

## **MAINTAINING A HEALTHY GASTRO-INTESTINAL TRACT**

**By Bart Halsberghe, DVM**

**We as veterinarians are often questioned about medications and supplements that are potentially helpful in preventing and treating gastro-intestinal tract disorders. There are literally hundreds of products out there that are marketed as beneficial to the equine gastro-intestinal tract. Our goal at PenEq is to educate owners, trainers and horse people about the different medications and supplements that are out there, so that you understand why and when certain products are used and are able to make an informed decision about which products contain the right ingredients in the right amount to be effective for preventing or treating a specific gastro-intestinal problem. Dr. Bart Halsberghe has made a selection of products that he believes have the science behind them. He has provided a comprehensive summary of these products on our website [www.peneq.com](http://www.peneq.com). Do not be discouraged when you work through our online article, it is not designed to tell you what to do but to give you the knowledge to form your own opinion. And should you have questions, the veterinary staff here at PenEq is always here to help you interpret the data and make the best decisions for your horse!**

### **1. Understanding the digestive tract**

#### **1.1. Mouth and Stomach**

The amount of saliva secreted in the mouth depends on the quantity of feed material that is ingested. Though it does not contain digestive enzymes like amylase, saliva serves several other important functions. The bicarbonate buffers the acidity in the stomach and the high mucus content lubricates the feed.

The stomach is divided into two sections, which have both anatomical and physiological differences. Three digestive processes take place in the stomach: fermentation, enzymatic digestion and acid secretion. In the cranial non-glandular section, bacterial fermentation of the ingested feed starts. This mainly involves lactobacteria, which convert any available simple sugars or starches to lactic acid. This microbial activity and degradation stops when the gastric contents pass to the glandular section and mix with the acid stomach juice containing pepsin and lipase (two digestive enzymes). When large concentrate meals are fed, there is reduced mixing of the feed with the gastric juices and therefore an increased risk of dysfermentation. Whether this results in a clinical or subclinical problem will depend on the amount of available sugars and starches and the individual microbial population, etc.

## **1.2. Small intestine**

The small intestine is the main site of digestion and absorption of protein, fat, starch and sugars, vitamins, and minerals. Similar to the stomach, intake level of the feed influences rate of flow of ingesta through the small intestine. Large amounts fed in meal feedings increase rate of flow to the large intestine. Physical form of the feed also influences rate of flow, as pelletized rations, which pass more quickly than textured grain mixes combined with hay. Liquids pass to the cecum quickly, a few hours after ingestion of a meal.

Enzymes secreted in the small intestine (pancreatic and mucosal enzymes) break down carbohydrates (sugars, starch), protein and fat into simple sugars (glucose, etc.), amino acids and fatty acids that then can be absorbed through the intestinal wall into the blood circulation.

The horse, however, has a limited capacity to digest starch in the small intestine. The exact limit does seem to vary with the individual. Values of around 2 g starch/kg bodyweight per meal (an average grain or sweet feed mix contains 40-50% starch, this means 2-2.5 kg concentrate for a 500kg horse) have been suggested in the literature to be the maximum that should be fed in any one meal.

## **1.3. Large intestine (cecum and colon)**

Ingesta moves through the small intestine quite rapidly, reaching the caecum within 45 min to 3 hours of eating. The rate of passage will depend on the nature of the feedstuff and the way in which it is fed. Small particles, ground and pelleted types of feed tend to move more quickly through the large intestine than fresh grass. Fibrous feeds such as hays tend to have the slowest passage of all.

The large intestine does not have mucosal enzymes and does not have any significant active transport mechanisms for sugars and amino acids. Digestion and absorption of residual carbohydrates relies instead on microbial action and absorption of the end products of microbial fermentation. The intensity of these processes depends on the amount and the temporal influx of fermentable material arriving from the small intestine. If fed appropriately, a high proportion of the available starch ingested can be degraded to glucose for absorption in the small intestine (unless for example the digestive capacity of the small intestine is overwhelmed). However, depending on the extent of lignification, a varying proportion of the dietary fiber will be subjected to microbial fermentation, primarily in the large intestine, producing predominantly short chain or volatile fatty acids (VFA). These can be used directly as an energy fuel by the gut cells themselves, but the majority is absorbed and converted to either glucose or fat.

## **2. Optimizing intestinal tract health**

### **2.1. The use of gastroprotectants in treating gastric ulceration**

Gastroprotectants are among the most commonly used drugs in veterinary medicine because the gastrointestinal (GI) tract can be injured secondary to a plethora of

diseases and administration of various drugs. Many drugs have been developed to decrease intra-luminal acidity and/or promote mucosal protective defense mechanisms to prevent and treat ulcerative disorders. These drugs include histamine (H<sub>2</sub>)-receptor antagonists, proton pump inhibitors (PPIs), sucralfate, misoprostol, antacids and bismuth subsalicylate.

