Maintaining a Healthy Rumen – An Overview

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Take Home Message

- The rumen microbial population gets first access to any feed eaten by the cow. Therefore, most of the cow's absorbed nutrients are the result of microbial fermentation or modification, not what the cow actually eats.
- The rumen environment of high producing dairy cows is considerably different from the environment under which most rumen microbes and the host animal evolved. Therefore, our assumption of what is a "healthy" rumen may not even exist for some high producing cows.
- Despite our best efforts, current feeding practices may result in sub-optimal rumen conditions.

Introduction

Cattle and other ruminants are markedly different from monogastric animals, such as man, in that they have an additional organ, the rumen, at the beginning of the gastrointestinal tract. This organ allows ruminants to extract and absorb energy from fibrous plant material not otherwise available to mammalian enzymes. Additionally, the microbial protein, produced as a byproduct of this digestion, provides the host with much of its protein needs.

The modern dairy cow is truly a marvel for its capacity to digest feedstuffs that are poorly digested or indigestible by monogastric animals and convert them into milk, one of nature's best sources of food. Central in this process is the rumen and the resident microbial population. When the rumen becomes dysfunctional, feed digestion is impaired and cows become susceptible to a range of metabolic diseases. In order that cows can achieve their genetic potential for milk production and remain healthy, it is critical that the rumen environment be kept in a "healthy" state.

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It may be that the very genetic potential for milk production is the worst enemy of a healthy rumen. At lower levels of milk production (up to about 25 kg/d), cows can meet their nutrient needs for energy and protein from the fermentation of forages and the subsequent production of microbial protein within the rumen. Above this level of milk production, cows must rely, at least in part, on the supply of additional nutrients to the small intestine (i.e. UIP or bypass protein). In many cases our method of solving nutrient imbalances for lactating dairy cows has become an "everything but the kitchen sink" approach. In this approach, whenever a nutrient deficiency is encountered, a new feed or ingredient is added to the ration to overcome the deficiency. Eventually we have a diet that contains "everything but the kitchen sink". What we fail to recognize, when we do this, is that there are myriad of interactions that occur among nutrients and many of these occur within the rumen environment. The new ingredient may actually counteract other components of the diet and create another nutritional problem that needs to be "cured" by adding another ingredient.

The rumen microbial population has the first opportunity to digest any feed consumed by the cow and anything that affects the rumen ecosystem will ultimately affect what and how nutrients are available to the cow for productive purposes. Nutritionists need to take a step back and examine the diet as a whole and understand its impact on the rumen environment.

The Rumen Environment

The rumen is essentially a fermentation chamber in which the resident microbial population helps to digest the diet. The partially fermented food and the micro-organisms then pass out of the rumen, into the small intestine. Digestion of food in the rumen occurs by a combination of microbial fermentation and physical breakdown during regurgitation of the food by rumination. Microbial attack is carried out by a mixed population of bacteria, ciliate protozoa and a small number of anaerobic fungi. The products of microbial fermentation, mainly volatile fatty acids and microbial protein, are available for absorption by the host cow. Volatile fatty acids can supply up to 80% of the animal's energy requirement, while microbial protein leaving the rumen can account for between 50 and 90% of the protein entering the small intestine. In view of the importance of the rumen in the nutrition of the host, it is perhaps not surprising that a great deal of effort has been devoted to investigating methods for manipulating this complex ecosystem.

The rumen microbial population is very dense, containing 10^{10} bacteria/ml, 10^6 protozoa/ml, and 10^3 fungi/ml. Because protozoa are so much larger than bacteria, the protozoa can make up close to half of the total microbial biomass. These microbes are very specialized to survive and thrive within the rumen.

Conditions are strictly anaerobic. In fact, the presence of oxygen is highly toxic to most rumen microbes. The rumen is buffered over a range of 5.7 to 7.3 by phosphate and bicarbonate from saliva and bicarbonate from rumen fermentation. Temperature is tightly controlled in a range of 36 to 41°C. Rumen microbes are well adapted to these conditions and their specific growth requirements reflect the availability and types of nutrients present in the feed.

