Nutritionally related disorders affecting feedlot cattle[1]

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Galyean, M. L. and Rivera, J. D. 2003. **Nutritionally related disorders affecting feedlot cattle**. Can J. Anim. Sci. **83**: 13–20. Digestive disorders account for approximately 25 to 33% of deaths in feedlot cattle and likely contribute to decreased performance and efficiency of production. A variety of nutritional, management, genetic, behavioural, and environmental factors seem to be involved in the development of metabolic disorders in feedlot cattle. Excessive production of acid in the rumen is often either the cause of or a significant contributing factor to metabolic and nutritional disorders, including acute or sub-acute (chronic) acidosis, liver abscesses, and feedlot bloat. Decreasing the percentage of highly fermentable concentrates in feedlot diets by increasing roughage level or limiting feed intake should decrease the incidence of these disorders, but this approach is usually not economically feasible. Careful feed bunk management is often thought to be important for decreasing the incidence of nutritionally related disorders, but research to support its importance is limited and conflicting. Certain feed additives like ionophores seem to be effective for decreasing the incidence of acidosis and feedlot bloat, presumably through decreased total feed intake, smaller and more frequent meals, and direct effects on the ruminal microbial population. The incidence of liver abscesses can be decreased by the feeding of various antibiotics. Polioencephalomalacia (PEM) can result from several causes, including thiamine deficiency; however, recent data suggests that overproduction of H_2S in the rumen is an important cause of PEM when intakes (feed and water) of sulfur are high in feedlot cattle.

Key words: Beef cattle, acidosis, bloat, liver abscesses, polioencephalomalacia

Galyean, M. L. et Rivera, J. D. 2003. **Troubles nutritionnels des bovins élevés en parc d'engraissement**. Can J. Anim. Sci. **83**: 13–20. Les affections du système digestif expliquent de 25 à 33 % des morts de bovins dans les parcs d'engraissement et concourent sans doute à une baisse du rendement et de la productivité. Chez les bovins élevés en parc d'engraissement, les troubles du métabolisme semblent résulter d'une série de facteurs nutritionnels, zootechniques, génétiques, comportementaux et environnementaux. La surproduction d'acide dans le rumen se trouve souvent à l'origine des troubles métaboliques ou nutritionnels comme l'acidose aiguë ou sub-aiguë (chronique), les abcès du foie et le météorisme, ou constitue l'un des principaux facteurs à leur origine. En diminuant la proportion de concentrés qui fermentent très facilement dans la ration des animaux par l'addition d'une plus grande quantité de fourrages grossiers ou en limitant l'ingestion des aliments, on devrait atténuer l'incidence des troubles nutritionnels, mais pareille approche s'avère rarement réalisable sur le plan économique. On croit souvent qu'une gestion méticuleuse de l'approvisionnement des mangeoires concourt largement à réduire l'incidence des troubles nutritionnels, cependant les recherches qui pourraient confirmer cette impression sont peu nombreuses et leurs résultats contradictoires. Quelques additifs tels les ionophores paraissent réduire l'incidence de l'acidose et du météorisme, sans doute parce qu'ils diminuent la prise alimentaire, incitent l'animal à manger moins mais plus souvent ou agissent directement sur la microflore du rumen. On peut aussi combattre les abcès du foie en administrant divers antibiotiques. La polioencéphalomalacie a plusieurs origines, y compris une carence en thiamine, mais des données récentes laissent croire que la surproduction de H_2S dans le rumen joue un rôle important dans son étiologie quand les bovins absorbent beaucoup de soufre (présent dans l'eau et les aliments).

Mots clés: Bovins de boucherie, acidose, météorisme, abcès du foie, polioencéphalomalacie

Nutritionally related metabolic disorders in feedlot cattle are often complex disease conditions that typically occur at low frequencies. In a recent survey [National Animal Health Monitoring System survey (NAHMS); USDA 2000b], averaged over all types of feedlots, 1.9% of cattle placed in feedlots developed digestive disease conditions, with values of 1.1% for feedlots with a capacity of 1000 to less than 8000 animals and 2.0% for feedlots with a capacity greater than 8000. The incidence of mortality from nutritionally related metabolic disorders also is typically low. Smith (1998) reviewed three summaries of mortality data from large feedlots and indicated that mortality ranged from 0.17 to 0.419% of inventory per month and that digestive disorders accounted for 30 to 42.4% of all mortality. Greater mortality was noted with Holstein cattle than with beef breeds (Smith 1998). Digestive death rates seem to vary somewhat with feedlot size (Fig. 1; Vogel and Parrott 1994), being slightly greater with larger-capacity feedlots. In the NAHMS survey, the average cost of treating animals diagnosed with digestive problems was US$6.19 (USDA 2000b).

