vermunt, J. J., and P. R. Greenough. 1994. Predisposing factors of laminitis in cattle. Brit. Vet. J. 150:151–164.

Vestweber, J. G. E., and F. K. Al-Ani. 1983. Udder edema in cattle. Compendium Continuing Education Practicing Vet. 5:S5–S12.

Vestweber, J. G. E., and F. K. Al-Ani. 1984. Udder edema: Biochemical studies in Holstein cows. Cornell Vet. 74:366–372.

Vicini, J. L., W. S. Cohick, J. H. Clark, S. N. McCutcheon, and D. E. Bauman. 1988. Effects of feed intake and sodium bicarbonate on milk production and concentration of hormones and metabolites in plasma of cows. J. Dairy Sci. 71:1232–1238.

Vigue, R. F. 1963. Management of udder edema in cattle. Can. Vet. J. 4:236–241.

Wagner, J. R., H. B. Green, J. T. Symanowski, J. I. D. Wilkinson, J. S. Davis, M. R. Himstedt, M. S. Allen, E. Block, J. J. Brennen, H. H. Head, J. J. Kennelly, J. N. Nielsen, J. E. Nocek, J. J. Vand Der List, and L. W. Whitlow. 1999. Effect of monensin on feed intake, body weight, and body condition in dairy cows. J. Dairy Sci. 82(Suppl. 1): 75 (Abstr.).

Weaver, L. D. 1987. Effects of nutrition on reproduction in dairy cows. Vet. Clin. North Am. [Food Anim. Pract.] 3:513–531.

Weiss, W. P., and B. A. Amiet. 1990. Effect of lasalocid on performance of lactating dairy cows. J. Dairy Sci. 73:153–162.

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 diary herds. J. Dairy Sci. 73:381–390.

West, J. W., C. E. Coppock, D. H. Nave, J. M. Labore, and L. W. Greene. 1987. Effects of potassium carbonate and sodium bicarbonate on rumen function in lactating Holstein cows. J. Dairy Sci. 70:81–90.

Williams, P. E. V., C. A. G. Tait, G. M. Innes, and C. J. Newbold. 1991. Effects of the inclusion of yeast culture (*Saccharomyces cerevisiae* plus growth medium) in the diet of dairy cows on milk yield and forage degradation and fermentation patterns in the rumen of steers. J. Anim. Sci. 69:3016–3026.

Wise, G. H., F. W. Atkeson, M. J. Caldwell, and J. S. Hughes. 1946. Effect of level of protein in the prepartum ration of dairy heifers and cows on the degree of mammary edema. J. Anim. Sci. 5:397 (Abstr.).

Wohlt, J. E., T. T. Corcione, and P.K. Zajac. 1998. Effect of yeast on feed intake and performance of cows fed diets based on corn silage during early lactation. J. Dairy Sci. 81:1345–1352.

Wonsil, B. J., J. H. Herbein, and B. A. Watkins. 1994. Dietary and ruminally derived trans-18:1 fatty acids alter bovine milk lipids. J. Nutr. 124:556–565.

Wu, Z., R. J. Fisher, C. E. Polan, and C. G. Schwab. 1997. Lactational performance of cows fed low or high ruminally undegradable protein prepartum and supplemental methionine and lysine postpartum. J. Dairy Sci. 80:722–729.

Xin, Z., W. B. Tucker, and R. W. Hemken. 1989. Effect of reactivity rate and particle size of magnesium oxide on magnesium availability, acid-base balance, mineral metabolism, and milking performance of dairy cows. J. Dairy Sci. 72:462–470.

Xu, S., J. H. Harrison, R. E. Riley, and K. A. Loney. 1994. Effect of buffer addition to high grain total mixed rations on rumen pH, feed intake, milk production, and milk composition. J. Dairy Sci. 77:782–788.

Yokoyama, M. T., and K. A. Johnson. 1988. Microbiology of the rumen and intestine. Page 125 *in* The Ruminant Animal: Digestive Physiology and Nutrition. D. C. Church, ed. Waveland Press, Inc., Prospect Heights, IL.

Yoon, I. K., and M. D. Stern. 1995. Influence of direct-fed microbials on ruminal microbial fermentation and performance of ruminants: A review. Austral. Asian J. Anim. Sci. 8:533–555.

Yoon, I. K., and M. D. Stern. 1996. Effects of *Saccharomyces cerevisiae* and *Aspergillus oryzae* cultures on ruminal fermentation in dairy cows. J. Dairy Sci. 79:411–417.

Yurawecz, M. P., J. A. G. Roach, N. Sehat, M. M. Mossoba, J. K. G. Kramer, J. Fritsche, H. Steinhart, and K. Ku. 1998. A new conjugated linoleic acid isomer, 7 trans, 9 cis-octadecadienoic acid, in cow milk, cheese, beef and human milk and adipose tissues. Lipids 33:803–809.

Zamet, C. N., V. F. Colenbrander, C. J. Callahan, B. P. Chew, R. E. Erb, and N. J. Moeller. 1979a. Variables associated with peripartum traits in dairy cows. I. Effect of dietary forages and disorders on voluntary intake of feed, body weight and milk yield. Theriogenology 11:229–244.

Zamet, C. N., V. F. Colenbrander, R. E. Erb, C. J. Callahan, B. P. Chew, and N. J. Moeller. 1979b. Variables associated with peripartum traits in dairy cows. II. Interrelationships among -disorders and their effects on intake of feed and on reproductive efficiency. Theriogenology 11:245–260.