# General Session I

"Feeding the Rumen: That Vat That Pays Your Bills"

Moderator: **Paul Johnson**



# Microbiology and Physiology of the Rumen and Its Response to Different Feeding Strategies

**Jane A. Z. Leedle, Ph D**
*Department of Anatomy & Physiology,*
*College of Veterinary Medicine*
*Kansas State University, Manhattan KS 66502-5602*

## Introduction

The rumen is inhabited by a complex and highly competitive microbial population composed predominantly of strictly anaerobic bacteria ($10^{10}$ to $10^{11}$/g ruminal contents) and protozoa ($10^5$ to $10^7$/g) with smaller numbers ( $\leq 10^4$/g) of anaerobic fungi and facultatively anaerobic bacteria. These microorganisms are responsible for the degradation of ingested feedstuffs to fermentation acids (primarily the volatile fatty acids: acetate, propionate and butyrate) which are absorbed by the host through the ruminal epithelium and serve as the dominant carbon and energy sources for growth, maintenance and production. The ruminant animal then is a symbiotic association between mammal and microorganisms which evolved to enable the animal to live on high fiber diets (19). The ruminal regulatory systems developed by the animal were intended to cope with the microbial fermentation of forage-based rations and they operate very effectively for this purpose. These regulatory systems include temperature control, ruminal pH control through the buffering action of saliva, provision of extra nutrients such as urea or phosphate which enters the rumen also via the saliva, removal of inhibitory soluble (end) products through absorption, and removal of indigestible solids through passage to the lower tract (28). The animal can regulate the activities of the ruminal microorganisms only in so far as it can vary these processes. For the rest, the microorganisms are controlled only by the limitations of their growth physiology and by synergism and competition between species.

With the practice of high grain feeding, the ruminal fermentation has become a liability to the host animal. When diets high in readily fermentable carbohydrates, i.e., diets high in starchy cereal grains, are fed to ruminants, caloric intake and substrate availability are no longer controlled by physical constraints such as fiber particle size and ruminal volume. The animal can now ingest more calories in a shorter time because of the smaller sized, energy-dense nature of cereal grains compared to cellulosic materials. As a result, members of the microbial community are released from substrate limitation restraints. For example, the activities of the amylolytic (starch-degrading) and the saccharolytic (sugar-degrading) bacteria are no longer limited by the relatively slow degradation of fibrous polymers to fermentable oligomers and thus starch degradation and fermentation can occur at very rapid rates.

## Acidosis and Predisposition to Illness

Rapid and (or) extensive fermentation of feed starches by the rumen microbial community causes ruminal instability which may lead to acidosis. Acidosis is initiated by ruminal bacteria fermenting the dietary starches and producing large amounts of organic acids, including lactic acid, to which the cattle are unaccustomed. Under these conditions, the quantity of metabolic hydrogen produced by fermentative bacteria exceeds that which the methanogenic bacteria and other members of the population can remove, and hydrogen increases within the rumen (29). Feedstuff degradation continues however, with the microbes diverting the metabolic hydrogen from volatile fatty acid to lactic acid production (29). This occurs at the expense of microbial protein synthesis and costs energy to the bacteria, even those producing lactic acid (31). Increased acid loading and redirection of the anaerobic fermentation toward lactic acid can lead to subsequent lowering of the ruminal pH as lactic acid is 10 fold stronger than volatile acids. The ruminal acid load can be so great that the natural balances between ruminal acid production and consumption by the microflora, as well as ruminal ab-

sorption and buffering capacity by the animal, are disrupted (17, 32), and acidosis ensues.

Development of acidosis in the beef animal or dairy cow can be detrimental. If ruminal balance is not restored, the acidosis may cause and (or) predispose the animal to a variety of maladies including "off-feed" syndrome (cyclic pattern of feed intake), founder, rumenitis, malabsorption and liver abscesses (4, 5, 11, 32). It may also be a major metabolic factor in predisposing cattle to BRD although the relationship of ruminal instability to etiologic disease is unclear. Successful feeding adaptation during step-up programs means maintenance of ruminal balance and avoidance of acidosis. Ruminal balance helps regulate feed consumption in beef and dairy cattle, and cattle that eat consistently appear to have fewer health problems.