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Abbreviations: **BW**, body weight; **DM**, dry matter; **PEM**, polioencephalomalacia; **VFA**, volatile fatty acids

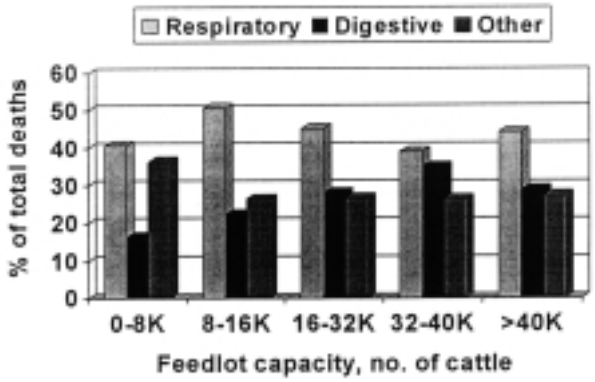

Fig. 1. Metabolic death losses in feedlots of varying size. Adapted from Vogel and Parrott (1994). Mortality as a percentage of occupancy per month averaged 0.326, 0.323, 0.265, 0.239, and 0.204 for capacities of 0 to 8K, 8 to 16K, 16 to 32K, 32 to 40K, and > 40K, respectively, where K = $\times 10^3$.

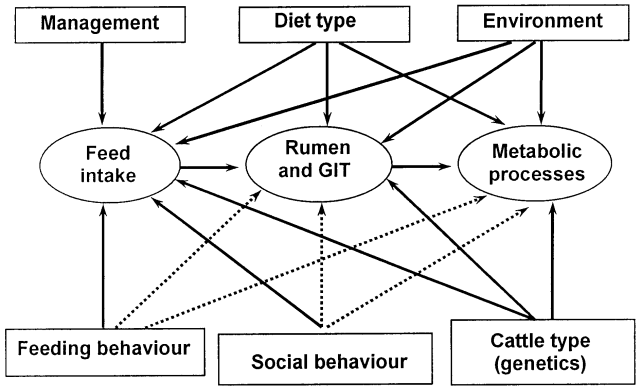

Fig. 2. Possible factors and interrelationships among factors that affect nutritionally related metabolic disorders in feedlot cattle. Solid arrows signify relationships that are known to exist, whereas dotted arrows represent probable and(or) possible relationships. Adapted and modified from Galyean and Eng (1998). GIT = gastrointestinal tract. This component includes effects of overall GIT physiology, and ruminal/intestinal microbial populations.

Research and diagnostic efforts with feedlot metabolic disorders has been hampered by both their complexity and low rates of occurrence. Galyean and Eng (1998) outlined several factors and interrelationships among those factors that potentially affect metabolic disorders in feedlot cattle. Figure 2 is an updated and modified version of one presented by Galyean and Eng (1998). Their approach was based on the idea that metabolic disorders occur as a result of changes in the rumen or other parts of the digestive tract and in metabolic processes, and that these changes are a function of various interrelated external factors (such as environment, management, diet, and so on). Galyean and Eng (1998) recognized that the role of some individual components (e.g., diet type and environment) is understood better than others (e.g., feeding and social behaviour), and that potential interactions among these components often are poorly described. They concluded that a greater understanding of the etiology of metabolic disorders of feedlot cattle will require a multidisciplinary research approach; increased insight into digestive, microbial, and metabolic aspects of disorders; definition of potential interactions among factors that affect these disorders; and identification and elucidation of currently unknown aspects of these disorders.

This review will focus on describing the cause(s) of the most prevalent nutritional and metabolic disorders that occur in feedlot cattle, including acidosis, liver abscesses, and bloat. In addition, PEM, a feedlot disorder that can be linked to nutrition in many cases, will be considered. Efforts will be made to identify areas in need of further research.

ACIDOSIS

Importance and Etiology

Acidosis occurs as a consequence of the extensive use of highly processed grain and low levels of roughage in beef cattle finishing diets. In the NAHMS survey of nutritional and management practices in US feedlots (USDA 2000a), 61.3% of feedlots with a capacity of greater than 8000 animals used steam-flaked (or steam-rolled) grain in finishing diets. Summaries of in vivo data have documented that ruminal, intestinal, and total tract digestibility of starch is greater with steam-flaked corn and sorghum than with their dry-rolled counterparts (Huntington 1997). Processing cereal grains by heat and moisture methods like steam flaking gelatinizes starch, increasing both the rate and extent of starch fermentation in the rumen, with associated acid production and increased risk for acidosis.

