



Subclinical Acidosis: Is Your Horse At Risk?



It is no secret among informed horse owners that the horse's gastrointestinal tract is both a wondrous and a delicate creation. When working at full capacity, the tract is able to efficiently convert grasses and grains to energy. When things go awry with the gastrointestinal tract, however, a horse's life might hang in the balance.

Diagrams and descriptions of the equine gastrointestinal tract neatly differentiate the foregut from the hindgut. The foregut includes the mouth and its many components, esophagus, stomach, and small intestine, and accounts for about 35 to 40% of the capacity of the gastrointestinal tract. The hindgut includes the cecum, large colon, small colon, and rectum. The capacities of the foregut and hindgut of the horse are markedly different than those of its barnyard contemporaries. Ruminants such as cattle have significantly more voluminous foreguts that account for 85 to 90% of total gastrointestinal capacity.

Because of the limited size of the horse's foregut, digesta (swallowed food as it undergoes digestion) spends little time there when compared to the hours it spends progressing through the hindgut. One core feature of the hindgut is the fragile population of microorganisms that inhabit it. Anaerobic bacteria, fungi, and protozoa coexist contentedly in the hindgut when the system is working proficiently. Together the microbes' primary responsibility is to digest fiber.

The breakdown of fiber in the hindgut results in the production of volatile fatty acids (VFA), which permeate the walls of the cecum and colon, hitch a ride in the bloodstream, and end up in the liver, where they are used by the horse to fuel athletic or reproductive endeavors.

Causes of Subclinical Acidosis

Certain situations trigger the pH of the hindgut to drop sharply. The two most common causes are the overconsumption of high-starch concentrates or pasture grasses rich in fructan. The demands placed on horses—as athletes and as breeding animals—dictate that substantial quantities of energy-laden feeds be consumed.

When either of these feeding scenarios occurs, it is impossible for the stomach and small intestine to sufficiently digest and absorb the massive onslaught of starch. Accordingly, some starch moves into the hindgut without being adequately digested. As digestion of easily-fermentable starch progresses in the hindgut, the production of VFA and lactic acid increases, causing a significant decrease in the pH. When the hindgut endures insults such as this several times a day, it teeters on becoming overwhelmed with acid. Additionally, because lactic acid is stronger than VFA, it can cause serious damage to the intestinal mucosa. In severe

cases, lactate may contribute between 50 and 90% of the total acids in the hindgut.

The shift in pH provides an unfavorable environment for some of the many microorganisms that inhabit the hindgut and aid in digestion. In particular, fiber-digesting 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, which is often the case with subclinical acidosis, 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 VFA 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 cause a condition known as subclinical acidosis.

