

Ruminant Nutrition Symposium: Acidosis: New Insights Into the Persistent Problem

1144 Role of fermentation acid absorption in the regulation of ruminal pH. J. R. Aschenbach*¹, G. B. Penner², F. Stumpff³, and G. Gäbel⁴, ¹University of Veterinary Medicine Vienna, Vienna, Austria, ²University of Saskatchewan, Saskatoon, Canada, ³Free University of Berlin, Berlin, Germany, ⁴University of Leipzig, Leipzig, Germany.

Diets with high energy density are rapidly fermented to acids (short chain fatty acids, SCFA; lactic acid) within the rumen. The resulting release of protons can constitute a challenge to the ruminal ecosystem and animal health. Health upsets resulting from acidogenic diets are classified as subacute and acute acidosis based on the degree of ruminal pH dysregulation. While increased acid production is a nutritionally desired effect of increased concentrate feeding, the accumulation of protons in the rumen is not. Consequently, mechanisms of proton removal and their quantitative importance are of major interest. From the 1950's, salivary buffers (bicarbonate, phosphate) have been identified as important mechanisms for ruminal proton removal. However, it appears that a larger proportion of protons is removed from the rumen by transport processes across the ruminal epithelium. Proceeding initially from exclusively diffusional absorption of fermentation acids, several protein-dependent mechanisms have been discovered over the last 2 decades. Although the molecular identity of these proteins is mostly uncertain, acetate absorption proceeds to a major part via acetate-bicarbonate exchange in addition to another nitrate-sensitive, bicarbonate-independent transport mechanism and lipophilic diffusion. Propionate and butyrate also show partially bicarbonate-dependent transport modes. In sheep, susceptibility to subacute ruminal acidosis was positively related to the efficiencies of protein-mediated acetate uptake and diffusional butyrate uptake. The latter seemed attributable to high rates of butyrate metabolism in the epithelial cells. Finally, SCFA absorption also accelerates urea transport via UT-B into the rumen which via ammonium recycling may remove protons from the rumen to the blood.

Key Words: nutrient absorption, ruminal acidosis, short chain fatty acids

1145 Molecular adaptation of ruminal epithelia to highly fermentable diets. G. B. Penner*¹, M. A. Steele², and B. W. McBride², ¹University of Saskatchewan, Saskatoon, Canada, ²University of Guelph, Guelph, Ontario, Canada.

Feeding highly fermentable diets to ruminants is one strategy to increase energy intake. However, the increase in short-chain fatty acid (SCFA) production and reduced ruminal pH associated with highly fermentable diets imposes a metabolic challenge to the ruminal epithelia. In response to greater SCFA supply, the ruminal epithelia respond with coordinated actions of altered cell function and proliferation. While the proliferative response is well documented, emerging evidence at the mRNA level indicates that temporal changes in epithelial cell function is the initial response. It is not surprising that gene expression analysis has identified pathways involved in fatty acid metabolism, ion transport, and intracellular homeostasis to be the pathways dominantly affected during adaptation and following adaptation to a highly fermentable diet. It is widely acknowledged that intraepithelial metabolism of SCFA, particularly butyrate, helps to maintain the concentration gradient between the cytosol and lumen thereby facilitating absorption. Current data suggests that for butyrate metabolism, 3-hydroxy-3-methylglutaryl-CoA synthase 1 is the regulatory point with transient up and downregulation during diet adaptation. Interestingly, rate-limiting enzymes involved in chole-

sterol biosynthesis appear to be downregulated during diet adaptation. In addition to nutrient transport and utilization, genes involved in the maintenance of tight cell junctions and induction of the inflammatory response have been identified as differentially expressed genes during adaptation to highly fermentable diets. This may have important implications on ruminal epithelial barrier function and the inflammatory response associated with subacute ruminal acidosis.

Key Words: adaptation, gene expression, rumen epithelia

1146 Animal productivity and health responses to hind-gut acidosis. T. F. Gressley*¹, M. B. Hall², and L. E. Armentano³, ¹University of Delaware, Newark, ²US Dairy Forage Research Center, Madison, WI, ³University of Wisconsin, Madison.