Owens et al. (1998) described both acute and sub-acute acidosis as conditions that can occur in cattle fed high-concentrate diets. Acute acidosis is an overt illness that occurs in conjunction with consumption of an excessive quantity of readily fermented carbohydrate, which increases the total acid supply from the rumen (Owens et al. 1998). Lactic acid, normally found at very low concentrations in the rumen, can increase substantially, along with increased concentrations of volatile fatty acids (VFA). As a result of the increasing acid load, cattle with acute acidosis experience a drastic decrease in ruminal pH, with a pH of 5.2 considered as a benchmark (Owens et al. 1998). Absorption of acids into the bloodstream can lead to systemic acidosis, stiffness in the legs, and laminitis or founder (Nocek 1997), severe ruminal lesions, and sometimes death. In contrast to acute acidosis, cattle with sub-acute acidosis do not typically show outward signs of illness, but often exhibit decreased feed intake and lower performance (Owens et al. 1998). In sub-acute acidosis, ruminal pH drops substantially, and decreased feed intake follows the severity of the acid load. A ruminal pH of 5.6 has been considered as a benchmark of sub-acute acidosis (Owens et al. 1998).

In their excellent review article, Owens et al. (1998) described the sequence of events leading to acidosis (Fig. 3) and discussed potential points of control. The disease process begins when the animal consumes a large quantity of starch or other rapidly fermented carbohydrate. Glucose, normally found at very low concentrations in the rumen, is

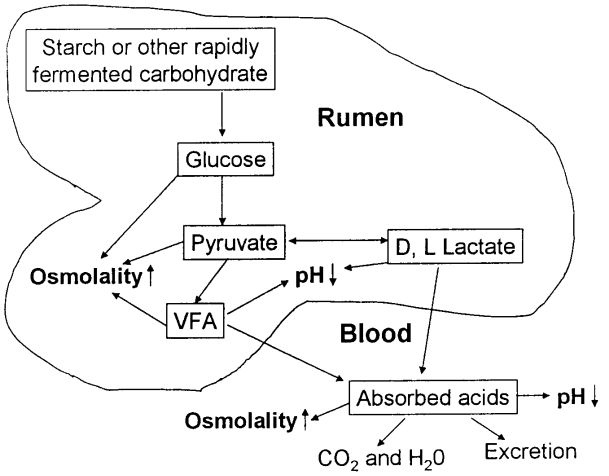

Fig. 3. The etiology of acidosis in cattle. Adapted from Owens et al. (1998).

liberated from starch, resulting in ruminal glucose concentrations that can exceed those in the blood. These high glucose concentrations can cause several negative consequences, including: (1) growth of organisms like *Streptococcus bovis* and other microbes that produce lactic acid; (2) growth of coliforms and amino-acid decarboxylating microbes that produce endotoxins and amines like histamine that can subsequently contribute to the development of laminitis (Nocek 1997); and (3) increased ruminal osmolality, which exacerbates ruminal acidity by inhibiting VFA absorption from the rumen (Owens et al. 1998). For reasons unknown at present, ruminal glucose concentrations remain high for an extended period during acute acidosis. Glucose is subsequently fermented via glycolysis through pyruvate to lactate or VFA. Ruminal microbes that ferment glucose to lactate tend to be fairly insensitive to low pH, whereas most lactate-utilizing microbes in the rumen are sensitive to low pH (Owens et al. 1998), thereby favoring production and accumulation of lactate. Lactate is produced as both the D and L isomers, with concentrations of greater than 40 mM considered reflective of severe acute acidosis (Owens et al. 1998). Total VFA concentrations increase along with lactate, and ruminal pH decreases substantially because increased ruminal osmolality decreases absorption of acids. As pH continues to decrease and acid concentrations increase, absorption proceeds by passive diffusion. The capacity of the ruminal wall and liver to metabolize acids can be overwhelmed, which leads to high blood concentrations of acids that can potentially overpower the bicarbonate buffering system. In addition, blood osmolality increases because increased ruminal osmolality pulls fluid from the blood into the rumen and because acids that have accumulated in the blood have a direct effect on osmolality. If the animal is unable to restore homeostasis, death can result; however, even when animals recover, damage to the ruminal epithelium, laminitis, and subsequently lowered or erratic feed intake can have significant long-term effects on productivity.

