Feeding Protected Sodium Bicarbonate Attenuates Hindgut Acidosis in Horses Fed a High-Grain Ration

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Hindgut acidosis (HGA) is a common problem in horses consuming either large quantities of grain or fructan-rich forages. Horses suffering from HGA may develop anorexia, colic, or laminitis or display stereotypical behaviors such as wood chewing and stall weaving. Feeding a protected sodium bicarbonate (PSB) is a safe and effective method of attenuating HGA without producing a metabolic alkalosis. Authors' address: Kentucky Equine Research, Versailles, KY 40383; e-mail: pagan@ker.com (Pagan).

1. Introduction

Horses evolved as wandering herbivores with voluminous hindguts adapted to process large quantities of high-fiber forage. The primary microorganisms populating the hindgut of horses are fiber-fermenting bacteria that depend on cellulose and hemicellulose as their primary energy substrates. Smaller populations of bacteria capable of rapidly fermenting soluble carbohydrates also inhabit the hindgut. When horses eat a diet high in fiber, the environment in the hindgut favors the fiber-fermenting bacteria. When large grain meals are fed to horses, a portion of the starch may escape digestion in the small intestine and be rapidly fermented in the cecum and colon. Volatile fatty acid (VFA) and lactic acid production increases, causing a significant decrease in pH. Lactic acid is a stronger acid than the VFA and may cause irritation or damage to the intestinal mucosa. In severe cases, lactate may contribute between 50% and 90% of the total acids in the hindgut.[1] Furthermore, lactic acid accumulation increases the permeability of the large intestinal mucosa to toxins and larger molecules that have been implicated in the development of equine laminitis.[2] A downward shift in pH provides an unfavorable environment for many of the fiber-fermenting microorganisms that inhabit the hindgut. In particular, bacteria such as *Ruminococcus albus* and *Fibrobacter succinogenes* are sensitive to precipitous decreases in pH. For optimal performance, these bacteria favor an environment with a pH between 6.5 and 7.0. When pH drops $<$6.0, fiber-digesting bacteria become less efficient and begin to die off. In contrast to fiber-digesting bacteria, lactate-producing and lactate-using bacteria thrive in an environment with a low pH. Certain microorganisms such as *Streptococcus bovis* actually shift their metabolism and produce lactic acid rather than VFAs when exposed to acidic conditions, serving only to compound the problem.[1] Changes in the pH of the hindgut caused by alterations in the microbial populations and acid profiles may result in hindgut acidosis (HGA).

NOTES

Horses suffering HGA may develop anorexia, colic, or display stereotypical behaviors such as wood chewing and stall weaving.[3] Furthermore, long-term exposure to pH <5.8 will begin to have deleterious effects on the epithelial lining of the colonic and cecal walls that may affect absorptive capacity.

Rumen acidosis is a common problem in dairy cattle fed high-grain diets. Sodium bicarbonate is often added to a cow's ration as a buffer to attenuate drops in rumen pH that decrease feed intake and milk production. Sodium bicarbonate has been shown to be effective in treating hindgut acidosis in horses when it is infused directly into the cecum through a cecal fistula.[4] Unfortunately, feeding raw sodium bicarbonate to horses is ineffective because of the anatomy of the gastrointestinal tract. Ideally, the sodium bicarbonate should be protected so that it is delivered to the hindgut intact. Kentucky Equine Research, in conjunction with Balchem Corporation, has recently developed a protected sodium bicarbonate (PSB)[a] that survives transit through the stomach and small intestine of the horse. The following study was conducted to assess its effectiveness at treating HGA in horses fed a high-grain ration.

2. Materials and Methods

Six 5-yr-old Thoroughbred horses were used in a study to evaluate the effect of PSB supplementation on hindgut acidosis in horses fed a high-grain ration. The horses were in a regular training program for several months before beginning the study and were considered physically fit. The study used a switchback design with each period lasting 4 wk. The horses remained in training on a high-speed treadmill throughout the study.

Horses were fed a basal diet of unfortified sweet feed, timothy grass hay, and 50 g of loose salt per day. Grain intakes ranged from 4 (two horses) to 6 kg (four horses) per day. Hay was fed at a rate of 5 kg for the first 2 wk of each period. Hay fed was decreased to 4 kg for the latter half of each period. Horses were split into two groups and assigned to one of two treatments. The treatments were 168 g/day of PSB[a] or the basal diet (control group). Horses switched treatments for period 2. Both the hay and grain portion of the diet were split into two equal feedings. The grain portion of the diet was fed at 7:00 a.m. and 4:00 p.m., and the hay portion of the diet was fed at 7:00 a.m. and 10:00 p.m. One half of the PSB (84 g) was added to each grain meal.

