# Abnormal Acid Production in the Rumen: Newer Findings From Around the World*

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Active research on the problem of excess or abnormal acid production in the rumen is currently being carried out in many countries: Australia, Canada, Cuba, Denmark, Germany (East and West), India, Kenya, the Netherlands, Norway, Sweden, United Kingdom, and the United States. Their published findings will form the basis of this short review.

*Introduction*

Acute acidosis, founder, laminitis, rumen engorgement, grain overload or D(–) lactic acidosis are the terms used to describe the disease complex in ruminants resulting from the over-consumption of highly fermentable carbohydrate. As described in the review by Dunlop and Hammond in 1965 (18), fermentation patterns in the ruminoreticulum change during the course of the disease, resulting in an accumulation of lactic acid both intraruminally and systemically. Specifically, the D(–) isomer of lactic acid has been implicated as the end-product most responsible for the resulting pathophysiology (19).

Lactic acidosis is truly a management disease. Any number of situations which allow an unadapted animal access to large quantities of grain or other feed carbohydrate predisposes the individual to the condition. Observations on management factors leading to the development of the condition in feedlot situations have been described recently by Elam (20). Other reviews have also been published resulting from symposia on this general subject (47,49,51).

Diet remains the basic factor which determines the pattern of fermentation by microbiota in the rumen (46). Since the mixture of acids produced in the rumen profoundly affects the productivity of cattle (2), manipulation of the rumen fermentation in order to achieve maximum feed utilization has been under intense study (37). These studies have resulted in the establishment of eight factors which are of greatest importance in influencing total acid production in the rumen and the specific acids produced (37): 1) ratio of roughage to concentrate; 2) particle size of roughage; 3) level of feeding; 4) frequency of feeding; 5) extent to which the concentrate was processed; 6) type of dietary carbohydrate (e.g., starch vs. glucose); 7) presence of additives (buffers, lipids); 8) inhibition of methane production.

*Rumen Acids*

Although the above factors are all important in determining the end products of microbial fermentation in the rumen, acetate, propionate and butyrate are nearly always most prominent regardless of the diet fed (14). The dietary factors listed above may change the absolute production rates of these acids but acetate is usually the most abundant, then propionate, followed by butyrate which seldom accumulates to more than 15% of the total (14). Lactic acid does not accumulate in the rumen under normal feeding programs as it is not only absorbed but sufficient microbes exist which utilize it as an energy source following its production (9,44). This is especially true in animals which have been adapted to rations containing high levels of carbohydrate (27).

**As reviewed by Dunlop and Hammond (18), lactic acidosis results from abruptly switching an animal from roughage to grain or other readily fermentable carbohydrate. Under these conditions a balanced microbial population capable of fermenting the substrate to the normal volatile fatty acids is not present in the rumen.**

The sequential microbial change occurring in the rumen of animals suffering from acidosis has been identified (1,10,11). Of most significant importance in these changes is the initial and extremely rapid growth of *Streptococcus bovis,* a predominately L(+) lactic acid producer (13). In hay-fed animals this organism is found at a level of about $10^5$ to $10^7$ per ml (10,25). In animals artificially overloaded with barley, this organism increases during the first four hours with a mean generation time of only 26 minutes (13). Peak numbers are usually reached by 12 hours. Numbers of lactobacilli increase more slowly, with a mean generation time of 60 minutes (13). Isolates of these organisms indicate they produce predominately D(–) lactic acid. The sequential changes occurring in the rumen are summarized in Figure 1.