Factors Affecting Acidosis

In commercial feedlots, cattle are adapted gradually to a final high-grain diet. This adaptation process is designed to minimize the risk of acidosis. It is presumed that careful bunk management designed to provide cattle with relatively constant day-to-day feed intake during the adaptation period is important in preventing acute and sub-acute acidosis. However, because pens of cattle are fed in commercial feedlots, and wide swings in intake by individuals within the pen are possible, this transition period represents the time during which acidosis is most likely to occur. Roughages typically have lower fermentable carbohydrate content than grains do, so increasing roughage decreases the chance for an acute ruminal acid load. The ability of roughage to stimulate chewing and saliva flow also should aid in moderating ruminal pH. Inherent buffering capacity of roughage could be a factor, but Allen (1997) suggested that buffering by feeds would most likely occur at a pH less than 5 and that the potential for direct buffering by dietary ingredients was a small fraction of buffering by saliva. Sources of fiber other than those provided by traditional roughages might have beneficial effects on acidosis. For example, based on results with individually fed cattle used to measure variation in feed intake and on ruminally cannulated cattle exposed to an acidosis challenge model, Krehbiel et al. (1995) concluded that a digestible fiber source like wet corn gluten feed did not eliminate acidosis but decreased the time that cattle were exposed to an acid insult.

Animal-to-animal variation in the ability to handle an acid load seems fairly substantial, even in model systems where attempts have been made to apply a relatively constant acid load. For example, Brown et al. (2000) used a sub-acute acidosis model that involved adapting ruminally cannulated steers (five per treatment) to a 50% concentrate diet at a restricted intake [1.7% of body weight (BW)], withholding feed the day before the challenge, and then dosing a challenge diet (all-concentrate, wheat:corn mixture) into the rumen at a rate of 1.5% of BW. Similarly, the acute acidosis model used by Brown et al. (2000) involved adapting five ruminally cannulated steers to grass hay, withholding feed the day before the challenge, and then dosing steam-flaked corn (3% of BW) into the rumen over a 4-h period (0.75% of BW h^{-1}). After analyses of the ruminal and blood samples taken following the challenges, Brown et al. (2000) concluded that, on the basis of ruminal pH, ruminal and plasma lactate concentrations, and dry matter (DM) intake, two of the five acute-challenge steers did not experience acute acidosis, and one of these two did not experience sub-acute acidosis. Likewise, only one of the five sub-acute-challenge steers experienced sub-acute acidosis. These results suggest that beef cattle have a fairly remarkable ability to adapt to abrupt changes in acid load. Moreover, given the degree of variation among animals noted by Brown et al. (2000), the relatively low rates of occurrence of and mortality from digestive disorders in feedlots noted previously are not surprising.

Although our understanding of the sequence of events leading to acute acidosis is reasonably complete, our understanding of sub-acute acidosis is not. Low or erratic feed

intake and decreased BW gain are often associated with sub-acute acidosis, but results of production-level research to verify these effects are limited. Feed bunk management is an important aspect of nutritional consulting (Galyean 1996); however, the extent to which feeding management systems affect the incidence of metabolic disorders like acidosis is largely unknown. Research is needed to identify the relationships between feeding management practices (e.g., restricted feeding, programmed feeding, and clean bunk management), feeding patterns, and the incidence of acidosis. Variation in feed intake has been suggested to increase the incidence of metabolic disorders like acidosis (Bauer et al. 1995), and Galyean et al. (1992) found that varying intake decreased performance by feedlot cattle. Nonetheless, Zinn (1994) reported no effects, and in some cases slight positive effects, of variation in intake on feedlot cattle performance. Galyean and Eng (1998) suggested that given the complexity of metabolic disorders like acidosis, a comprehensive evaluation of the relationship of feed intake variation to performance and the incidence of acidosis and other metabolic disorders is needed.

Feeding behaviour might affect acidosis, but the extent of its impact is largely a matter of speculation because research in this area is limited. Because feedlot cattle are typically fed in large groups, social behaviour patterns (e.g., dominance structure in a pen of cattle, role models, previous experience, and so on) might affect eating patterns and the incidence of metabolic disorders like acidosis. Research is needed to establish baseline data on the feeding and social behaviours of beef cattle fed high-concentrate diets. Studies conducted under commercial feedlot conditions in large pens would likely be the most useful. The effects of previous nutritional history on feeding behaviours and potential interactions of feeding behaviour with nutrition and management need to be evaluated. If feeding and social behaviour patterns affect the incidence of metabolic disorders, quantifying their effects should allow development of more effective model systems that would allow the use of individual animals to mimic metabolic disorders in feedlot cattle.

