Optimizing Milk Production and Reproductive Efficiency by Controlling Metabolic Disease

David I. Byers

120 Crestview Drive, Galax VA 24333 USA
E-mail: David_Byers@mcimail.com

Take Home Message

- A healthy cow will give more milk and have better reproduction.
- Controlling metabolic diseases is necessary for optimum performance.
- Metabolic diseases are complexes (e.g., one condition leads to another, and to another, etc.).
- Fatty liver, hypocalcemia, and acidosis-laminitis are major metabolic complexes that adversely affect dairy cattle performance.

Introduction

The basic tenet of this paper is that cows that have fewer health disorders have greater milk production and better reproductive performance. Conversely, unhealthy cows do not produce or reproduce well. Controlling metabolic diseases, then, is paramount for good lactational and reproductive performance (28).

Also, noting that metabolic diseases in the dairy cow are complexes is important (11, 24, 25, 29). That is, one condition leads to another. For example, a cow that has milk fever (MF) is four times more likely also to have a retained placenta (RP) and 16.4 times more likely to develop ketosis (29).

This paper will focus attention on controlling three metabolic diseases. They are fatty liver (FL), hypocalcemia (HC), and rumen acidosis (RA). It is this individual's conviction that they are key diseases hindering lactation yield and reproductive performance in dairy cattle today.

▪ Fatty Liver Disease

Description

Some misconceptions exist about FL. One, we commonly think that FL disease develops after calving when the cow is mobilizing large amounts of body fat. Recent research (8, 14, 15) shows that FL often develops prepartum or at parturition. Thus, it is a periparturient disease. Two, we thought that FL disease was the result of obesity. These cows are more susceptible, but some surveys show that all cows have some degree of fatty liver (17).

Cows show depression, lack of appetite, general weakness. Also, there are generally complicating conditions such as MF, RP, displaced abomasum (DA) metritis, and mastitis. The outstanding clinical feature of this disease is the poor response to treatment of the accompanying diseases. For example, a cow with metritis may die or recover slowly and milk poorly.

Etiology

Fatty liver begins with mobilization of body fat. Many factors stimulate lipolysis of adipose tissue, including negative energy balance (NEB), hypoglycemia, and low serum insulin concentrations and relatively high serum concentrations of lipolytic hormones, such as growth hormone, placental lactogen, and prolactin (7, 16, 17). Estrogen, a potent regulator of hepatic FA metabolism in nonruminants, may play a key role in FL development (9, 13). Mobilization of fat from adipose tissue results in an increased level of serum nonesterified fatty acids (NEFA) and increased uptake by the liver. Fatty acids (FA) in high concentrations are toxic to tissues. Once in the liver, they are subject to two metabolic pathways: oxidation or esterification (16). Re-esterification leads to triglyceride (TG**)** synthesis. Triglyceride is a source of energy for body tissues and the mammary gland. For TG to be exported from the liver, the liver must package them into lipoprotein (LP) particles. When hepatic production of TG exceeds LP export, FL results (8, 15, 17).

Nutrient Partitioning and Fatty Liver

Competition among organs for nutrients also affect the development of FL. Fetal needs receive top priority in advanced pregnancy. Understanding the dynamics of fetal growth is helpful to our understanding of the development of FL.

Gestational Nutritional Requirements

The goal of most consultants is to feed cows during the nonlactating, pregnant cow for maintenance and gestation (9). The debatable question is: What is the requirement for gestation? The National Research Council (19) states that the gestational requirement is 30% of maintenance (e.g., 80 Kcal of NEL/BW

$kg^{0.75}$). Plainly, the energy requirement for gestation is 24 Kcal of NEL/BW $kg^{0.75}$ (NRC) (18) However, some consultants think this number is low.

Fetal growth during the last trimester is exponential (7, 11, 29). This creates a very large increase in the gestational requirement before parturition. Providing the nutrients that are required during this period of rapid growth is imperative. Work by Bell (7) and Ferrell (10) showed that this is a significant nutritional requirement.The NRC gives a range of 3 to 6 Mal of NEL per day. This compares to about 3 Mcal from the above formula for a 1400-lb cow (i.e., 24 kcal of NEL/600 kg $BW^{0.75}$). Based on a maintenance requirement of 10 Mcal of NEL, then the requirement for maintenance and gestation is 13 or 16 Mcal. Energy densities of 0.59 or 0.73 Mcal of NEL/lb. of DM, respectively, are necessary to meet the energy needs for a close-up cow consuming 22 lb. DMI. Obviously, most dairy consultants are using a requirement closer to the latter figure (e.g., 0.73 Mcal of NEL/lb. of DM).