Blood and fecal samples were taken at 2-h intervals for an 8-h period on day 15 of each period. The first samples (0 h) were taken immediately before the horses received their morning grain. Subsequent samples were taken at 2, 4, 6, and 8 h after feeding. pH, pCO_2, HCO_3^-, Na^+, K^+, Cl^-, and tCO_2 were measured in whole blood samples using an automated electrolyte and blood gas analyzer.[b] VFAs, pH, and L- and D-lactate concentration were measured in fecal grab samples taken from the rectum. pH was measured in 50 g of feces diluted in a 3:1 ratio with distilled water using a pH electrode.[c] Fecal total VFAs were measured using gas chromatography, and lactates were measured colorimetrically using a commercially available kit. Data were analyzed using analysis of variance (ANOVA) for repeated measures, with a significance level set at $p < 0.05$.

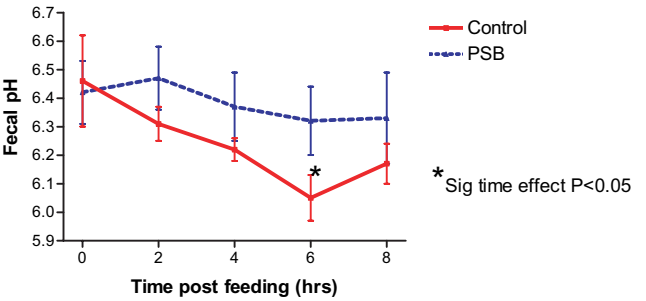

Fig. 1. Fecal pH on day 15.

During week 4 of each period, horses were fitted with collection harnesses, and a 5-day complete fecal and urine collection was conducted. A 2-day harness adaptation preceded the 5-day collection. Fecal and feed samples were analyzed for dry matter (DM), crude protein (CP), acid detergent fiber (ADF), neutral detergent fiber (NDF), fat, ash, Ca, P, Mg, K, Na, Cl, Fe, Zn, Cu, and Mn. Urine samples were analyzed for mineral content.

3. Results

Fecal pH in the control group decreased significantly from baseline by 6 h after feeding (Fig. 1). Fecal pH in the PSB group did not exhibit any significant fluctuations during the 8-h sampling period.

Fecal L-lactate and D-lactate were significantly higher ($p < 0.05$) in the control group after feeding (Figs. 2 and 3). L-lactate was higher 2 and 6 h after feeding, and D-lactate was higher 6 h after feeding.

Fecal VFAs were significantly higher ($p < 0.05$) in the PSB group than the control group at 0 h. VFAs increased in the control group after feeding and

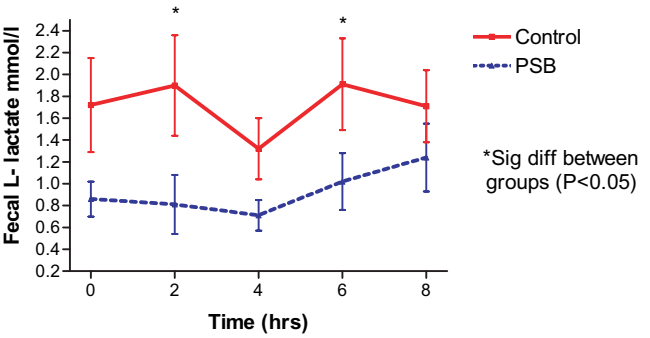

Fig. 2. Fecal L-lactate concentration on day 15.

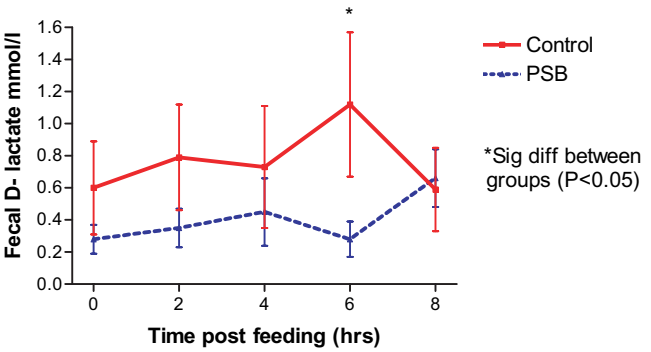

Fig. 3. Fecal D-lactate concentration on day 15.

were significantly higher than 0 h at 2, 4, 6, and 8 h after feeding ($p < 0.05$) (Fig. 4). Fecal VFAs were significantly higher in the PSB group 0 h after feeding ($p < 0.05$).

There was no significant difference in tCO_2 between the two groups at any time before or after feeding (Fig. 5).