Feed additives can be an important tool for managing acidosis. Monensin has been shown to decrease day-to-day variation in feed intake (Stock et al. 1995), which, coupled with a potentially greater frequency of smaller meals, a lower total feed intake, and direct effects on inhibition of lactate-producing bacteria (Coe et al. 1996; Owens et al. 1998), might decrease the odds of high acid production in the rumen. Ionophores like lasalocid and laidlomycin propionate do not seem to have major effects on feed intake (NRC 1996), and their effect on acidosis and other metabolic disorders is not as well documented as with monensin. The role that dietary buffers play in feedlot acidosis is not well defined, nor is the role of dietary cation-anion difference (Galyean and Eng 1998). Changes in dietary cation-anion difference might affect both ruminal acid production and the ability of the animal to compensate for a metabolic acid load. Further research with buffers and effects of dietary cation-anion difference is needed.

LIVER ABSCESSES

Importance and Etiology

Liver abscesses are a common disorder in feedlot cattle in the United States, with occurrence rates ranging from 0 to nearly 100% (Nagaraja and Chengappa 1998). Liver abscesses accounted for 46% of liver condemnations in the United States, which was more than double the rate for liver flukes (22%), the second highest cause of condemnation (Nagaraja and Chengappa 1998). The 1998–1999 Canadian Beef Quality Audit [Canadian Cattlemmen's Association (CCA) 2002] indicated that 14% of livers were condemned in Canadian processing plants, 14% were approved for use in pet food, and 72% were approved for human consumption. Liver abscesses were responsible for the majority of condemnations (CCA 2002). The occurrence of liver abscesses is generally considered to be closely related to ruminal acidosis because it is thought that a continual high acid load in the rumen can lead to parakeratosis or similar insults to the ruminal wall, leading, in turn, to clumping and necrosis of ruminal papillae (Ørskov 1986). These ulcerative lesions, along with hairs or other foreign objects that become embedded in the ruminal epithelium, provide routes of entry into the portal system for microbes that cause liver abscesses; however, the exact mechanism of pathogenesis is unknown (Nagaraja and Chengappa 1998). The incidence of liver abscesses can be high in certain geographical regions of the United States, especially the Midwest feeding belt and northern Great Plains region. Affected livers are condemned at slaughter, which represents a loss of carcass weight of approximately 2% (Nagaraja and Chengappa 1998). Cattle with severely abscessed livers (one or more large, active abscesses) have a lower BW gain and are less efficient than cattle with no abscesses, but performance by cattle with less severely abscessed livers (but nonetheless condemned at slaughter) is not affected greatly (Brink et al. 1990; Table 1).

Fusobacterium necrophorum is the chief organism cultured from liver abscesses in cattle, with *Actinomyces pyogenes* being a distant second among isolated pathogens (Nagaraja and Chengappa 1998). *F. necrophorum* is a Gram-negative, anaerobic rod that normally inhabits the rumen, and it is found both in the ruminal contents and associated with the ruminal wall (Nagaraja and Chengappa 1998). It uses lactate as a substrate (Nagaraja and Chengappa 1998), which coincides with its presence in the rumen of cattle fed high-grain diets and the potential link to acidosis. *F. necrophorum* produces several toxins and virulence factors that have been described in detail by Nagaraja and Chengappa (1998).

Controlling Liver Abscesses

Liver abscesses are visible at slaughter, and as noted previously, only severe abscesses affect animal performance; hence, pre-slaughter detection of feedlot cattle with abscesses is virtually impossible. Ultrasonography has proven useful for monitoring experimentally induced abscesses, but its value in practice has been deemed negligible (Nagaraja and Chengappa 1998). Because detection of liver abscesses in the live animal is impractical, prevention is the only feasible approach.

Table 1. Effects of liver abscesses on feedlot performance in beef cattle[z]

Item[x]	Liver abscess severity score[y]			
	None	A–	A	A+
No. of steers	405	52	37	72
Days on feed	131 (0.8)	133 (2.1)	131 (2.1)	131 (1.4)
Initial BW (kg)	305 (1.9)	320 (4.8)	316 (5.6)	311 (3.4)
Final BW (kg)	470 (2.1)	484 (5.6)	477 (7.2)	461 (4.8)
Hot carcass wt. (kg)	289 (1.4)	298 (3.9)	293 (4.7)	276 (3.1)
ADG (kg d^{-1})	1.27 (0.01)	1.23 (0.04)	1.24 (0.04)	1.15 (0.03)
DMI (kg d^{-1})	8.39 (0.06)	8.27 (0.19)	8.42 (0.15)	7.96 (0.50)

[z]Adapted from Brink et al. (1990).
[y]A– = one to two small abscesses; A = two to four small, active abscesses; A+ = one (or more) large, active abscess.
[x]Numbers in parentheses are standard errors. No differences were significant ($P < 0.05$) in the overall data set; however, significant differences were detected in subsets of the data. See Brink et al. (1990) for details.