The rumen microbial population exists in a highly dynamic state. The total population can change dramatically with any number of factors such as feeding frequency and type of diet. When animals were fed once daily, Leedle et al. (1982) found bacterial numbers to be lowest 2 to 4 hour after feeding and to gradually increase until 16 hours post-feeding. Warner (1966) found very little fluctuation in the numbers of bacteria in the rumen when sheep were fed every three hours. Much of the shifts observed in rumen microbial numbers can be attributed to a dilution effect due to feed and liquid associated with the meal. Also, many of the bacteria in the rumen are free-floating in the liquid just before feeding and become attached to new feed particles after feeding. It is very difficult to remove and count the bacteria attached to feed particles and thus may explain the low numbers observed in the rumen after feeding, when fermentation rate is generally at its greatest.

Bacterial numbers are generally assumed to be higher on high concentrate diets compared to high forage diets (Hespell et al., 1997). However, there are more fluid-associated bacteria with high concentrate diets and thus, easier to enumerate. The biggest differences due to diet are in the type of bacteria rather than the total number.

Unlike the bacterial population, ruminants can live successfully without the presence of rumen protozoa. Rumen protozoa can be divided into two general groups: the "holotrichs" and "entodiniomorphs". Until recently, it has been very difficult to study the different groups in isolation from the other so most research refers to faunated animals (with protozoa) and defaunated animals (without protozoa).

In general, the major changes that can be attributed to protozoa consist of an increase in bacterial numbers, a slight decrease in rumen digestibility and little or no difference in animal growth. Total tract digestibility remains the same, presumably because of compensatory digestion in the hindgut.

The holotrich protozoa are very important in utilizing soluble sugars. Because their growth rate is slower and their size larger than bacteria, the holotrichs help to control the rate of carbohydrate fermentation when large quantities of soluble carbohydrates are present in the diet. The entodiniomorphs are responsible for controlling starch digestion by engulfing whole starch granules. This restricts bacterial access to the starch and slows down the rate of fermentation. If large amounts of starch are present, these protozoa will actually engulf so many starch granules that they actually burst!

The major negative impact of protozoa is their effect on ruminant protein metabolism. The entodiniomorph protozoa are predators of rumen bacteria and engulf and digest them just as they engulf starch granules. This is why bacterial numbers are greater when animals are defaunated. Since protozoa tend to stay in the rumen and do not pass to the small intestine, they contribute little to the flow of proteins and because they digest the bacteria, total protein flow to the small intestine is generally reduced in the presence of protozoa.

Life and Death in the Rumen

From the perspective of the cow and the nutritionist, it would be best if rumen bacteria digested feed as fast as possible, reproduced and never died until they passed out of the rumen, into the small intestine. This would maximize the digestion of plant fibre and production of microbial protein. Under perfect continuous culture conditions, bacterial growth rate is equal to the rate that nutrients are made available and the rate that older, mature bacteria are washed out of the system. This means that carbohydrates, nitrogen, and all other growth factors are constantly present at the optimal amounts. Unfortunately, perfect conditions seldom, if ever, exist in the rumen. In fact, while we often think of the rumen as a continuous culture system, we know that cows do not eat continuously, even when offered a TMR several times per day. What actually occurs in the rumen is more kin to a "fed-batch system", where feed sporadically enters the system, with a fluctuating rate with which undigested feed and bacteria leave the rumen for the small intestine.

Under a fed-batch system, carbohydrates and nitrogen may be available for microbial growth at different times. This means that at certain times, bacteria will have to shift their metabolic machinery to accommodate a new growth pattern or a new nutrient. The metabolic machinery of the cell is made up of enzymes, which are proteins. In terms of energy usage, protein synthesis is the single most expensive process in a cell. Therefore, there is a tendency to only produce the minimum machinery that is needed to metabolize the substrates at hand. Every time we change the nutrient supply by providing a new substrate (by changing feeds) or altering the delivery of the substrate (by limit feeding or slug-feeding), we cause an inefficiency in the rumen by forcing the microbes to shift their metabolic machinery.

When ruminants are fed forages (the situation under which the rumen evolved), carbohydrates are almost always the first limiting nutrient for microbial growth. The cell machinery of rumen bacteria has been developed around the premise that energy will be first limiting. However, when we feed typical dairy diets containing starch, there are sporadic periods when the energy is not limiting.

During these periods, rumen bacteria have no ability to limit the uptake of the extra carbohydrates. Therefore, in order to maintain their cell machinery in balance, the bacteria must use a system to get rid of the extra energy. One such mechanism is "energy spilling" (Russell, 1998). Bacteria that do not spill energy have a low survivability. Therefore excess carbohydrate can be toxic.