### **a) H<sub>2</sub>-receptor antagonists**

H<sub>2</sub>-receptor antagonists (H<sub>2</sub>-RAs) are analogues of histamine that competitively and reversibly inhibit the binding of histamine to H<sub>2</sub> receptors on the gastric parietal cell and thereby inhibit the secretion of acid by gastric parietal cells.

Four H<sub>2</sub>-RAs are available: cimetidine, ranitidine, famotidine and nizatidine. These drugs differ in their potency, duration of action, prokinetic effects and tendency to interact with other drugs. The order of potency of gastric acid inhibitors is: Famotidine > Ranitidine = Nizatidine > Cimetidine.

Acid inhibition after cimetidine administration peaks at 75% within 1.5 hours, and the effect of the drug is negligible after 5 hours. This relatively short biologic effect necessitates administration of the drug at least every 8 hours. Cimetidine can also interfere with the clearance of drugs. Ranitidine inhibits acid secretion to a greater extent than does cimetidine and has a longer duration of action, necessitating only twice daily dosing. Ranitidine has fewer drug interactions than cimetidine and also has a prokinetic effect on the GI tract.

Famotidine, the most potent H<sub>2</sub>-RA, has a longer duration of action than does cimetidine or ranitidine and thus requires only once daily dosing. Famotidine has no drug interaction and concurrent use with other medications is therefore not a concern.

Nizatidine, like ranitidine, has a prokinetic effect and like famotidine, nizatidine has no drug interactions.

To treat ulcer disease in humans, standard doses of H<sub>2</sub>-RAs are effective in healing approximately 75% to 90% of uncomplicated NSAID-related ulcers after discontinuation of the offending drug. If NSAID therapy is not discontinued, H<sub>2</sub>-RAs are not as effective in ulcer healing. The duration of treatment in humans is generally 4 to 8 weeks for uncomplicated ulcers and 6 to 12 months for complicated or recurring ulcers. Because of the lack of such information in veterinary patients, treatment times need to be extrapolated from human studies.

A plethora of large trials in human medicine have examined the effect of H<sub>2</sub>-RAs in the prevention of NSAID-induced ulcers. Human studies showed that standard doses of H<sub>2</sub>-RAs significantly reduced the risk of NSAID-induced duodenal ulcers but were ineffective in preventing NSAID-induced gastric ulceration. However, high- or double-dose H<sub>2</sub>-RAs are effective against both NSAID-induced duodenal and gastric ulcers.

### **b) Proton pump inhibitors (PPI)**

PPIs inhibit acid secretion by blocking the proton pump within the gastric parietal cell. The PPI available for horses is omeprazole (gastrogard®, ulcergard®). Because an acidic environment promotes absorption of food-borne minerals, including iron, calcium, phosphorus, magnesium, and zinc, and is necessary for releasing vitamin B<sub>12</sub> from food,

mineral and vitamin deficiencies are possible but have not been reported in human or veterinary patients. PPIs should be administered 1 hour before a meal so that the peak serum concentration coincides with the maximal activity of proton pump secretion. In the first 24 hours of therapy with omeprazole in humans and dogs, gastric acid output is reduced by only approximately 30% because not all proton pumps can be inactivated with one dose. Up to 3 to 5 days are required before gastric acid production is nearly completely inhibited. Because of this delay in gastric acid suppression, H<sub>2</sub>-RAs can be used concurrently with PPIs for the first few days of treatment. PPIs has shown to provide superior control of intragastric pH over a 24-hour period compared with H<sub>2</sub>-RAs and effect greater relief of clinical signs and healing of mucosal damage. PPIs are most useful in treating diseases in which profound inhibition of acid secretion is necessary. In addition, PPIs are useful in controlling bleeding from GI ulcers.

The use of PPIs in healing and preventing NSAID-induced ulcers has also been widely evaluated in human studies. Both H<sub>2</sub>-RAs and PPIs are efficacious in healing NSAID-induced ulcers in humans when NSAID use is discontinued. H<sub>2</sub>-RAs are not effective when NSAID therapy is not discontinued, whereas PPIs are effective. PPIs are also superior to H<sub>2</sub>-RAs in preventing NSAID-induced ulcers in humans. Multiple equine studies show that omeprazole is effective both in healing and preventing gastric ulcers in horses.