## Ruminal Stability

Ruminal stability plays a larger role than we realize regarding maintainance of animal health since the normal flora can ward off undesirable, opportunistic bacteria and (or) metabolic processes. And despite attempts to the contrary, we feed the microbes first and the animal second. Thus, understanding the altered microbial activities under conditions of high grain feeding may help reduce animal morbidity, avoid alimentary ailments such as acidosis, and reduce digestive losses.

Our knowledge of ruminal microbiology and physiology of ruminants fed high grain diets is extensive (1, 10, 14, 22, 25), but far from complete. We believe, however, that a smooth transition from forage to grain diets can be made with the successful interaction of 4 groups of ruminal bacteria. Three of the 4 groups ferment starches and sugars with the production of either normal volatile fatty acids (VFAs) or lactic acid. The fourth group degrades lactic acid. One of the starch fermenting groups produces only VFAs. Its members are gram-negative and typified by *Ruminobacter amylophilus.* The second starch degrading group produces either VFAs or lactic acid depending on the environmental conditions. This group is typified by *Selenomonas ruminantium* (a gram-negative bacterium) and *Streptococcus bovis* (a gram-positive bacterium). The third starch group produces only lactic acid. It is composed of gram-positive *Lactobacillus* sp. Members of the lactic acid consuming group are all gram-negative, namely, *Selenomonas ruminantium* ssp. *lactilytica, Megasphaera elsdenii,* and *Veillonella alkalescens.*

During adaptation to grain based diets, these groups interact in synchrony to enable starch fermentation to proceed with normal acid production. However, if feed is overconsumed or illness prevents consistent intake, the faster growing, gram-positive members of the starch fermenting groups predominate (1, 2, 14). The streptococci shift from acetic to lactic acid production as acid accumulates and ruminal pH decreases. With the shift to lactic acid synthesis, the streptococci become less efficient with respect to production of microbial protein (energy cost is 2 ATP per mole sugar fermented), and since they are sensitive to the acid they produce, they yield to the more acid tolerant lactobacilli which produce only lactic acid. Ruminal dominance by the lactobacilli group is associated with low ruminal pH ($\leq 5.5$) and spells potential disaster for most of the remaining flora. The fiber-degrading, cellulolytic bacteria and the protozoa decrease dramatically at ruminal pH values less than 6.0 (8, 9, 30). Even *Megasphaera elsdenii,* a bacterium able to ferment DL-lactic acid below pH 6.0 (7) ceases its activity below pH 5.2. With the cessation of microbial lactic acid consumption, lactic acid continues to increase and severe damage to the host animal may result. If, on the other hand, the starch degrading groups are allowed to adapt to the type, quantity and frequency of dietary starch input, the lactic acid consuming group can keep up with microbial production (17, 18, 23, 24).

## Components of High Starch Diets

As practitioners, our goal is to maintain ruminal stability to maintain animal health. The challenge is that we are feeding cattle readily degradable materials which can be fermented by nearly every type of microorganism present in the intestine. So, how can we give the normal ruminal bacteria an edge to remain active and dominant? An examination of feedstuff components in high grain diets from the microbial point of view will reveal that feeding consistency gives the normal flora that edge.

Dominant in forage-based rations are components of the plant cell wall matrix: cellulose, hemicellulose (xylan) and associated lignin complexes. Dominant in grain-based rations are components of the infrastructure of plant cells: starches, sugars, pectins, galactans, *B*-glucans, etc. (33). In balancing ruminant rations, we pay attention to net energy, soluble carbohydrate, effective fiber, buffering capacity, and metabolizable protein content (36). But it is the soluble carbohydrate element, i.e., the non-fibrous carbohydrate, that has the major impact on beef animal performance, on ruminal efficiency, and successful microbial adaptation.