One method of coping with excess energy is to store the carbohydrate as intracellular polysaccharides (Cheng et al., 1973) or extracellular polysaccharides (Costerton et al., 1974). The extra-cellular polysaccharides are the "slime" that is associated with feedlot bloat. Another method is to shift the fermentation pathway that is used. Some rumen bacteria such as *Strep bovis* can shift from producing acetate and propionate to lactate. Lactate production results is less available energy for microbial growth than acetate or propionate, but when energy is not a limitation, these bacteria can grow rapidly producing large amounts of lactic acid (Russell, 1998). Other bacteria use a different diversionary pathway called the methylglyoxal shunt pathway to produce D-lactic acid. Methylglyoxal is extremely toxic to both mammals and bacteria. Therefore if the formation of D-lactic acid is inhibited, the bacteria may form an auto-toxic compound.

Excess carbohydrates in the rumen is not only limited to high grain feeding, if amino acid nitrogen or ammonia is in short supply, carbohydrates can be excessive even on forage-based diets.

• pH - The Central Issue

Keeping the rumen healthy and in balance means that fibre will be digested at a maximal rate and feed intake will be maximized as well. Forages are seldom the sole source of feed for dairy cows and because concentrates are fermented faster in the rumen. More fermentation means more VFA production and a lower pH. While the rumen is very well buffered around pH 6.8, the buffering capacity is poor below pH 6.0. A major consequence when ruminal pH falls below 6 is that fibre digestion declines dramatically. There are two reasons for this. Firstly, the enzymes necessary for fibre breakdown do not function effectively at pH <6.0 and secondly, the growth rate of fibrolytic activity declines markedly at low pH. Not only are these bacteria not able to obtain the sugars necessary for growth, the low pH impedes growth itself. Fibrolytic bacteria are unable to maintain the pH inside their cells when ruminal pH is low. This incapacitates the cell machinery making growth impossible (Russell and Wilson, 1996).

Rumen pH affects non-fibrolytic microbes as well and has a major impact on the type of endproducts produced in the rumen. When bacteria produce acetate, H_2 gas is a necessary product. However, even small accumulations of

 H_2 will block further acetate production. Only if H_2 is further utilized, can bacteria obtain energy via acetate production. Methane and propionate production are two routes by which the rumen ecosystem can get rid of H_2 . If methane is produced, energy is lost from the system in a form the cow cannot use. If propionate is produced, the resultant energy is trapped in a form that the cow can metabolize. A declining pH greatly affects the bacteria that produce methane as well as the fibrolytic bacteria that produce the H_2 gas. This is why high concentrate diets result in low acetate, high propionate production in the rumen.

Effect of Direct-fed Microbials on Stabilizing the Rumen

Yeast cultures based on *Saccharomyces cerevisiae* are widely used in ruminant diets Available products vary widely in both the strain of *S. cerevisiae* used and the number and viability of yeast cells present. Not all strains of the yeast are capable of stimulating digestion in the rumen (Newbold *et al.*, 1995). These differences are not related to the number of viable yeast cells in the preparations (Newbold & Wallace, 1992), their ability to stimulate rumen fermentation may be related to differences in metabolic activity. It is also likely that the method of growing, harvesting and storing the culture will affect the final activity.

The effect of yeast culture on VFA proportions in the rumen has been reported widely. In some studies yeast culture stimulated the production of propionate at the expense of acetate (Newbold *et al.*, 1995). The highly variable nature of the response in VFA proportions to yeast supplementation, suggests that the effects of yeast on VFA proportions are a consequence of the effect of yeast on microbial numbers in the rumen rather than a direct effect of the yeast, that might help explain the gains observed in productivity (Wallace & Newbold, 1992).

An increase in the number of total culturable and cellulolytic bacteria that can be recovered from the rumen would appear to be one of the most consistently reported responses to *S. cerevisiae*, and while the increases in culturable bacteria in many studies might not reach statistical significance, studies in which yeast culture fails to stimulate bacterial numbers are rare. There is general agreement that the increased bacterial count seems to be central to the action of the yeast driving both the increased rate of fibre degradation in the rumen and the increased flow of microbial protein from the rumen (Wallace & Newbold, 1992).