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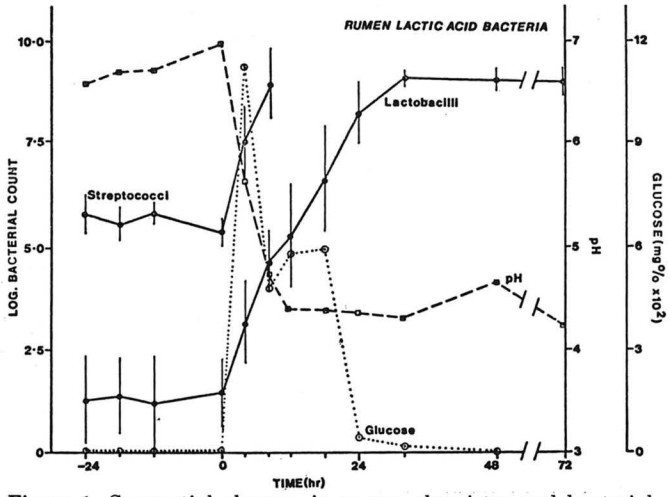


Figure 1. Sequential changes in rumen chemistry and bacterial populations after experimental barley overload in sheep (13).

The identification of the D(–) and L(+) isomers of lactic acid in the rumen of diseased animals has led to a better understanding of the pathophysiology involved (19). The nomenclature of lactic acid has been described by Brin (7) (Table 1). The work of the 1960's indicated that the D(–) isomer, unlike the L(+) isomer, was not initially metabolized or excreted by the bovine kidney and therefore accumulated in body fluids leading to acidosis (18,19). There is now evidence, however, that rat (45) and bovine (40,42) tissues are capable of some metabolism of the D(–) isomer either alone or as part of a racemic mixture (22). Within the rumen, the two isomers may interconvert, especially in the presence of supplementary sodium or potassium (29). Interestingly, blood lactic acid concentrations appear to be related to hereditary "double muscling" (muscular hypertrophy) and to an excitable temperament in affected cattle (23).

Table 1

Nomenclature of Lactic Acids (Brin, 1964)

| Descriptive Name | Rotation of Polarized Light | Old Nomenclature | Current Nomenclature |
|---|---|---|---|
| Natural | dextro- | d– | L(+) |
| Unnatural | levo- | l– | D(–) |
| Racemic | None* | dl– | DL– |

*For an exactly equal mixture of both isomers.

*Gastro-intestinal Motility*

Ruminal atony is a prominent and important clinical feature of lactic acidosis (17,47). As lactic acid fermentation develops, rumen pH falls rapidly (10,17). If it decreases to a pH of 5.0, motility is severely depressed and usually stops if the pH continues to drop (17). Extensive work has been conducted on attempting to define the relative effectiveness of different acids at inhibiting rumen motility. Bruce and Huber (8) have shown the infusion of lactic acid into the duodenum of sheep will produce a stasis of forestomach motility. It is presumed that the intestinal hormone secretin was being released (24) and inhibiting forestomach motility. This may be a protective mechanism in that rumen stasis would reduce absorption of lactic acid and reduce the mixing of substrate with bacteria thereby preventing further acid production (26). Of the VFA's which accumulate in the rumen of normal animals, butyric acid is reported as most effective in reducing gastrointestinal motility as shown by Svendsen (47). The inhibitory effect in this case appears to be directly on the smooth muscle rather than through hormonal or central nervous mechanisms (47). In similar studies, however, infused butyric acid decreased abomasal emptying rate but metabolic acidosis *per se* did not (38). The infusion of lactic acid into the rumen, however, does not diminish ruminal contractions (47), and the situation still remains unclear.

*Natural and Experimental Models*

The clinical episodes of acute or subacute acidosis in feedlot cattle are well recognized by practitioners (5). In general, as noted at the outset, lactic acidosis as induced and then described by Dunlop (19) is assumed to be the underlying abnormality. Recent work, however, seems clearly to show that the specific substrate (i.e., diet) in the rumen actually determines the specific acids which are involved (35). Barley