Paradoxical Decline in Feed Intake

Unfortunately, DMI begins to decline around 3 weeks before calving. Feed intake declines about 30% (8). It begins to decline around 5 weeks with twins. The increased gestational requirement coupled with a decline in DMI make it necessary to feed a nutrient dense ration for 3 weeks before calving. Cows carrying twins should receive a greater supply of nutrients for 5 weeks.

Use of Maternal Energy

The conversion of maternal energy into energy deposited into the gravid uterus is poor. Ferrell (10) reported a 14% conversion of dietary metabolizable energy (ME) into energy retention of the gravid uterus. This compares with a conversion of ME to NEL of 60% (19).

Fuel for the Fetus

The fetus has very specific metabolic needs. Chandler (9) sheds light on this subject:

- Use of ME from the dam is exceedingly low (i.e., 14%).
- The fetus has a very high metabolic rate. There is a temperature gradient of 0.50 to 1.0EC over the dam. This high metabolic requirement results in 60% or greater of the energy being dissipated as heat.
- The placenta has an oxidative requirement equal to or exceeding that of the fetus.
- The primary substrates for achievement of this condition via oxidative metabolism are glucose, lactate, and amino acids. Since lactate originates from glucose, we can state that the fetus runs on glucose and amino acids.
- The amino acid role is significant with data showing that almost 60% of the nitrogen uptake are lost in oxidative forms.

Fuel for the Dam

With the fetus dominating the use of glucose for energy, the dam must rely largely on the VFA, acetate, and long chain FA. Fatty acid mobilization occurs to supply energy for the dam. No doubt, this predisposes her to FL. Also, if there is any additional depression in feed intake or if the ration lacks proper nutrient balance, an exacerbation of FL and other metabolic problems may occur.

Treatment and Prevention

No treatment is effective. Prevention is the best course of action. Because FL develops by day 1 after calving, strategies to prevent metabolic disorders must start before calving (15). The key is to avoid excessive FA mobilization of body fat. Plausible strategies include the following:

Body Condition Management

Cows should not lose body condition (BC) during the dry period. Dairymen should score dry cows each week. Cows losing weight may have twins. Move these cows to a higher energy ration. Increasing BC minimally is possible (i.e., 0.25- to 0.50-point) during the dry period.

Avoid getting cows over conditioned (i.e., >4.0). Fat cows have reduced appetites after calving. Though not any studies are showing greater depression of DMI before calving (15), anecdotal evidence suggests that this be the case. The best way to avoid fat cows is to manage energy balance during the latter half of the lactation (9).

Properly Balanced Close-Up Dry Cow Rations

Provide 6 to 10 lb. of grain daily to promote growth of the rumen papillae and allow the rumen micro flora to acclimate to grains. Feed some silage or haylage to allow adaptation to fermented feeds, if the milking rations contain them. Provide a protein balance of 15 to 16% crude protein that is 25 to 30% soluble protein and 35 to 40% rumen undegradable (18, 29). High-quality protein supplementation (e.g., blood meal, fish meal, meat meal, etc.) that provides a good supply of essential amino acids is critical. This is necessary to support gluconeogenesis.

Provide Glucose Precursors

Feed 4 to 8 oz. of propylene glycol or 8 to 12 oz. of calcium propionate if ketosis is a problem. Provide fermentable carbohydrates (e.g., corn meal, high moisture corn, barley, etc.) Corn meal is the ideal choice, because it ferments slowly.

Prompt Treatment of Fresh Cow Problems

Do not allow a fresh cow to get lost in the herd. Treat health problems promptly. Encourage fresh cows to eat. Force feed cows off-feed to prevent excessive loss of body weight.

Optimize Dry Matter Intake

Provide feed *ad libitum* to maximize feed intake during the transition period. Energy intake follows feed intake (i.e., DMI). Dry matter intake depends on many variables. They fall into three general categories: 1) environment, 2) cow, and 3) ration. Table 1 summarizes these variables.