## Starch Quality

Starches vary widely in quality and quality is based on their physicochemical structure. The basic types of starch present in grain diets are amylose and amylopectin. Amylose is composed of linear chains of glucose moieties linked together in the -1,4 position. These linear chains are responsible for the starch's crystallinity characteristic (12). Crystallinity is disrupted by cooking, such as in the flaking process, which allows hydration (gelatinization) of the starch and increases its ease of digestion. Upon cooling

however, the amylose chains can realign, resuming the crystalline structure and diminishing the digestibility increment gained in flaking (34).

Amylopectin starch is composed of linear amylose chains containing branch points linked in the -1, 6 position. Due to the branching, amylopectin chains are unable to realign, thus remain digestible regardless of processing (34).

Grains contain a mixture of amylose and amylopectin starches, but more characteristic of a specific grain is its organization of component starches into granules. When amylose and amylopectin are synthesized by the plant cell, they are laid down in a relatively anhydrous matrix usually in association with protein(s). This structure gives the starch granule its insoluble nature. The term "insoluble starch" denotes starches that require enzymatic or fermentative action before becoming "soluble" (35). The grain's starch presents the ruminal bacteria with the challenge to fabricate the enzymatic machinery necessary to degrade its physicochemical structure into component sugars for cellular anabolism.

## Bacterial Degradative Strategies

Among the major bacteria that are in high population levels in the rumen that can utilize high molecular weight forms of starch (i.e., amylopectin and amylose) are *Bacteroides ruminicola, Butyrivibrio fibrisolvens, Ruminobacter amylophilus* and *Streptococcus bovis.* Each of these has its own strategy for degrading its preferred substrate(s) which includes extracellular and intracellular enzyme systems as well as attachment structures, transport proteins, etc. Enzyme systems are synthesized by the bacteria according to the specific forms of starch substrates present in the feed. Each degradative strategy reflects an investment of energy by the microorganism. A large cost is incurred with each dietary change, particularly with different grains, type of processing or relative abundance. With each dietary change, ever-present competitive processes open the door to risk of ruminal takeover by opportunistic bacteria resulting in abnormal fermentation products or runaway fermentation rates.

Much information on the mechanisms by which different ruminal bacteria hydrolyze starches and how they regulate production of their enzyme systems is lacking. However, studies to date suggest that the rumen amylolytic bacteria differ widely in the types of starch (maltodextrins versus high molecular weight starch) they can hydrolyze (16, 20, 21), the subcellular location and activity levels of their amylases (6, 26, 37), and the nature of the regulation of these enzymatic systems(6,15). This variety in bacterial starch degrading enzyme systems results in different starch digestion rates by the different amylolytic bacteria in the rumen.

## Ruminal Stability = Consistency

Ruminal stability is an orchestration of the different enzymatic systems degrading the diversity of starches present in well balanced diets. However, we quickly get at odds with starch fermentations when feeding consistency is dictated by other factors. Switching grain sources, dry to wet feedstuffs, or the type of processing based on availability or economics courts ruminal disaster since many bacterial enzyme systems are subject to regulation. As an example, the microbial degradation of insoluble starches is subject to repression by low molecular weight sugars (mono-, di-and oligo-saccharides). This means that if whole or rolled corn or rolled milo is being fed, the addition of high moisture corn or flaked grain can upset the ruminal balance by introducing a source of rapidly fermentable short chain sugars. Bacteria preferring to grow on the sugars may then predominate at the expense of those which have "invested" in the fermentation of the whole or rolled grain. If a sizable quantity of high moisture corn (or other sugar source) is fed, the degradation of the insoluble portion of the diet may be significantly hindered. Ruminal instability may result.

With all the variability inherent in the bacterial strategies for starch degradation, can a balance be struck? A balance may be achieved by keeping dietary composition and the pattern of feed consumption as consistent as possible. As previously mentioned, the most important facet to bacteria is consistency in starch composition. This aspect has more impact on adaptation (as well as on lactation or finishing) than previously appreciated. Since changing the type of starch or its processing requires the bacteria to change their strategic hardware, large, sudden, or haphazard shifts in dietary composition should be avoided. The cost to the microbe is in terms of ATP and the gear-up time during which another bacterium may outcompete the first for the new substrate. The cost to the host animal is ruminal instability and its possible secondary effects. The key to a successful step-up program is to increment dietary energy with consistency in ration ingredients, particularly with respect to their starch (grain) type and processing method.