A number of mechanisms by which the yeast might stimulate bacterial numbers have been proposed (Wallace & Newbold, 1992). Nisbet & Martin (1993) found

that malate stimulated the growth of the prominent gram-negative rumen bacterium *Selonomonas ruminantium* in medium containing lactic acid. Newbold *et al* (1998) found that yeast culture increased the number of *S. ruminantium* that could be recovered from rumen fluid

As previously mentioned, the rumen is widely considered to be anaerobic; nevertheless, the rumen gas, even in non-fistulated animals, contains between 0.5 and 1.0 % oxygen (McArthur & Multimore, 1962). Oxygen is toxic to anaerobic rumen bacteria, it inhibits bacterial growth and the adhesion of cellulolytic bacteria to fibre and yeast are well known for their high respiratory rate. Oxygen uptake by S. cerevisiae suggest that they have respiratory rates several orders of magnitude greater than rumen fluid alone (Newbold, 1995). Thus even at the low inclusions used in ruminant diets, yeast might be predicted to be beneficial to the rumen microflora. However, it is unlikely that the probiotic activity of yeast is derived solely from its ability to remove potentially harmful oxygen from the rumen.

Conclusion

Maintaining the rumen ecosystem is a balancing act. By maximizing fermentation, the cow obtains more VFA for energy and more microbial protein. However, more fermentation means more acid production and a lower rumen pH. Low rumen pH can depress fibre digestion and lead to metabolic disorders. As we expect more production from our cows, they will have to eat more readily fermentable diets to meet their energy and protein requirements. This, in turn, may result in the cow getting less nutrients out of the diet than we expected. A better understanding of the workings of the rumen as a whole ecosystem will enable us to maintain the fine balance between productivity and acidosis.

References

- Cheng, K.-J., R. Hironaka, D.W.A. Roberts, and J.W. Costerton. 1973. Cytoplasmic glycogen inclusions in cells of anaerobic Gram negative rumen bacteria. Can. J. Microbiol. 19:1501-1509.
- Coleman, G.S. 1988. The importance of rumen ciliate protozoa in the growth and metabolism of the host ruminant. Intl J. Anim. Sci. 3:75-95.
- Costerton, J.W., H.D. Damgaard and K.J. Cheng. 1974. Cell envelope morphology of rumen bacteria. J. Bacteriol. 118:1132-1143.
- Hespell, R.B., D.E. Akin, B.A. Dehority. 1997. Bacteria, fungi and protozoa of the rumen. Pages 59-149 *in* Gastrointestinal Microbiology. Volume 2. Gastrointestinal Microbes and Host Interactions. R.I. Mackie, B.A. White and R.E. Isaacson *eds.* International Thompson Press, New York.

- Leedle, J.A.Z., M.P. Bryant and R.B. Hespell. 1982. Diurnal variations in bacterial numbers and fluid parameters in ruminal contents of animals fed low- or high-forage diets. Appl. Environ. Microbiol. 44:402-412.
- McArthur, J.M. and J.E. Multimore. 1962. Rumen gas analysis by gas solid chromatography. Can. J. Anim. Sci. 41:187-192.
- Newbold, C.J. and R.J. Wallace. 1992. The effect of yeast and distillery byproducts on the fermentation in the rumen simulation technique (Rusitec) Anim. Prod. 54:504. (abstr.).
- Newbold, C.J., F.M McIntosh, and R.J. Wallace. 1995. Different strains of *Saccharomyces cerevisiae* differ in their effects on ruminal bacteria *in vitro* and in sheep. J. Anim. Sci., 73:1811-1818.
- Newbold, C.J., F.M McIntosh, and R.J. Wallace. 1998. Changes in the microbial population of a rumen-simulating fermenter in response to yeast culture. Can. J. Anim. Sci,. 78:241-244.
- Nisbet, D.J. and S.A. Martin. 1991. The effect of *Saccharomyces cerevisiae* culture on lactate utilization by the ruminal bacterium *Selenomonas ruminantium*. J. Anim. Sci. 69:4628-4633.
- Russell, J.B. 1998. The importance of pH in the regulation of ruminal acetate to propionate ratio and methane production in vitro. J. Dairy Sci. 81:3222-3230.
- Russell, J.B. and D.B. Wilson. 1996. Why are ruminal cellulolytic bacteria unable to digest cellulose at low pH? J. Dairy sci. 79:1503-1508.
- Warner, A.C.I. 1966. Periodic changes in the concentrations of microorganisms in the rumens of sheep fed to appetite in pens or on pasture. J. Gen. Microbiol. 45:237-242.

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