TABLE 1. Variables that influence dry matter intake.

ENVIRONMENT	COW	RATION
Temperature	Milk production	Physical texture
Ventilation	Body size	Palatability
Humidity	Hormonal status	Fiber contest
Feedings per day	Breed	Nutrient balance
Water	Body condition	Moisture content
Sprinklers, fans, etc.	State of health	Forage quality

▪ Hypocalcemia

Description and Etiology

With the initiation of lactation, most cows experience some degree of hypocalcemia (i.e., low blood Ca) (6, 25). There are two types of hypocalcemia: clinical and subclinical (25). Hypocalcemia results from the sudden flow of Ca from blood into colostrum. There is an influx of 23 g of Ca from blood during the first 24 hours after calving. This is 9x the available Ca pool in plasma (25, 29).

In most cows activation of Ca homeorhetic mechanisms restores normal blood Ca early in the postpartum period (12, 24). If the system malfunctions, however, increases in severity and duration of hypocalcemia occur. This predisposes the cow to periparturient disorders (6, 25). Milk fever is the clinical manifestation of hypocalcemia. There is an accentuation of the degree and duration of hypocalcemia in milk fever. Clinical symptoms reflect changes in neuromuscular function (22). Initial symptoms are tremors. Subsequent neuromuscular dysfunction leads to sternal recumbency and lateral recumbency. Death generally results if not treated. It occurs in about 5 to 10% of cows (22).

Calcium homeostasis functions to maintain normal Ca concentration. These mechanisms maintain blood Ca by adjusting the supply and loss of Ca. The supply side of Ca is gut absorption and bone resorption. When compared with normal cows, milk-fever cows are the result of a breakdown on the supply side. The problem is inefficient Ca absorption from the gut and poor Ca resorption from bone (23).

Relation to Other Diseases

Parturient hypocalcemia is a risk factor for several metabolic diseases that negatively affect postpartum health and performance (1, 2, 3, 4). Cows with milk fever are 3 to 9x more likely to develop other calving disorders (e.g., dystocia, RFM, ketosis, DA=s, mastitis, and uterine prolapse) (24, 29). Risco (24, 25) showed a significant relationship between PP, Dystocia, and RFM.

Subclinical hypocalcemia is generally a greater problem than clinical hypocalcemia. Therefore, greater economic losses occur due to the non-obvious form of hypocalcemia. The uterus, rumen, and abomasum have significant smooth muscle function. Subclinical hypocalcemia can adversely affect their performance. Beede (6) described a hypocalcemia cascade that illustrates the association of hypocalcemia to other health disorders. Several studies confirm the relation of subclinical hypocalcemia to metabolic disease (1, 2, 3, 4, 26).

Treatment and Prevention

The aim of treatment is to correct the paresis and to sustain the cow until the reestablishment of normal Ca levels in the blood. Calcium deficits are generally in the amount of 8 g. Giving 500 mL of 23% calcium gluconate intravenously provides 10.8 g of calcium (25). We commonly give an additional 500 mL subcutaneously to reduce the incidence of relapses. This commonly alleviated clinical signs, but hypocalcemia remains for 2 to 3 additional days.

Cows generally respond favorably to a single Ca treatment. Other results include the following (22):

- Incidences of relapse 12 to 48 hours after treatment range from 25 to 40%.
- Ten percent will remain recumbent for 24 hours but eventually return.
- Ten percent will die or become Adowner≅ cows.

Programs for prevention of clinical hypocalcemia and correction of subclinical hypocalcemia usually revolve around 1) manipulating the Ca and P content of the diet, or 2) manipulating the ionic balance of the diet. Four programs commonly encountered in the field are as follows:

Animal Nutrition, Incorporated (ANI)

This scheme recommends high daily levels of Ca and P, 200 g and 100 g, respectively. The promoted diet is a bulky diet of alfalfa hay, oats, and wheat bran.

Dietary Electrolyte Deprivation (DED)

This is a very controversial dry cow feeding scheme. It calls for acclimation to all milk-cow feed ingredients. The aim is to restart the blood electrolyte transport mechanism by depriving cows of all major minerals the last 2 to 3 weeks before calving. No supplemental limestone, dicalcium phosphate, salt, sodium bicarbonate, magnesium oxide, etc. are fed. This method does call for vitamin and trace mineral supplementation. Some nutrients (e.g., vitamin E) are fed at increased levels.