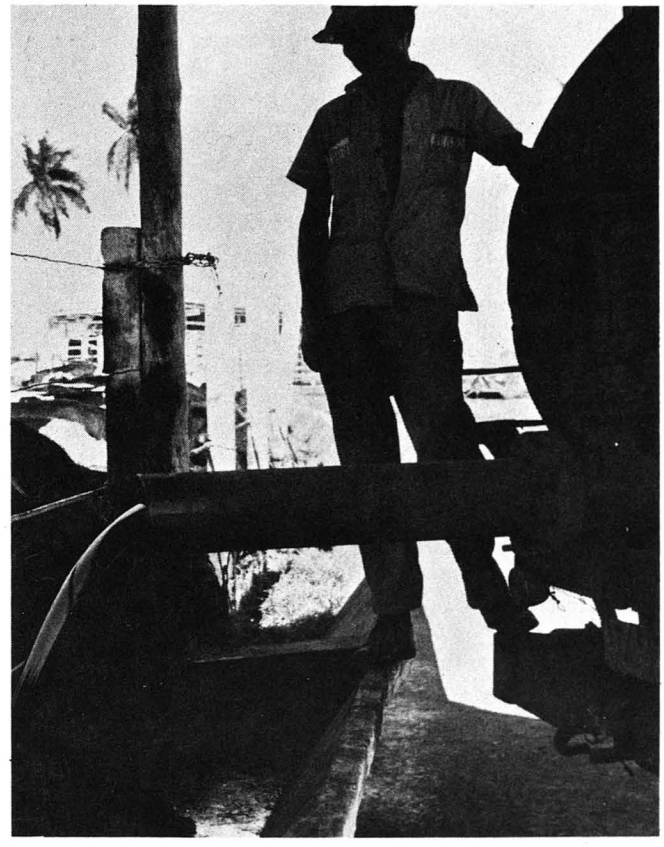

Figure 2. Molasses-urea feedlot in Cuba (30,33).

gives results different from wheat, for example; wheat overfeeding leads to lactic acidemia (accumulation of lactate in blood) but soon, a butyric acidosis within the rumen (35). In other words, the offending acid(s) may differ in rumen and blood. Other evidence using a barley diet indicates that VFA production is completely suppressed for up to 72 hours after overloading (10). Overeating of wheat in Australia ("wheat sickness") (4) would differ then from overeating of barley, corn, or another grain in terms of amount and type of acid in blood and rumen (36).

In Cuba, and now in Kenya (15,33), systems for feeding cattle principally on molasses have been developed based on the innovative research of Preston (41) (Figure 2). Interestingly, the pH remains relatively high and the principal intraruminal VFA is butyric acid (32) in this feeding system.

A condition of free gas bloat rather than the typical foamy bloat has been reported under feedlot conditions (11) which is seldom recognized by research workers. Animals fed high levels of barley have often been observed suffering from bloat. Upon passing a stomach tube the majority of the gas can be removed without interference from foam, verifying the free gas condition. It is possible that the rumen stasis which accompanies this anomaly results from an elevated production of rumen acids (3) and an increased turnover of rumen contents (16) which occurs in animals fed high levels of grain. Under these conditions, inflammation of the esophagus and rumen wall surrounding the cardia could ensue or sufficient levels of acids could pass into the lower GIT to suppress forestomach contractions.

*Thiamine*

Ever since the observation of Broberg (cited in 18) in overloaded cattle that urinary thiamine excretion decreased during and immediately following acidosis, there has been interest in the possible therapeutic use of this vitamin in ruminant acidosis. Indeed, parenteral administration of thiamine to human subjects experimentally made lactic acidemic reduced urinary lactic acid excretion, i.e., presumably hastened its metabolism (50). On the other hand, thiamine injections (2 gm/wether, i.v.) did not increase blood D-lactate clearance in experimental sheep (25). Thiamine is said to be used in treatment in the United Kingdom (34), although its efficacy has not been proved. The role of thiamine in the etiology of bovine polioencephalomalacia (PEM) is well-known (30,31), and further work is required to determine whether lactic acidosis and PEM are related (see below). There is some speculation that they represent only different points along a single continuum (20). Although intraruminal thiaminase activity has been said to develop only in the presence of acidosis (6), this is contrary to the experience in molasses-associated PEM (30,33) or even in North American outbreaks. Furthermore, the pH optimum for thiaminase I, reported to be fairly high (5.8-7.0 or more), needs more definition.