Increasing dietary roughage level decreases the quantity of fermentable starch in the diet, thereby decreasing the ruminal acid load, which should decrease the incidence of liver abscesses. Although some studies have shown a relationship between roughage level and the incidence of liver abscesses (Gill et al. 1979; Zinn and Plascencia 1996), others have not (Kreikemeier et al. 1990). In addition, the practical potential to increase roughage levels in feedlot diets is small given the advantage in cost per unit energy for grains over roughages. Thus, it is generally believed that careful adaptation of cattle from high-roughage to high-grain diets should be practiced to lessen the risk of acidosis and liver abscesses. Nonetheless, as noted with acidosis, the role of adaptation or step-up programs and feed bunk management in the development of liver abscesses has not been extensively studied.

The addition of antibiotics to the feed is the primary means of controlling liver abscesses in feedlot cattle. In the United States, bacitracin methylene disalicylate, chlortetracycline, oxytetracycline, tylosin, and virginiamycin are approved for prevention of liver abscesses (Nagaraja and Chengappa 1998), with tylosin (8.8 to 11 mg kg^{-1} of diet or 60 to 90 mg $animal^{-1}$ daily) being the most effective and most commonly used of these antibiotics. Decreases in liver abscess rates in the order of 75% have been reported in commercial-scale studies with tylosin (Nagaraja and Chengappa 1998). To date, efforts to develop vaccines as a means of controlling liver abscesses have been less successful than using antibiotics like tylosin (Nagaraja and Chengappa 1998).

FEEDLOT BLOAT

Importance and Etiology

Bloat is often reported as a cause of death in feedlot cattle, and it has been recognized as an important component of mortality associated with digestive disorders (Nagaraja et. al. 1998). Bloat occurs when the animal is prevented from expelling ruminal gas, which subsequently places pressure on the diaphragm and lungs, thereby affecting breathing and potentially resulting in death. Feedlot bloat is characterized as frothy, with the formation of stable foam. Eructation of gas through the esophagus is inhibited when the cardia is covered with foam (Cheng et al. 1998). Free-gas bloat, which usually occurs as a result of physical obstruction or damage to the cardia or esophagus, or as a result of decreased ruminal motility, occurs in feedlot cattle, but frothy bloat is more common (Cheng et al. 1998). Feedlot (frothy) bloat often occurs during adaptation to high-grain diets (Nagaraja et al. 1998), but sporadic outbreaks have been reported in cattle that are well adapted to a finishing diet. These "bloat blizzards" as they are sometimes called by frustrated nutritionists and feedlot managers are typically of limited duration and almost always of unknown cause(s).

With frothy pasture (e.g., alfalfa or annual winter wheat) bloat, high soluble protein concentrations in the forage, along with rapid fermentation of the forage and possible animal factors are thought to contribute to the formation of stable foam. With highly fermentable grain-based diets, however, microbial factors seem to be responsible for the formation of frothy (stable foam) contents (Nagaraja et al. 1998). Production of mucopolysaccharide slime by ruminal bacteria, coupled with release of undefined macromolecules when bacterial cells are lysed, increases ruminal fluid viscosity and contributes to development of stable foam (Cheng et al. 1998). *Streptococcus bovis* is known to produce slime, and increased numbers of *S. bovis* have been reported with bloat (Cheng et al. 1998); however, other bacteria might be involved, and the microbial changes leading to and associated with feedlot bloat need to be described more completely.

Factors Affecting Feedlot Bloat

An overview of factors affecting feedlot bloat, adapted from Nagaraja (1994) is shown in Fig. 4. As with acidosis and liver abscesses, feedlot bloat is largely a disease that results from the feeding of high-grain diets. Thus, factors associated with the feeding of grains will affect the incidence and severity of bloat. Because cereal grains differ in the rate and extent of fermentation of starch, differences exist in the rate of acid and gas production among grains. Among cereal grains, the ranking of ruminal and total tract starch digestion is oats = wheat > barley > corn > sorghum (Huntington 1997). Differences in the nature of endosperm protein also exist among grains, which are associated with differences in ruminal starch digestion. Theurer (1986) indicated that the total tract protein digestibility of corn, sorghum, and barley was inversely related to ruminal and total tract starch diges-