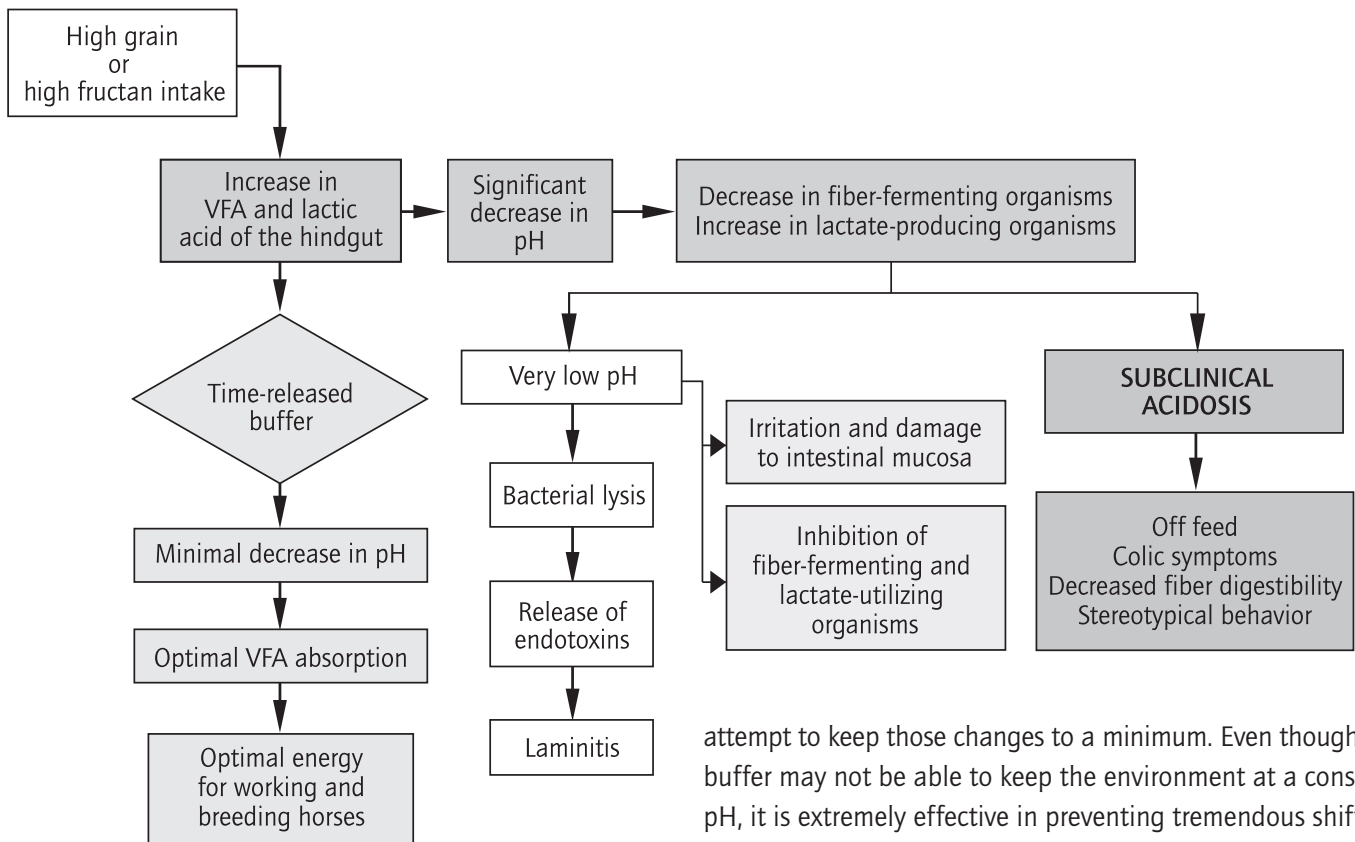
Signs of Subclinical Acidosis

One of the primary signs of subclinical acidosis is inappetence or decreased appetite. A horse is often reported to be "off his feed." Because the hindgut is overwhelmed with lactic acid when a horse is experiencing acidosis, the intestinal lining becomes inflamed and irritated, causing the horse discomfort. The irritation may be severe enough to induce behavior characteristic of colic. Furthermore and perhaps most detrimental to equine athletes is a reduction of feed efficiency. Long-term exposure of the intestinal lining to a low-pH environment may negatively affect the absorptive capacities of these structures, limiting the amount of energy available for performance.

In addition to these health concerns, a link between subclinical acidosis and stereotypies such as wood chewing and stall weaving has been suggested by researchers.

Because of the precarious nature of the hindgut of a horse afflicted with subclinical acidosis, it is less able to handle metabolic crises that healthy horses may be able to fend off. Therefore, horses with subclinical acidosis are more susceptible to colic and laminitis.

Figure 1. The chain of events that leads to subclinical acidosis can be offset with a time-released buffer such as EquiShure.



Managing Clinical Acidosis

Acidosis is not a novel health risk among domestic animals. Researchers first set out to solve the problem of acidosis in dairy cattle. Because cows must consume large quantities of grain to support milk production, their rumens—which, in terms of microflora population, are analogous to the cecum and colon of the horse—become overwhelmed with VFA and lactic acid following each meal, inciting ruminal acidosis. Afflicted cows might exhibit abnormal or erratic eating patterns. More often than not, they eat less and that leads to decreased milk production and reduced profit. As a result, the need to regulate the pH in the rumen became apparent. Researchers and dairymen accomplished this successfully by adding a buffer such as sodium bicarbonate to the feed.

A buffer is an agent that helps a target environment—in this case a gastrointestinal organ—resist changes in pH. Sodium bicarbonate is a well-known buffer. Because scientists know that changes in pH are unavoidable in these organs under certain feeding conditions, a buffer is used prophylactically in an

attempt to keep those changes to a minimum. Even though the buffer may not be able to keep the environment at a constant pH, it is extremely effective in preventing tremendous shifts in pH. Ultimately, it is these dramatic ups and downs that bring on subclinical acidosis.

With research on dairy cattle as a premise, the solution for managing horses with subclinical acidosis seemed simple enough: supply the hindgut with a buffer that minimizes fluctuations in pH when significant quantities of easily fermentable carbohydrates are offered.

As researchers delved into this possibility, they were confronted with a frustrating problem almost immediately. The horse's fermentation vat, collectively composed of the cecum and colon, is located at the end of the gastrointestinal tract rather than near the beginning, like the cow's rumen. In order to reach the horse's hindgut, a buffer must withstand passage through the stomach and small intestine. Regrettably, the enzymes secreted in these organs are not particularly hospitable to buffers. By the time an ordinary buffer reaches the hindgut, it loses its efficacy. Researchers went back to the drawing board and found the answer, a time-released buffer.