National Research Council (NRC)

The NRC (19) recommendation for a mature dry cow during the prepartum period is 36 to 43 g per day (i.e., 0.39% of ration DM). This is below maintenance requirements. This recommendation assumes a positive Ca balance at the beginning of the dry period. Field experience reveals two basic problems with this approach: 1) This level is not low enough commonly to reduce the incidence of milk fever; 2) achieving low levels of Ca from the available feedstuffs desirable to feed dry cows is not possible. Therefore, the frequently suggested compromise is to feed <100 g of Ca and <50 g of P daily. Typically this approach is effective in preventing clinical hypocalcemia; however, in other incidences it has been completely ineffective (11).

Dietary Cation-Anion Difference (DCAD)

This scheme is a more reliable method of preventing milk fever when the Ca intake exceeds NRC requirements (1, 2, 3, 4, 5, 11, 12, 25). It is a method that balances rations for cations and anions. Synonymous names are dietary cation-anion balance, dietary electrolyte balance, cation-anion balance, strong ion balance, and fixed ion balance. It has become popular in recent years. Cows are fed an anionic ration (i.e., a negative DCAD) the last 2 to 3 weeks before calving. A negative DCAD causes mild acidosis. Following are increases in mobilization of Ca from bone and possible gut absorption of Ca from the gut (6). This increases the cows ability to maintain normal blood Ca concentrations and reduces the incidence of clinical and subclinical hypocalcemia (6). This method also calls for Ca, 120 - 180 g/day (5).

The most common formula used for DCAD is as follows:

DCAD= mEq [(%Na/0.023) + (%K/0.039)] - [(%Cl/0.0355) + (%S/0.016)]/100 g DM

For example, if the dry-matter content of a ration is 0.10%, 1.2%, 1.0% and 0.45% of sodium, potassium, chlorine, and sulfur, respectively. Calculation of DCAD is as follows:

DCAD= [(0.10/0.023) + (1.5/0.039)] - [(1.0/0.0355) + (0.45/0.016)] mEq/100 g DM
[(4.35) + (38.46)] - [(28.17) + (28.13) mEq/100 g DM -13.49 mEq/100 g DM

▪ Rumen Acidosis

Description

It is the result of acids accumulating in the rumen (20, 21, 27). It generally occurs early in lactation with a shift to high-grain rations. There are few symptoms in mild cases and the condition often goes undiagnosed. Severe overfeeding causes overt clinical signs.

There are several causes of rumen acidosis. Basically, they all relate to excessive grain feeding (20, 21, 27). It can result from feeding too much grain too quickly to fresh cows. Another cause is feeding grain before forage in component herds. It can develop from feeding forages that are cut so fine that they lack sufficient effective fiber (i.e., the ability to stimulate cud chewing).

The basic course is that grain ferments into volatile fatty acids (**VFA**) in the rumen. When production exceeds absorption of VFA, these acids accumulate in the rumen causing the pH to drop to 6 or lower (21).

There are three types based on degree of acidosis (27). They are subacute, acute, and peracute.

Subacute (Mild) Acidosis

Indigestion and off-feed problems characterize the mild form. It is a common underlying factor for ketosis and displaced abomasum. It may also produce laminitis and foot problems. Consequently, it results in fresh cows with reduced feed intake, accentuated body condition loss, and delayed return to estrus.

Acute (Moderate) Acidosis

Weight loss, poor milk production, chronic lameness, and inferior reproduction characterizes this form. Also, there is damage to the rumen lining (i.e., ulceration). This results in bacteremia and bacterial localization in the liver, lungs, and other target tissues (e.g., heart valves, joints, and kidneys). Liver and lung abscesses are common findings in cows that suffered an attack of acute acidosis.

Peracute (Severe) Acidosis

This form results from extreme over consumption of grain. Death is common without prompt veterinary intervention.

Treatment and Prevention

The difficulties of diagnosing the subacute and acute forms at the time of the insult make treatment enigmatic. Peracute acidosis demands immediate and drastic action. Rumenotomy and removal of rumen contents, followed by intensive fluid therapy is a common practice.