### **c) Sucralfate**

Sucralfate is a sulfated disaccharide–aluminium hydroxide complex. After oral administration, sucralfate dissociates in the acid environment of the stomach into its primary components—aluminium hydroxide and sucrose octasulfate. The latter undergoes polymerization to form a viscous paste-like complex that binds in the base of ulcers or erosions for up to 6 hours. The cytoprotective effects of sucralfate are further augmented by its ability to stimulate formation of local mediators, such as prostaglandins (PGs) and growth factors, that protect the gastric mucosa. Sucralfate works best in an acidic environment; however, it is effective at acidic to near-neutral pH and can therefore be used concurrently with antisecretory drugs, such as H<sub>2</sub>-RAs or PPIs. However, no synergistic or additional therapeutic effect has been proven in animals or humans via coadministration of sucralfate with an antisecretory agent. Sucralfate is minimally absorbed after oral administration and thus is quite safe. The only known side effect of sucralfate is constipation secondary to aluminium hydroxide. Sucralfate may bind to and interfere with absorption of concurrently administered drugs. In general, sucralfate should not be administered within 2 hours of other medications. In humans, sucralfate has been shown to be more effective in treating duodenal ulcers than gastric ulcers. In a study of humans with NSAID-induced mucosal lesions, sucralfate was superior to a placebo, similar to ranitidine, but significantly less effective than omeprazole in ulcer healing. The use of sucralfate to prevent NSAID-induced ulceration in humans has been disappointing. Therefore, sucralfate cannot be recommended as a prophylactic agent to prevent NSAID-induced gastropathy.

#### **d) Misoprostol**

Misoprostol, a synthetic analogue of PGE<sub>1</sub>, is a gastric cytoprotective agent with both acid-inhibitory and mucosal-protective properties. Its cytoprotective effect is mediated by increasing bicarbonate secretion, mucus production and mucosal blood flow, which increases the oxygen and nutrient supply to the healing mucosa, ultimately increasing epithelialization. In addition, it causes a modest decrease in gastric acid secretion.

Because the serum half-life is less than 30 minutes, misoprostol must be given three to four times per day. Misoprostol may stimulate intestinal motility and secretion, leading to diarrhea, abdominal discomfort and nausea. Misoprostol also stimulates uterine contractions and therefore should not be used during pregnancy.

Misoprostol prevents NSAID-induced gastroduodenal ulcers but is not particularly effective in healing existing NSAID-induced ulcers. In most comparative studies in humans, healing rates associated with the use of H<sub>2</sub>-RAs, sucralfate and PPIs are higher than those associated with misoprostol use.

#### **e) Antacids**

Antacids have been used for centuries and were the mainstay treatment of acid-peptic disorders until the introduction of H<sub>2</sub>-RAs and PPIs. Antacids are weak bases that transiently neutralize gastric acid (HCl) in the gastric lumen. Although their principal mechanism of action is reduction of gastric acidity, they may also promote mucosal defense mechanisms by stimulating mucosal PG production, decreasing pepsin activity and binding to bile acids in the stomach.

Antacids vary in the cations and anions they contain, each with different buffering capacity and unique side effects. Common cations of antacids include aluminium, calcium, magnesium and sodium. Aluminium- or magnesium-containing antacids are the most efficacious. Antacids containing magnesium hydroxide or aluminium hydroxide neutralize hydrogen chloride, forming magnesium chloride or aluminium chloride and water. Aluminium-containing antacids have the added benefit of inactivating pepsin, binding bile acids, and inducing local PG synthesis. The most common side effects of aluminium- and magnesium-containing antacids are constipation and diarrhea, respectively. Therefore, these compounds are commonly administered together in proprietary formulations to minimize the impact on bowel function.

Sodium- and calcium-containing antacids, although efficacious, can cause more side effects than do other antacids. They react rapidly with HCl, producing carbon dioxide (CO<sub>2</sub>). CO<sub>2</sub> formation results in gastric distension and belching.

The use of antacids in veterinary medicine has significant limitations. Although antacids have been shown to be as effective as H<sub>2</sub>-RAs in reducing gastric acidity, their duration of action is only 2 to 3 hours. Therefore, they must be administered at least six times per day for maximum therapeutic benefit. Noncompliance with the frequent dosing regimen required with antacid use may result in increased total daily acid secretion or "acid rebound," which may exacerbate the initial condition. Acid rebound occurs because of loss of the normal inhibitory influence of acid pH on gastrin release. The

resultant hypergastrinemia can stimulate acid secretion when the antacid effects are gone. Hypergastrinemia also occurs with H<sub>2</sub>-RA and PPI use, but their longer duration of action leaves little time for hypergastrinemia to stimulate acid secretion. Because antacids increase intragastric pH, they may interfere with the rate of dissolution, absorption and bioavailability of concurrently administered drugs (H<sub>2</sub>-RAs) and iron supplements. In general, concurrent administration of antacids and other drugs should be avoided.

#### **f) Bismuth Subsalicylate**

Bismuth subsalicylate has several beneficial actions in the GI tract. In the stomach, it dissociates into bismuth and salicylate. Bismuth absorbs toxins and coats ulcers and erosions, creating a protective layer against acid and pepsin. It may also stimulate PG, mucus, and bicarbonate secretion. Bismuth has mild antimicrobial actions and binds toxins, accounting for much of its benefit in treating diarrhea. The salicylate component has an inhibitory effect on intestinal PGs and chloride secretion, thereby decreasing intestinal secretions and stool frequency.