Microbial fermentation of carbohydrates in the large intestine of dairy cattle is responsible for 5 to 10% of total tract carbohydrate digestion. When dietary, animal, and/or environmental factors contribute to abnormal, excessive flow of fermentable carbohydrates to the large intestine, hind-gut acidosis can occur. Hind-gut acidosis is characterized by increased rates of production of volatile fatty acids (VFA) including lactic acid, decreased digesta pH, and damage to gut epithelium as evidenced by the appearance of mucin casts in feces. In parallel to ruminal disorders, it is possible that hindgut acidosis can also be classified as acute or sub-acute. In the more severe situations, hind-gut acidosis is characterized by an inflammatory response; the resulting breach of the barrier between animal and digesta may contribute to laminitis and other disorders. In a research setting, hind-gut acidosis has been evaluated using pulse-dose or continuous abomasal infusions of varying amounts of fermentable carbohydrates. Continuous low dose infusions of pectin or fructans (1 kg/d) into lactating cows resulted in decreased diet digestibility, increased intake variation, decreased milk fat percentage, decreased milk urea N concentration, and increased fecal N output, without affecting fecal pH or VFA. Pulse dose fructan infusions (1 g/kg BW) into steers resulted in watery feces, decreased fecal pH, and increased fecal VFA, without causing an inflammatory response. Daily 12 h abomasal infusions of a high dose of starch (~4 kg/d) have also induced hind-gut acidosis as indicated by decreased fecal pH and watery feces. Evidence of hind-gut acidosis has also been noted on farms as detected by fecal signs of excessive fermentation (watery or foamy appearance) or of epithelial damage (presence of mucin casts). In summary, hind-gut acidosis occurs as a result of relatively high rates of large intestinal fermentation, likely as a result of digestive dysfunction in other parts of the gut. A better understanding of the relationship of this disorder to other animal health disorders is needed.

Key Words: acidosis, hind-gut

1147 Bovine endotoxemia: Does acidosis cause inflammatory responses? P. H. Andersen*, Copenhagen University, Copenhagen, Denmark.

Endotoxins (LPS) are structural parts of the gram-negative bacteria membrane and belong to the group of pathogen-associated molecular patterns (PAMP), that are instantly recognized by the innate immune system. LPS and gram-negative bacteria are generally present in the rumen of cattle, and it is now widely accepted, that the LPS concentration increases when grain is added to the diet. LPS in even very minute amounts (10 ng per kg BW) elicit an inflammatory response in cattle.

Clinical and biochemical signs such as decreased GI motility, anorexia, increased concentrations of acute phase proteins, leukocytosis and leukopenia appear in endotoxemia as well as in acute ruminal acidosis. In cattle, fever is not a consistent sign of endotoxemia. If ruminitis is present, the ruminal barrier may become leaky. In experimentally induced acute ruminal acidosis, increased amounts of LPS can be detected in the portal vein and sometimes also in the systemic circulation. Controversy exists whether this is the case also in cows with subclinical ruminal acidosis. However, concentrations of the acute phase protein serum amyloid A increase in subclinical ruminal acidosis, and results from recent in vitro experiments have shown that the permeability of the rumen epithelium to LPS increases in the presence of LPS and low pH. Whether the inflammatory signs detected in ruminal acidosis

may be ascribed to the occurrence of ruminitis, to the consequences of increased translocation of LPS from the inflamed rumen epithelium to the portal blood system, or to combination of these, is however not yet entirely clear. Interestingly, large variations in the clinical susceptibility of cattle to LPS are reported from various studies on rumen acidosis and mastitis. Cows may be divided into “moderate” and “severe” responders. The detoxifying capacity of the macrophages in the liver may play a role, but the causative factors are far from elucidated. It is suggested that further investigation of the response of cattle to LPS or other PAMP challenges may provide a basis for the identification of indicators of robustness or health.

Key Words: endotoxin, ruminal acidosis, inflammation