Prevention is always better than treatment. It should go like this.

Prepare the Rumen

The transition period is a time to equip the rumen for moderate grain feeding after parturition. Feeding 6 to 10 lb. grain during the close-up dry phase helps growth of the rumen papillae (i.e., finger-like projections of rumen epithelium). This increases the ability of the rumen to take up VFA. Also, feeding a modest amount of grain allows the rumen micro flora to adapt to highly fermentable feeds (e.g., high moisture shelled corn, ground shelled corn, barley, etc.). In addition, introducing small amounts of any silage or haylage that they are feeding to the milk cows is advisable. Remember: during the transition period we are trying to feed the feeds that will help the dairy cow to make the transition as smoothly as possible into the milking herd.

Protect the Rumen

There are two major considerations:

1) **Introduce Grain Slowly in Early Lactation**. This is a time when DMI is lagging. Also, the rumen papillae are continuing to elongate. The transition fresh-cow feeding program should not hinder either of these. There is, however, the tendency too get as much grain in the cow as quickly as possible to reduce NEB in early lactation (21). This usually results in decreased DMI and even greater NEB. Consequently, we must strive for a balance between adequate fiber and energy. With TMR herds this is just simply a matter of a properly balanced ration. Component fed herds present a difficult task. My recommendation is to increase grain slowly, 1 lb. per day until the cow reaches peak grain level. Some recommend a more conservative approach (27). They advocate feeding cows no additional grain the first week after calving. Afterwards, increase grain 0.50 to 0.75 lb. per day until peak grain level. This results in weekly grain increases of 3 to 5 lb. Anecdotal experience is that this is a very conservative approach and may allow for excessive weight loss, ketosis, and fatty liver.

2) **Reduce the Acid Produced after Each Meal**. The properly formulated TMR is the best way to accomplish this (27). This permits a constant ratio of forage to grain. However, even with a TMR, providing adequate effective fiber is absolutely necessary. That is, fiber that promotes cud chewing. This requires forages that have adequate particle length. Fifteen to 20 percent of the pieces should be greater than 1.5 inches long. With component-fed herds, avoid ‘slug

feeding' of grain. Divide grain into three or four daily feedings. Always feed forage before grain. Buffers may also help to maintain pH. Use buffers as aids, not as substitutes for good nutritional management.

Summary

- The transition period is from three weeks before to three weeks after calving.
- It is a critical time in the life of the dairy cow. During this time the modern dairy animal makes a great metamorphosis. She moves from a dry cow with marginal nutrient requirements to a lactating cow with massive metabolic needs.
- How well she makes this transition affects her health, production and reproduction.
- Our challenge is to feed and manage the modern dairy cow so that she makes this progression smoothly.
- Fatty liver, hypocalcemia, and rumen acidosis are metabolic diseases that adversely affect reproduction.
- Transition management and feeding programs that successfully reduce their incidence will generate greater lactational and reproductive performance.

References

1. Beede, D. K. 1992. Formulation strategies for cation-anion difference in diets of late pregnant cows. Page 137 *in* Proc. Large Dairy Herd Manag. Symp. H. H. Van Horn and C. J. Wilcox, ed. Manage. Serv. Am. Dairy Sci. Assoc., Champaign, IL.
2. Beede, D. K. 1992. Macrominerals. Page 272 *in* Proc. Large Dairy Herd Manag. Symp. H. H. Van Horn and C. J. Wilcox, ed. Manage. Serv. Am. Dairy Sci. Assoc., Champaign, IL.
3. Beede, D. K., C. A. Risco, G. A. Donovan, C. Wang, L. F. Archibald, and W. K. Sanchez. 1991. Nutritional management of the late-pregnant dry cow with particular reference to dietary cation-anion difference and calcium supplementation. Proc. 24th An. Convent. Am Assoc. Bovine Pract, p. 51.
4. Beede, D. K., C. Wang, G. A. Donovan, L. F. Archibald, and W. K. Sanchez. 1991. Dietary cation-anion difference (electrolyte balance) in late pregnancy. Proc. Florida Dairy Product. Conf. p. 1.
5. Beede, D. K. 1992. The DCAD concept: Transition rations for dry pregnant cows. Feedstuffs 64 (53):12.
6. Beede, D. K. 1995. Macromineral element nutrition for the transition cow: practical implications and strategies. Proc. Tri-State Nutr. Conf. p. 175.