Blood pH was significantly lower compared with baseline in both groups 8 h after feeding. Plasma chloride concentration was significantly higher compared with baseline within both groups 2 h after feeding. Values remained elevated in the control group up to 8 h after feeding. No significant differences were recorded in blood PCO_2, HCO_3, sodium, and potassium.

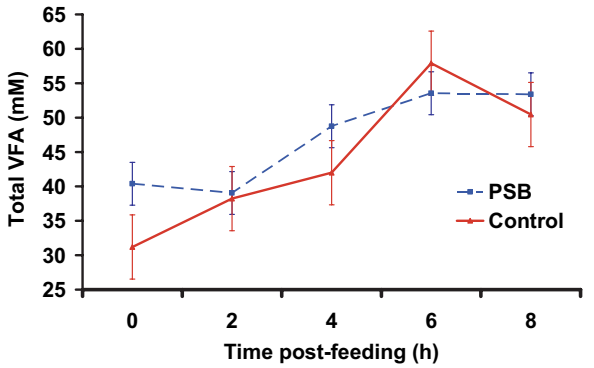

Fig. 4. Total fecal VFA measured on day 15.

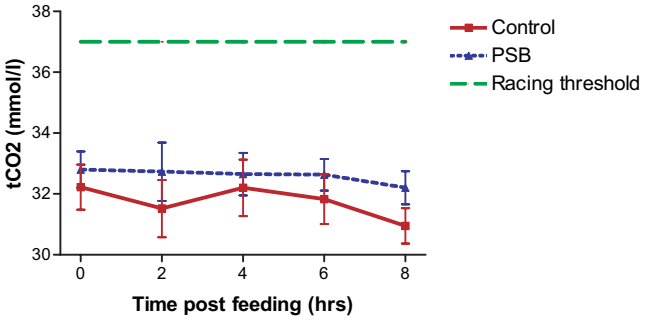

Fig. 5. Blood tCO_2 values on day 15.

Despite a trend toward increased apparent digestibility values for NDF, hemicellulose, fat, and sodium in horses receiving PSB, no statistical differences were measured in the apparent digestibility of any of the nutrients analyzed. However, the total amount of fat ($p < 0.05$) and sodium ($p < 0.001$) absorbed was significantly higher in horses receiving PSB. Overall, sodium retention was higher in horses receiving PSB (9.6 ± 1.8 versus 6.6 ± 0.5 g).

4. Discussion

Feeding 2–3 kg of sweet feed to Thoroughbreds in a single meal resulted in a significant drop in fecal pH 6 h after feeding. This drop was the result of a combination of microbial production of VFAs and lactic acid in the hindgut. Addition of PSB to the diet attenuated the drop in fecal pH and reduced the concentration of fecal lactic acid, but it had no effect on VFAs, suggesting that its primary action was related to either producing lactate or using microorganisms.

The encapsulation agent used to protect the sodium bicarbonate in the PSB is hydrogenated vegetable oil. Increased absorption of fat and sodium in the supplemented group indicates that some of the PSB was digested and absorbed, although the site of absorption is unclear. PSB did not adversely affect the digestibility of any other nutrients in the ration, and a trend toward improved hemicellulose and NDF digestibility suggests that there may have been an improvement in fiber fermentation resulting from a stabilized hindgut environment.

Sodium bicarbonate is sometimes administered as an alkalizing agent to racehorses in an attempt to enhance performance. Levels of sodium bicarbonate used to produce a metabolic alkalosis generally range from 0.3 to 0.6 g/kg body weight.[5] The dose rate of PSB used in this study contained 0.1 g $NaHCO_3$/kg body weight per feeding, and this did not produce a metabolic alkalosis in the horses as evidenced by the lack of a significant treatment effect on blood pH, PCO_2, HCO_3, or tCO_2. This was probably because of a combination of the low dose rate and the protective coating that reduces the breakdown of sodium bicarbonate in the stomach and small intestine. Because of the purported performance-enhancing effect of sodium bicarbonate, dose rates of sodium bicarbonate that result in blood tCO_2 levels of 37 mmol/l or higher are considered prohibited in most racing jurisdictions. The dose rate of PSB used in this study did not significantly elevate tCO_2, and a dose rate of PSB containing 0.3 g HCO_3/kg body weight/feeding did not produce prohibited levels of blood tCO_2.[d]

The PSB used in this study was effective in attenuating the HGA that resulted from high-grain intakes in exercised Thoroughbreds. More research is needed to evaluate how PSB supplementation affects intestinal epithelial health and integrity.

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[a]EquiShure, Kentucky Equine Research, Versailles, KY 40383.
[b]Idexx Vetstat, Idexx Laboratories, Westbrook, ME 04092.
[c]VWR SympHony, VWR International, San Dimas, CA 91773.
[d]Pagan JD. Unpublished results. 2007.