## Effects of Veterinary Pharmaceuticals on Normal Ruminal Bacteria

Along with consistency in starch components, avoidance of other potentially damaging effects to the viability of the normal ruminal bacteria also is key to maintenance of stability and health. Antagonistic effects may be observed with therapeutic antibiotics used to treat cattle for a variety of illnesses. The normal ruminal microflora must exist unimpeded to preserve its natural barrier to certain pathogens and (or) aberrant metabolism. Most of the ru-

minal microflora are sensitive to commonly used veterinary pharmaceuticals often at concentrations ≤10 ppm. These include tetracycline, chlortetracycline, oxytetracycline, penicillin, tylosin, bacitracin, chloramphenicol and erythromycin (13, 27, 38). Neomycin and streptomycin appear to have little to no effect on the vitality of the ruminal bacteria (13, 27, 38).

Sulfa drugs, such as sulfamethazine and sulfathiazole, often are administered directly into the ruminal compartment. Sulfa-based antibiotics are bacteriostatic in action (3). They interfere with those bacteria actively metabolizing which either require preformed folic acid or must synthesize it *de novo*. Many of the ruminal bacteria fall into this category. If, however, the host animal has been ill or off-feed for more than 24 hours, many of the ruminal bacteria may not be actively metabolizing (J.A.Z. Leedle and R.B. Hespell, unpublished observations). In this case, use of sulfa boluses is counterindicated because the goal is to revitalize the bacteria, not to compromise them. Thus, indiscriminate ruminal sulfa administration should be limited especially in starter cattle and freshening cows. In general, avoiding orally administered products and (or) prolonged treatment regimens is best to maintain the health and vigor of the rumen microbial flora.

## Summary

In summary, the rumen microbial population is complex and highly competitive. With the practice of high grain feeding to cattle, the rumen compartment becomes a liability to animal health due to the ease of starch fermentation and the potential for excessive acid production compared to more fibrous feedstuffs. This is because the bacteria lack the regulatory mechanisms which might prevent overgrowth by a favored species. Maintaining ruminal stability in a starch-based dietary program requires taking advantage of the variety of enzymatic degradative mechanisms by which the bacteria ferment the different physicochemical forms of starch. Each degradative strategy represents an investment by the bacterial species involved. The resultant interplay of starch fermenting and lactic acid consuming bacterial groups is key to stability and animal health. Each change in starch grain, processing method, or abundance upsets the preexisting balance among the bacteria. Feeding programs dictated by economic or other factors court ruminal instability. Management programs geared toward feeding balanced starch materials throughout step up or adaptation periods minimize animal health problems. Reducing antagonistic antibiotic therapies, especially those affecting the ruminal compartment directly, will retain the vigor of the rumen microbial population and encourage consistent feed consumption by the host. Dairy and feedyard operators incorporating these points into their feeding programs will have the advantage of the normal ruminal microflora working with them to achieve maximum benefit.