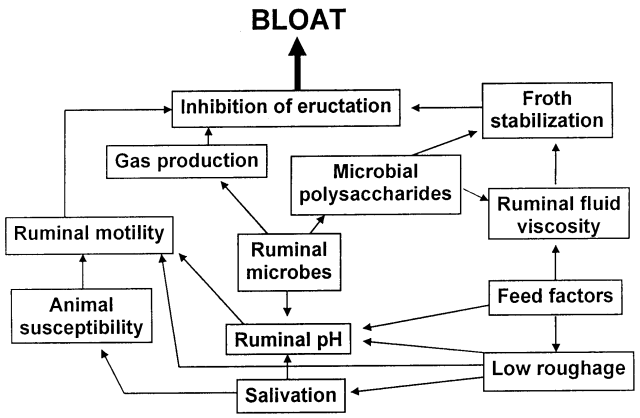

Fig. 4. Factors affecting feedlot bloat. Adapted from Nagaraja (1994).

tion. Wheat is often considered the most bloat-provocative grain in feedlot diets (Cheng et al. 1998). Indeed, wheat is rarely fed as the sole dietary grain, typically being mixed with corn or sorghum in practical feedlot diets.

Grain processing increases the rate and extent of starch digestion, thereby potentially affecting the incidence of bloat. Fine particle size seems to promote frothiness, perhaps through accelerated production of bacterial slime (Cheng et al. 1998). Moreover, smaller particles of both gelatinized and ungelatinized grain are digested at a faster rate than larger particles are (Galyean et al. 1981), leading to higher gas production. Care should be taken in processing and milling of grains to avoid creation of fine particles. One alternative for decreasing the potential for feedlot bloat is to feed less highly processed grain; however, performance responses, such as ADG and feed efficiency, to processing methods like steam flaking are large (Owens et al. 1997) and, therefore, feeding less extensively processed grain to avoid feedlot bloat is probably not an economically viable alternative.

As with acidosis and liver abscesses, adding roughage to the diet should slow the overall rate of fermentation, stimulate salivation, and lower acid production, thereby decreasing the chances of bloat. Feedlot bloat often occurs as cattle are transitioned from high-roughage, starter diets to high-grain, low-roughage finishing diets (Cheng et al. 1998). Careful, step-wise transitions should aid in decreasing the incidence of bloat, and higher roughage levels in finishing diets should prove beneficial. As noted with acidosis and liver abscess, however, increasing the level of roughage beyond a certain point is not economically feasible because of the high cost of energy from roughages vs grains and the potential for bulk to limit energy intake. Even so, in commercial feedlots, roughage level is sometimes increased temporarily as a means of bloat control at times when the incidence of feedlot bloat is extremely high. More research should be conducted to evaluate the effect of different roughage sources on feedlot bloat, but at present, there is no basis for choosing among roughages with respect to their ability to decrease feedlot bloat. The consistency of roughages, even within the same roughage type, further decreases the potential to use roughage source and level as a routine means of managing the incidence of feedlot bloat.

Ionophores, most notably monensin, seem useful for decreasing the incidence of feedlot bloat. Feeding monensin decreases the incidence of frothy bloat on irrigated wheat pasture (Branine and Galyean 1990). These compounds inhibit many of the Gram-positive bacteria that produce lactate and mucopolysaccharide slime (Cheng et al. 1998). In addition, effects of monensin on eating patterns and day-to-day variation in feed intake (Stock et al. 1995) of cattle might be beneficial for decreasing feedlot bloat. If the benefits of monensin on feedlot bloat occur totally through effects on feed intake, restricting intake might be equally beneficial, but this has not been tested in a research setting. Poloxalene, an antifoaming agent, is very effective for control of pasture bloat; however, the incidence of feedlot bloat is decreased but not eliminated by poloxalene (Cheng et al. 1998). Animal susceptibility to bloat varies, presumably as a result of differences in anatomy, ruminal motility, eating rate, production of saliva, and other unknown factors (Nagaraja et al. 1998).

POLIOENCEPHALOMALACIA

Outbreaks of PEM, a neurological disorder of ruminants, occur sporadically in feedlots. Clinically, the disease is characterized by blindness, incoordination, muscle tremors, and possible recumbency with seizures (Blood and Radostits 1989; Gould 1998). As noted by Gould (1998), the term PEM is used in two ways: (1) to describe a specific disorder caused by altered thiamine status, and (2) to describe a specific brain lesion that can result from various causes. Indeed, characteristic brain lesions are evident at post-mortem examination (Blood and Radostits 1989), and cerebrocortical necrosis is synonymous with PEM (Gould 1998).

The role of thiamine in PEM has been studied for many years; however, questions remain as to exactly how thiamine is involved in PEM. Animals treated at the onset of PEM symptoms with intravenous thiamine hydrochloride often respond and recover, and thiamine deficiency induced by production of thiaminase or thiamine anti-metabolites in the rumen has been implicated (Blood and Radostits 1989). Although decreased concentrations of thiamine have been noted in some studies, others have shown no relationship between tissue and ruminal fluid thiamine concentrations and PEM, and severe thiamine deficiency has not induced PEM (Gould 1998). Lead poisoning and salt toxicosis also result in brain lesions characteristic of PEM (Loneragan et al. 1998), with diagnosis of lead poisoning dependent on evaluation of blood and tissue lead concentrations (Gould 1998). Possible causes of PEM are shown in Fig. 5.