7. Bell, A. W., M. B. Rymph, R. Slepetis, W. A. House and R. A. Ehrhardt. 1992. Net nutrient requirements for conceptus growth in Holstein cows - implications for dry cow feeding. Proc. Cornell Nutr. Conf. p. 102.
8. 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.
9. Chandler, P. 1995. Energy needs of lactating dairy cow must be frequently evaluated. Feedstuffs, Apr. 10 p. 14.
10. Ferrell, C. L. And S. P. Ford. 1980. Blood flow, steroid secretion, and nutrient uptake of the gravid bovine uterus. J. Anim. Sci. 50:1113.
11. Gerloff, B. J. 1988. Feeding the dry cow to avoid metabolic disease. Vet. Clinics North Am. Food Anim. Pract. 4:379.
12. Goff, J. P. 1992. Cation-anion difference of diets and its influence on milk fever and subsequent lactation: the good and the bad news. Proc. Cornell Nutr. Conf. p. 148.
13. 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.
14. Grummer, R. R., and D. J. Carroll. 1991. Effect of dietary fat on metabolic disorders and reproductive performance in dairy cattle. J. Anim. Sci. 69:3838.
15. Grummer, R. R. 1993. Etiology of lipid-related metabolic disorders in periparturient dairy cows. J. Dairy Sci. 76:3882.
16. Herdt, T. H. 1988. Fuel homeostasis in the ruminant. Vet. Clinics North Am. Food Anim. Pract. 4:213.
17. Herdt, T. H. 1988. Fatty liver in dairy cows. Vet. Clinics North Am. Food Anim. Pract. 4:269.
18. McNeill, D. M., R. Sleptis, D. M. Smith, R. A. Ehrhardt and A. W. Bell. Protein requirements in late pregnancy: partitioning of nitrogen between the gravid uterus and maternal tissues. Proc. 1994 Proc. Cornell Nutr. Conf. p. 117.
19. National Research Council. 1989. Nutrient requirements of dairy cattle 6th rev. ed. National Academy Press, Washington, DC.
20. Nocek, J. E. 1995. Energy metabolism and rumen acidosis. Proc. Tri-State Nutr. Conf. p. 155.
21. Nordlund, K. V. 1995. Herd based rumenocentesis: A clinical approach to the diagnosis of subacute rumen acidosis. Proc. Northeast Dairy Prod. Symp. p. 1.
22. Oetzel, G. R. 1988. Parturient paresis and hypocalcemia in ruminant livestock. Vet. Clinics of North Am. Food Anim. Pract. 4:351.
23. Oetzel, G. R. and J. A. Barmore. 1992. Palatability of anionic salts fed in a concentrate mix. J. Dairy Sci. (Supp. 1). 75:797.
24. Risco, C. A. 1992. Calving related disorders. Page 192 *in* Proc. Large Dairy Herd Manag. Symp. H. H. Van Horn and C. J. Wilcox, ed. Manage. Serv. Am. Dairy Sci. Assoc., Champaign, IL.
25. Risco, C. A. 1995. Nutritional management of dry cows and calving related disorders. Proc. Acad. Dairy Vet. Consult. p. 1.

26. Sanchez, W. K., D. K. Beede, and M. A. DeLorenzo. 1992. Modeling the effects of macrominerals on lactational performance of dairy cattle. Proc. Nutr. Inst, Nat. Feed Ingred. Assoc. p. 1.
27. Schultz, L. H., L. Allenstein, and G. R. Oetzel. 1993. Fresh cow problems. W. D. Hoards & Sons Company, Fort Atkinson, WI.
28. Shearer, J. K. and H. H. Van Horn. 1992. Metabolic diseases of dairy cattle. Page 358 *in* Proc. Large Dairy Herd Manag. Symp. H. H. Van Horn and C. J. Wilcox, ed. Manage. Serv. Am. Dairy Sci. Assoc., Champaign, IL.
29. Van Saun, R. J. 1991. Dry cow nutrition. Vet. Clinics North Am. Food Anim. Pract. 7:599.