## References

1. Allison, M.J., R.W. Dougherty, J.A. Bucklin and E.E. Snyder. 1964. Ethanol accumulation in the rumen after overeating with readily fermentable carbohydrate. Science *144*:54-55. 2. Allison, M.J., I.M. Robinson, R.W. Dougherty and J.A. Bucklin. 1975. Grain overload in cattle and sheep: changes in microbial populations in the cecum and rumen. Amer. J. Vet. Res. *36*:181-185. 3. Bevill, R.F. 1988. Sulfonamides. pp. 785-795. IN: N.H. Booth and L.E. McDonald (eds). *Veterinary Pharmacology and Therapeutics.* Sixth edition. Iowa State University Press. Ames, IA. 4. Brent, B.E. 1976 Relationship of acidosis to other feedlot ailments. J. Anim. Sci. *43*:930. 5.Britton, R.A. and R.A. Stock. 1986. Acidosis, rate of starch digestion and intake. pp 125-136. IN: *Proceedings of the 1986 Feed Intake by Beef Cattle Symposium.* Oklahoma Agric. Expt. Stn., Oklahoma State University Publication. 6. Cotta, M.A. 1988. Amylolytic activity of selected species of ruminal bacteria. Appl. Environ. Microbiol. *54*:772-776. 7. Counotte, G.H.M. and R.A. Prins. 1979. Regulation of rumen lactate metabolism and the role of lactic acid in nutritional disorders of ruminants. Vet. Sci. Comm. *2*:277-303. 8. Dirksen, G. 1970. Acidosis. pp. 612-625. IN: A.T. Phillipson (ed.), *Digestion and Metabolism in the Ruminant.*Oriel Press, Newcastle, England. 9. Dunlop, R.H. 1971. Pathogenesis of ruminant lactic acidosis. Adv. Vet. Sci. Comp. Med. *16*:259-302. 10. Dunlop, R.H. and P.B. Hammond. 1965. D-Lactic acidosis of ruminants. Annals New York Acad. Sci. *119*:1109-1132. 11. Elam, C.J. 1976. Acidosis in feedlot cattle: practical observations. J. Anim. Sci. *43*:898-901. 12. French, D. 1973. Influence of processing on the utilization of grains (starch, by ruminants. J. Anim. Sci. *37*:1075. 13. Fulghum, R.S., B.B. Baldwin, P.P. Williams. 1968. Antibiotic susceptibility of anaerobic ruminal bacteria. Appl. Microbiol. *16*:301-307. 14. Grubb, J.A. and B.A Dehority. 1975. Effects of an abrupt change in ration from all roughage to high concentrate upon rumen microbial numbers in sheep. Appl. Microbiol. *30*:404-412. 15. Hobson, P.N. and M. MacPherson. 1952. Amylases of *Clostridium butyricum* and a *Streptococcus* isolated from the rumen of the sheep. Biochem. *52*:671-679. 16. Holdeman, L.V., R.W. Kelley and W.E.C. Moore. 1984. Anaerobic gram-negative straight, curved and helical rods. pp. 602-662. IN: N.R. Krieg and J.G. Holt (eds).*Bergey's Manual of Systematic Bacteriology.* Volume 1. Williams and Wilkins. Baltimore, MD. 17. Huber, T.L. 1976. Physiological effects of acidosis on feedlot cattle. J. Anim. Sci. *43*:902-909. 18. Huber,T.L., J.H. Cooley, D.D. Goetsch and N.K. Das. 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. 19. Hungate, R.E., M.P. Bryant and R.A. Mah. 1964. The rumen bacteria and protozoa. Annu. Rev. Microbiol. *18*:131-166. 20. Jones, D. and M.D. Collins. 1986. Irregular, nonsporing gram-positive rods. pp. 1261-1434. IN: P.H.A. Sneath, N.S. Mair, M.E. Sharpe and J.G. Holt (eds). *Bergey's Manual of Systematic Bacteriology.* Volume 2. Williams and Wilkins. Baltimore, MD. 21. Kandler, O. and N. Weiss. 1986. Regular, nonsporing gram-positive rods. pp. 1208-1261. IN: P.H.A. Sneath, N.S. Mair, M.E. Sharpe and J.G. Holt (eds). *Bergey's Manual of Systematic Bacteriology.* Volume 2. Williams and Wilkins. Baltimore, MD. 22. Krogh, N. 1961. Studies on the alterations in the rumen fluid of sheep, especially concerning the microbial composition when readily available carbohydrates are added to the food. IV. Identification of the gram-positive flora developing during the feeding experiments. Acta Vet. Scand. *2*:357-374. 23. Mackie, R.I., F.M.C. Gilchrist and S. Heath. 1984. An *in vivo* study of ruminal micro-organisms influencing lactate turnover and its contribution to volatile fatty acid production. J. Agric. Sci., Camb. *103*:37-51. 24. Mackie, R.I., F.M.C. Gilchrist, A.M. Robberts, P.E. Hannah and H.M. Schwartz. 1978. Microbiological and chemical changes in the rumen during the stepwise adaptation of sheep to high concentrate diets. J. Agric. Sci. *90*:241-254. 25. Mann, S.O. 1970. Some effects on the rumen microorganisms of overfeeding a high barley ration. J. Appl. Bacteriol. *33*:403-409. 26. McWethy, S.J. and P.A. Hartman. 1977. Purification and some properties of an extracellular alpha-amylase from *Bacteroides amylophilus.* J. Bacteriol. *29*:1537-1544. 27. Nagaraja, T.G. and M.B. Taylor. 1987. Susceptibility and resistance of ruminal bacteria to antimicrobial feed additives. Appl. Environ. Micro-