*Therapy*

A treatment of acute acidosis which is entirely successful remains elusive, regardless of the approaches taken. Degrees of success are obtained following siphonage and irrigation of the rumen with large quantities of water to remove the contents as described by Radostits and Magnusson (43). If the contents contain any residual roughage, the effectiveness of this approach is significantly reduced. Rumenotomy is also a logical approach but is as impractical as siphonage when large numbers of animals need immediate treatment. The use of oral antacids (39), which would appear to be a logical approach, is often contraindicated (34) because the severity of acidosis is often difficult to assess with only limited clinical facilities, and therefore an animal may well be in a self-recovery phase at the time of treatment. An additional load of antacids combined with the alkaline products from lactate metabolism and elevated respiration rate may impose a risk of embarrassment leading to alkalosis (13). It appears, however, that oral buffers can be effectively used to prevent the onset of acidosis (49).

Controlled experiments are needed to critically evaluate the therapeutic potential of thiamine (35,48) under field conditions. Practitioners have been evaluating this vitamin in practice without publishing the results.

Sulfonamides and antibiotics have been advocated (34) to inhibit intraruminal acid production, but few definitive results are available. Chaplin and Jones (13) have examined antibiotic sensitivities of *Streptococcus bovis* and lactobacilli cultures taken from acidotic animals and found erythromycin and penicillin G to be most effective at preventing growth. Limited results indicate that further rumen fermentation is only partially retarded if these antibiotics are given orally 12 hours after overloading. If they are given before the lactate-producing organisms are established, they may prove more effective, but this may be impractical in the field.

**An interesting approach for managing acidosis has been the possible exploitation of lactic acid-utilizing bacteria (26,27). Several of these organisms have been isolated (26,28). Through inoculation of unadapted animals with rumen fluid from adapted animals or with selected organisms (26) it is possible to place animals on high grain rations more rapidly. This is, however, limited to preconditioning and presently has not been applied to acidotic animals. Furthermore, this approach is not likely to be successful under acidotic conditions because the rumen environment seems too hostile to support the growth of these more desirable organisms.**

**References**

1. Allison, M. J., Robinson, I. M., Dougherty, R. W., and Bucklin, J. A. (1975): Grain Overload in Cattle and Sheep: Changes in Microbial populations in the Cecum and Rumen. Am. J. Vet. Res. 36:181-185. – 2. Annison, E. F. (1975): Energy Utiliza-

