

Hindgut acidosis common in horses

Hindgut acidosis is a common problem in horses consuming either large quantities of grain or fructan-rich forages. Feeding an encapsulated sodium bicarbonate is a safe and effective method of attenuating the condition.

By **JOE D. PAGAN***

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 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. 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.

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 below 6.0, fiber-digesting bacteria become less efficient and begin to die off.

In contrast to fiber-digesting bacteria, lactate-producing and lactate-utilizing 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.

Changes in the pH of the hindgut due to alterations in the microbial populations and acid profiles may result in hindgut

acidosis (HGA; Figure 1).

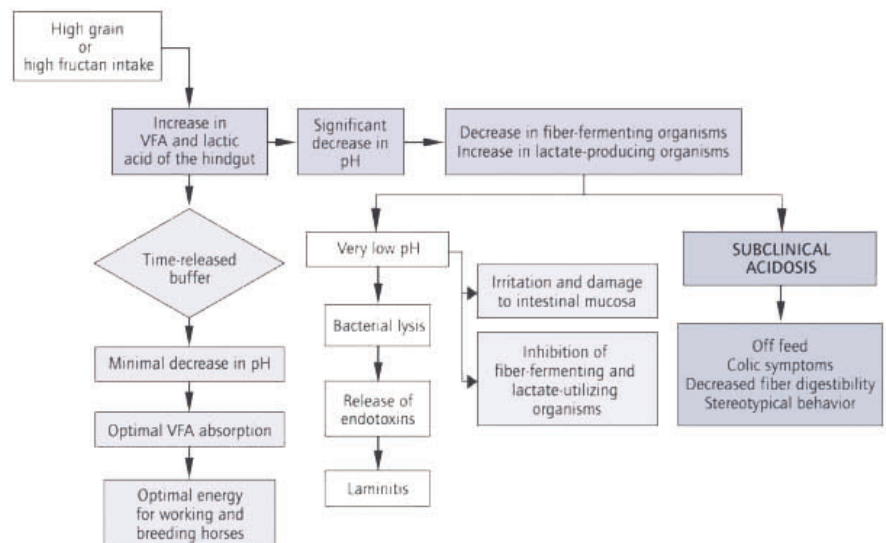
HGA can also be caused by pasture grasses rich in fructans. Fructans are polysaccharides of d-fructose that serve as storage forms of sugar within plants. They are resistant to breakdown by mammalian digestive enzymes.

Microbial digestion of fructans results in production of VFAs and lactic acid similar to cereal grain digestion in the hindgut. As with large amounts of grain, high fructan intakes overwhelm the hindgut, resulting in rapid fermentation, accumulation of lactic acid and a deleterious decrease in pH. Fructans are believed to be involved in the pathogenesis of pasture-induced laminitis in horses.

Horses suffering from HGA may develop anorexia, colic or display stereotypical behaviors such as wood chewing and stall weaving. Furthermore, long-term exposure to pHs below 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.

1. How hindgut acidosis can occur in hindgut and how it can be attenuated



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SODIUM bicarbonate has also been shown to be effective in treating HGA in horses when it is infused directly into the cecum via a cecal fistula.

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 (KER), in conjunction with Balchem Corp., has recently developed an encapsulated sodium bicarbonate (ESB; EquiShure) that survives transit through the stomach and small intestine of the horse.

KER conducted a series of studies to evaluate the effect of ESB on HGA in horses fed high levels of starch or fructans.

In one study, six thoroughbred horses in training 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 to 6 kg per day. Horses were split into two groups and assigned to one of two treatments. The treatments were 168 g per day of ESB 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. One-half of the ESB (84 g) was added to each grain meal.

Fecal samples were taken at two-hour intervals for an eight-hour period on day 15 of each period and were analyzed for VFAs, pH and L- and D-lactate concentration.

Fecal pH in the control group decreased significantly from the baseline by six hours post-feeding (Figure 2).

Fecal pH in the ESB group did not

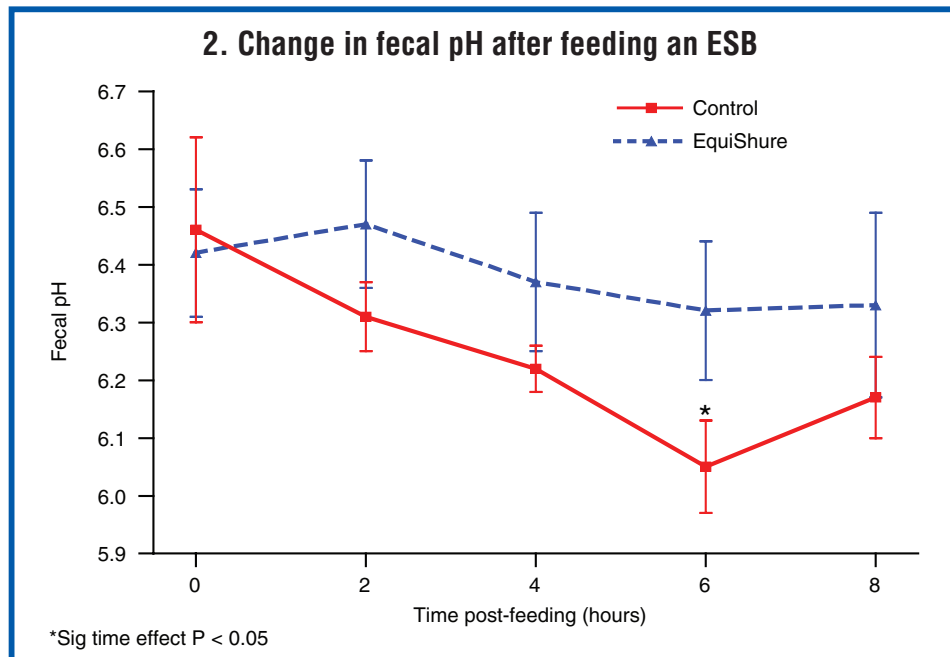


exhibit any significant fluctuations during the eight-hour sampling period.

Fecal L-lactate and D-lactate were significantly higher ($P < 0.05$) post-feeding in the control group compared to the ESB group. Fecal VFAs were significantly higher ($P < 0.05$) in the ESB-supplemented group, suggesting a more favorable environment for fiber-fermenting bacteria.

The ESB was effective in attenuating HGA that resulted from high-grain intakes in exercised thoroughbreds.

In another study, ESB-supplemented

horses experienced a much smaller increase in fecal lactate when they were exposed naively to pasture for a 24-hour period, suggesting that hindgut buffering is also effective at attenuating HGA caused by pasture fructan intake.

In summary, HGA is a common problem in horses consuming either large quantities of grain or fructan-rich forages. Feeding an ESB is a safe and effective method of attenuating HGA.

More research is needed to evaluate how ESB supplementation affects intestinal epithelial health and integrity.