Recent research (McAllister et al. 1997; Gould 1998; Loneragan et al. 1998) indicates that PEM can be caused by high levels of sulfur in the water or feed, with subsequent over-production of hydrogen sulfide (H_2S) in the rumen, accumulation of H_2S in the ruminal gas cap, and absorption into the blood stream. Sulfur intake from all sources, including water, should be considered, with the maximum tolerable level of 0.4% of dietary DM (NRC 1996) used as a

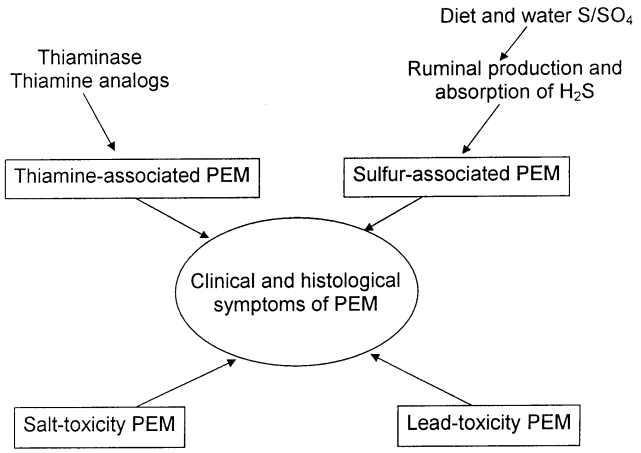

Fig. 5. Possible causes of polioencephalomalacia (PEM) in cattle.

guideline. High intakes of sulfates seem to increase the ability of ruminal microbes to produce H_2S, and consideration should be given to dietary S-containing amino acids as contributors to the H_2S pool (Gould 1998). Acidic conditions in the rumen favor higher concentrations of H_2S in the gas cap (Gould 1998), and factors affecting ruminal acidity discussed in previous sections on acidosis, liver abscesses, and feedlot bloat are likely relevant to the development of PEM.

Even in the absence of clinical cases of PEM, high concentrations of sulfate in the drinking water of feedlot cattle can negatively affect BW gain and hot carcass weight. Loneragan et al. (2001) reported that averaged over a 113-d feeding period, sulfur intakes (from all sources) equivalent to 0.29% of the diet were detrimental to feedlot performance and carcass characteristics.

Adaptation of cattle to higher concentrate diets also might affect the incidence of PEM. McAllister et al. (1997) determined that the incidence of PEM in cattle was greatest between 15 and 30 d after entering the feedlot. Perhaps changes in ruminal pH that occur with the adaptation of cattle to high-concentrate diets alter the incidence of PEM through effects on thiaminase activity or hydrogen sulfide production.

Further research is needed to more clearly define the dietary and management conditions that lead to sulfate-related PEM. In a field-study, the incidence of sulfur-associated PEM was highest in the first 30 d after cattle entered the feedlot, with virtually all cases occurring before the cattle had been on feed for 60 d (McAllister et al. 1997). In addition, McAllister et al. (1997) noted that when high-sulfur water is the primary reason for increased sulfur intake, the risk of PEM is likely to increase during hot weather when water intakes are higher. Attempts to decrease sulfur-associated PEM by the addition of high levels of dietary copper have been unsuccessful under feedlot conditions, as has thiamine fortification (McAllister et al. 1997). At present, decreasing total sulfate intake (feed and water) is the only means of dealing with the problem.

IMPLICATIONS

Metabolic and nutritional disorders are complex, likely involving nutritional, environmental, genetic, management, and animal behaviour aspects. Ruminal acidity is often the culprit in metabolic disorders. Acidosis occurs with consumption of high levels of rapidly fermented grains, potentially leading to overproduction of VFA and lactate in the rumen. Liver abscesses can result from acid insults that erode the ruminal wall and allow bacteria to reach the liver where abscesses form. Feedlot bloat is most likely to occur with high-grain diets that promote high rates of gas production and the growth of microbes that produce mucopolysaccharide slime, leading to the formation of stable foam. Polioencephalomalacia is most frequently associated with feeding high-grain diets. Although polioencephalomalacia can be caused by a thiamine deficiency, lead toxicity, and salt toxicity, recent research has implicated the production of hydrogen sulfide in the rumen, with subsequent absorption into the blood stream, as a potentially important basis for the disease.

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