biol. *53*:1620-1625. 28. Phillipson, A.T. 1977. Ruminant Digestion. pp.250-286. IN: *Duke's Physiology of Domestic Animals.* Ninth edition. Cornell University Press. Ithaca, New York. 29. Schwartz, H.M. and F.M.C. Gilchrist. 1975. Microbial interactions with the diet and the host animal. pp. 165-179. IN: I.W. McDonald and A.C.I. Warner (eds.), *Digestion and Metabolism in the Ruminant.* The University of New England Publishing Unit, Armidale, Australia. 30. Slyter, L.L. 1976. Influence of acidosis on rumen function. J. Anim. Sci. *43*:910-929. 31. Strobel, H.J. and J.B. Russell. 1986. Effect of pH and energy spilling on bacterial protein synthesis by carbohydrate limited culture of mixed rumen bacteria. J. Dairy Sci. *69*:2941-2947. 32. Uhart, B.A. and F.D. Carroll 1967. Acidosis in beef steers. J. Anim. Sci. *26*:1195-1198. 33. Van Soest, P.J. 1982. Chapters 7 and 10 IN: *Nutritional ecology of the ruminant.* O & B Books, Inc. Corvallis, OR. 34. Van Soest, P.J. 1987. Soluble carbohydrates and the non-fiber components of feeds. pp. 44-50. IN: *Large Animal Veterinarian.* September/October 1987. 35. Van Soest, P.J. 1987. Clearing confusion: soluble vs. available carbohydrates. p. 49. IN:*Large Animal Veterinarian.* September/October 1987. 36. Van Soest, P.J. and D.R. Mertens. 1984. The use of NDF vs. ADF in balancing dairy rations p. 75. IN: *Monsanto Technical Symposium.* Fresno, CA. Nutr. Chem. Div., Monsanto. St. Louis, MO. 37. Walker, J.G. 1965. The cell-bound *a*-amylases of *Streptococcus bovis.* Biochem J. *94*:289-298. 38. Wang, C.-L., B.B. Baldwin, R.S. Fulghum and P.P. Williams. 1969. Quantitative antibiotic sensitivities of ruminal bacteria. Appl. Microbiol. *18*:677-679.



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AGRICULTURAL POLICY REFORM
Politics and Process in the EC and USA
*H. Wayne Moyer, Timothy E. Josling*

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*Agricultural Policy Reform* begins by developing an analytical framework for the assessment of agricultural policy decision making. Next, a detailed examination of the agricultural policy process in the USA and the EC follows with a discussion of the "reforms" of the 1980's. The decision-making processes are compared for the 1981 and 1985 US farm bills, the milk quotas decision of 1984, and the agricultural stabilizers agreement of 1988 to show the applicability of the analytical framework to specific policy situations. There is discussion on the pressures to reform the international system for agricultural trade. The final chapter is devoted to comparing and contrasting the EC and US experiences and looks for lessons for those charged with reforming farm policies in industrial countries.

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**About The Authors:** H. Wayne Moyer is Professor of Political Science at Grinnell College, Grinnell, Iowa. He is Rosenfield Professor and Director of the Rosenfield Program in Public Affairs, International Relations and Human Rights. He received his PhD from Yale University, and has research interest in foreign policy decision making and agricultural policy. Timothy E. Josling is Professor at the Food Research Institute, Stanford California. He holds a PhD from Michigan State University, and has research interests in agricultural policy and trade. He has written widely on the Common Agricultural Policy and on trade problems in industrial countries.

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