tion in the Body. In: Principles of Cattle Production, H. Swan and W. H. Broster, eds., Butterworths, London (pp. 121-143). - 3. Bauman, D. E., Davis, C. L., and Bucholtz, H. F. (1971): Propionate Production in the Rumen of Cows Fed Either a Control or High-Grain, Low-Fiber Diet. J. Dairy Sci. 54:1282-1287. - 4. Bingham, M. L., and McManus, W. R. (1975): Whole Wheat Grain Feeding of Lambs. IV. Effects of an Antibiotic, Lactic Acid, Grain Moistening and Restricted Intake on Wheat Sickness of Young Sheep. Aust. J. Agric. Res. 26:729-41. - 5. Blood, D. C., and Henderson, J. A. (1974): Veterinary Medicine, 4th ed., Bailliere Tindall, London, (pp. 87-91). - 6. Brent, B. E. (1976): Relationship of Acidosis to Other Feedlot Ailments. J. Animal Sci. 43:930-935. - 7. Brin, M. (1964): Chemistry and Metabolism of D- and L- Lactic Acids. Assoc. of Food & Drug Officials of the U.S. XXVIII (4):178-188. - 8. Bruce, L. A., and Huber, T. L. Inhibitory Effect of Acid in the Intestine on Rumen Motility in Sheep. J. Anim. Sci. 37:164-168. - 9. Bryant, M. P. (1970): Microbiology of the Rumen. In: Dukes Physiology of Domestic Animals, M. J. Swenson, ed., Cornell Univ. Press, Ithaca (pp. 484-515). - 10. Chaplin, R. K., and Jones, G. A. (1973): Rumen Microbial Changes During Experimental Rumen Acidosis. J. Dairy Sci. 56:671 (abst). - 11. Chaplin, R. K., and Jones, G. A. (1973): Microbial Changes in the Rumen of Sheep During the Onset of Induced Grain Overload. Twelfth Ann. Rumen Function Conf., Chicago. - 12. Chaplin, R. K. (1973): Types of Feedlot Bloat and Associated Rumen Characteristics. In: Symposium on Ruminant Bloat. Saskatchewan Division of the Canadian Feed Manufacturers Assoc., Saskatoon, (pp. 11-23). - 13. Chaplin, R. K., and Jones, G. A. (1976): Unpublished Results. - 14. Church, D. C. (1975): Digestive Physiology and Nutrition of Ruminants. Vol. 1. O&B Books, Corvallis, Oregon. - 15. Creek, M. J., Redfern, D. M., Squire, H. A. (1974): Intensive Cattle Feeding in Kenya Using High Levels of Molasses. Wld. Rev. Anim. Prod. 10:60-68. - 16. Davis, C. L. (1967): Acetate Production in the Rumen of Cows Fed Either Control or Low-Fiber, High-Grain Diets. J. Dairy Sci. 50:1621-1625. - 17. Dougherty, R. W., Riley, J. L., and Cook, H. M. (1975): Changes in Motility and pH in the Digestive Tract of Experimentally Overfed Sheep. Am. J. Vet. Res. 36:827-829. - 18. Dunlop, R. H., and Hammond, P. B. (1965): D-Lactic Acidosis of Ruminants. N.Y. Acad. of Sci. 119(3):1109-1132. - 19. Dunlop, R. H. (1972): Pathogenesis of Ruminant Lactic Acidosis. Adv. Vet. Sci. and Comp. Med. 16:259-302. - 20. Elam, C. J. (1976): Acidosis in Feedlot Cattle: Practical Observations. J. Anim. Sci. 43:898-901. - 21. Flachowsky, G., Lohnert, H. J., and Hennig, A. (1974): Einige Aspekte zur vitamin $B_1$ versorgung von lammern nach provozierter pansenazidose. Arch. exper. Vet. med. 28:543-549. - 22. Giesecke, D., Bartelmus, C., and Stangassinger, M. (1976): Undersuchungen zur genese und biochemie der pansenacidose Zbl. Vet. Med. A. 23:353-363. - 23. Holmes, J. H. G., Robinson, D. W., and Ashmore, C. R. (1972): Blood Lactic Acid and Behaviour in Cattle with Hereditary Muscular Hypertrophy. J. Anim. Sci. 35:1011-1013. - 24. Horn, G. W., and Huber, T. L. (1975): Duodenal Acidification: Stimulus for the Release of Intestinal Hormones in Sheep. J. Anim. Sci. 41:1199-1205. - 25. Huber, T. L. (1969): Effect of Ration on Liver Oxidation of Racemic Lactate and of Thiamine on Blood Lactate Clearance. J. Anim. Sci. 28(1):98-102. - 26. Huber, T. L. (1976): Physiology of Systemic Lactic Acidosis. In: Buffers in Ruminant Physiology and Metabolism. Weinberg, M. S., and Sheffner, A. L., eds., Church and Dwight Co., Inc., publishers, (pp. 96-106). - 27. Huber, T. L., Cooley, J. H., Goetsch, D. D., and Das, N. K. (1976): Lactic Acid-Utilizing Bacteria in Ruminal Fluid of a Steer Adapted from Hay Feeding to a High-Grain Ration. Am. J. Vet. Res. 37:611-613. - 28. Hungate, R. E. (1966): The Rumen and its Microbes. Academic Press, New York. - 29. Lee, D., Jr., and Matrone, G. (1971): Influences of Monovalent Cations on Growth and Lactic Acid Metabolism of Sheep Fed Purified Diets. J. Nutri. 