Proven Time-Released Buffer

Research supports the use of a hindgut buffer in cases of high grain and high fructan intake. Scientists at the University of Kentucky examined the effects of a buffer infused directly into

Hours	Hay vs Concentrate		NaHCO ₃ vs Concentrate	
	pH	pH	pH	pH
3	7.01	6.77	7.26	6.77
4	6.92	6.43	7.15	6.43
5	6.89	6.27	6.76	6.27
6	6.57	6.12	6.82	6.12

Table 1. Researchers infused the cecums of horses with a buffer (NaHCO₃) in an attempt to raise the cecal pH. The data revealed that cecal infusion worked. This early research served as a premise for the work more recently completed at KER.

manufacture of VFA lowered pH. Lower pH as a result of increased VFA concentration is much less harmful than low pH caused strictly by lactic acid production. Why? VFA are weaker acids and the effect of VFA on the hindgut pH is short-lived. As an end product of digestion, VFA are absorbed by the hindgut to enable use as an energy source. When high concentrations of fructans are found in pasture and horses are processing these highly fermentable carbohydrates in the hindgut, a time-released buffer helps moderate pH by preventing the drastic drop, thus encouraging fiber-digesting bacteria to thrive.

Another study conducted at KER demonstrated the efficacy of EquiShure when given to horses that had been denied access to pasture (perhaps due to injury-related stall rest). Horses that had not been turned out on pasture for several weeks were given 24-hour free-choice access to fall pasture. One group of horses was given a time-released buffer (EquiShure) for one week prior to turnout, and another group served as controls and was given no buffer. Initial pH readings revealed a moderate decrease in pH despite the buffer, but analysis of VFA proved that the drop in pH was associated with increased VFA production; therefore more were being produced and available to the horse as energy sources (Figure 2). Additional analysis demonstrated that lactic acid was greater in the control group vs. the EquiShure-fed group, which meant the buffer was effective and the hindgut was functioning optimally (Figure 3).


Using scientific studies conducted at KER and other research institutions, KER has produced EquiShure, a time-released buffer designed especially for the horse. When EquiShure is fed, only a minimal decrease in hindgut pH occurs, allowing for optimal absorption of all feedstuffs and optimal production and absorption of VFA so horses have sufficient energy to perform. 

Figure 2. Total VFA from fecal samples after exposure to pasture.

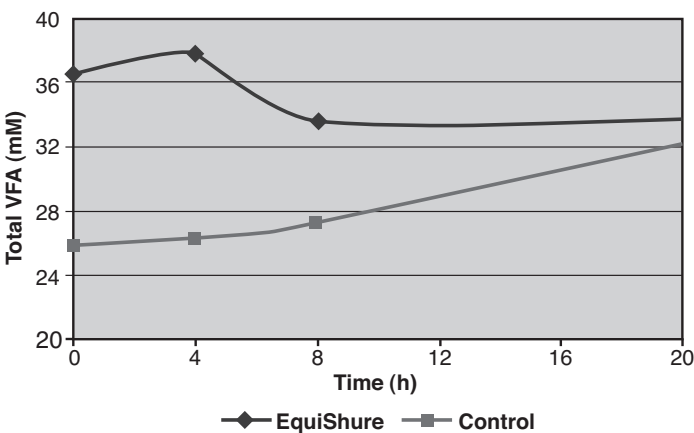
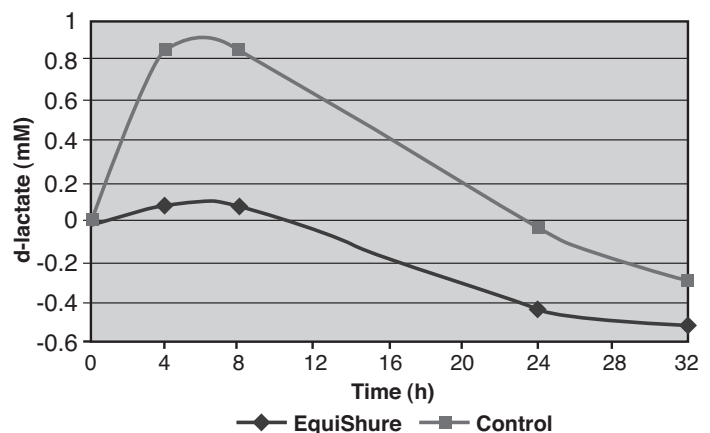


Figure 3. Change in fecal lactic acid (d-lactate) after exposure to pasture.





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