101:967-974. - 30. Loew, F. M. (1975): A Thiamine-Responsive Polioencephalomalacia in Tropical and Nontropical Livestock Production Systems. World Rev. Nutr. & Dietetics. 20:168-183. - 31. Loew, F. M. (1975): Nutrition and Bovine Neurologic Disease. J.A.V.M.A. 166:219-221. - 32. Marty, R. J., and Preston, T. R. (1970): Molar Proportions of the Short Chain Volatile Fatty Acids Produced in the Rumen of Cattle Given High Molasses Diets. Rev. Cub. Cienc. Agric. (Eng. ed.) 4:183-186. - 33. Mella, C. M., Perez-Oliva, O., and Loew, F. M. (1976): Induction of Bovine Polioencephalomalacia with a Feeding System Based on Molasses and Urea. Can. J. Comp. Med. 40:104-110. - 34. Mullen, P. A. (1976): Overfeeding in Cattle–Clinical, Biochemical and Therapeutic Aspects. Vet. Rec. 98:439-443. - 35. Mullen, P. A., and Penny, R. H. C. (1976): Concentrate Overfeed in Cattle: Biochemical and Therapeutic Observations. Vet. Rec. In press. - 36. Munchow, von H., Edvi, P., and Rossow, N. (1975): Verhalten des pH-Wertes und der fluchtigen Fettsauren im Pansensaft bei der latenten alimentaren metabolischen Azidose des Schafes. Mh. Vet. Med. 31:91-95. - 37. Orskov, E. R. (1975): Manipulation of Rumen Fermentation for Maximum Food Utilization. World Rev. Nutr. Diet. 22:152-182. - 38. Poulsen, J. S. D., and Jones, B. E. V. (1974): The Influence of Metabolic Alkalosis and Other Factors on the Abomasal Emptying rate in goats and Cows. Nord. Vet. Med. 26:22-30. - 39. Prasad, J., and Rekib, A. (1975): Clinical Management of Rumen Acidosis with Sodium Bicarbonate and Rumen Cud Transplant. Indian Vet. J. 52:317-319. - 40. Preston, A. M., and Noller, C. H. (1973): Metabolism of D-Lactate by Tissues of the Ruminant Digestive Tract. J. Anim. Sci. 37:1403-1407. - 41. Preston, T. R. (1972): Fattening Beef Cattle on Molasses in the Tropics. World Anim. Rev. (FAO). 1:24-29. - 42. Prins, R. A., van Vugt, F., Lankhorst, A., and Akkermans-Kruyswijk, J. (1974): Metabolism of Lactate Isomers by Rumen Mucosa. J. Dairy Sci. 57:556-561. - 43. Radostits, O. M., and Magnusson, R. A. (1971): A Modification of an Old Method for Emptying the Rumen of Cattle. Can. Vet. J. 12:150-151. - 44. Schwartz, H. M., and Gilchrist, F. M. C. (1975): Microbial Interactions with the Diet and the Host Animal. In: IV Int. Symp. on Ruminant Physiology, Sydney (pp. 165-179). - 45. Shichiri, M., Miller, M., and Woodward, H., Jr. (1967): Disappearance of DL-Lactate-2-$C^{14}$ From Blood in Normal and Diabetic Rats. Proceedings of the Soc. for Exp. Biol. & Med. 125:1238-1242. - 46. Sutton, J. D. (1975): Energy Supply from the Digestive Tract of Cattle. In: Principles of Cattle Production, H. Swan and W. H. Broster, eds., Butterworths, London. (pp. 121-143). - 47. Svendsen, P. E. (1975): Experimental Studies of Gastrointestinal Atony in Ruminants. In: IV Int. Symp. on Ruminant Physiology, Sydney (pp. 563-575). - 48. Vestweber, J. G. E., and Leipold, H. W. (1974): Experimentally Induced Ovine Ruminal Acidosis: Pathologic Changes. Am. J. Vet. Res. 35:1537-1540. - 49. Weinberg, M. S., and Sheffner, A. L., eds. (1975): Buffers in Ruminant Physiology and Metabolism. Symp., Univ. of Arizona. - 50. Wendel, O. W. (1960): A Study of Urinary Lactic Acid Levels in Humans. 1. Influence of Thiamine and Pyrithiamine. J. Vitaminology. 6:16-23. - 51. Wheat in Livestock and Poultry Feeds. (1970): Proceedings of an International Symposium, Okla. State Univ., Stillwater, Okla.

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