Good products are:

- Omeprazole (Ulcergard®, Gastrogard®) is the only FDA approved treatment for equine ulcers. If a horse has active ulcers, it needs a whole tube of Gastrogard® (4mg/kg) once daily for 28 days. A quarter of a tube of Gastrogard® (1mg/kg) once daily will work as a prevention for horses that are stressed ( transportation, stall confinement with twice daily feeding, unfamiliar environment and exercise).
- Sucralfate (Carafate®) (20mg/kg orally three to four times daily)
- Antacids: 30 g AlOH / 15 g MgOH orally every 2 hrs. (U-gard solution™ (cortaflix.com), Neigh-lox™ ([www.kppusa.com](http://www.kppusa.com)), Eqlipse™ (combination of antacid and sucralfate ([www.bramespharmaceuticals.com](http://www.bramespharmaceuticals.com)))
- Ranitidine (Zantac®): 6.6mg/kg orally twice daily
- Misoprostol (Cytotec®): 3-5µg/kg orally every 8hrs

### **2.2. Improving starch digestibility and reducing hindgut acidosis**

The horse has a limited capacity to digest starch in the small intestine, because the activity of amylase is lower than in other monogastric animals and the increase in amylase secretion is limited even when high starch diets are fed. Without sufficient amylase in the intestinal tract, much of the starch in the diet passes through to the large intestine where it is fermented. This is undesirable for two reasons. First, the amount of energy produced from starch by fermentation is less than the amount produced by enzymatic means. Second, excessive fermentation of starch drops the pH of the hindgut (due to lactic acid production), which will decrease the efficiency of the bacteria that digest fiber and produce energy and even can cause microbial death, toxin production, anorexia, stereotypic behavior, endotoxemia, colic and laminitis.

There are four things we can do to improve starch digestibility and prevent hindgut acidosis:

1. **Changing the physical form of the food so that the contact of starch with pancreatic amylase is increased.** Starch is often entrapped in rigid cell walls that hinder swelling and dispersion of starch. A proper digestion of valuable plant cell content therefore requires the thorough crushing of cells by a good set of teeth and cereal processing will enhance digestibility. A greater level of destruction enhances solubility of starch molecules and absorption in the small intestine. The disruption of the structure of starch is more extensive with heat- and steam-processed grain than with ground grain and more extensive with ground grain than with rolled or whole grain. This is especially important for barley and corn and less for oats, as the digestibility is 90% for whole oats and only 30% for whole corn and 26% for whole barley.
2. **Feeding small amounts of concentrate (starch) per meal:** Values of around 2 g starch/kg bodyweight per meal (an average grain or sweet feed mix contains 40-50% starch, this means 2-2.5 kg concentrate per meal for a 500kg horse) have been suggested in the literature to be the maximum that should be fed in any one meal.
3. **Supplementing exogenous amylase:** Enzymatic degradation of starch can be increased by applying exogenous amylase. Supplementation with *Bacillus Licheniformis* alpha-amylase in combination with amyloglucosidase derived from *Aspergillus Niger* (AMG, another starch digesting enzyme found in the small intestine) improves starch digestion in the horse. It was found that supplementation with the alpha-amylase and AMG resulted in the most efficient starch digestion, followed by supplementation with only the alpha-amylase enzyme. The addition of AMG alone made no difference.

Good product that supply exogenous enzymes are:

- Equine-zyme™ or Equine-zyme Plus™ ([www.eartsongranch.com](http://www.eartsongranch.com)) which contain exogenous enzymes, *saccharomyces cerevisiae* and live bacteria.
- Equipride™ ([www.equipride.biz](http://www.equipride.biz)) which contains exogenous enzymes and yeast culture.

4. **Buffering the hindgut:** Changes in the pH of the hindgut due to alterations in the microbial populations and acid profiles cause a condition known as subclinical acidosis.

Subclinical acidosis (hindgut acidosis) is thought to result from overconsumption of either high-starch concentrates or pasture grasses rich in fructans. Microbial digestion of starch or fructans results in an increased production of volatile fatty acids (VFA) and lactic acid 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 a stronger acid than the 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, in particular fiber-digesting bacteria, that inhabit the hindgut and aid in digestion. For optimal performance, these bacteria favor an environment with a pH between 6.5 to 7.0. When pH drops to 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 VFA when exposed to acidic conditions, serving only to compound the problem. **EquiShure™** ([www.kerx.com](http://www.kerx.com)), a protected sodium bicarbonate supplemented to horses, significantly lowers lactate concentrations, suggesting that lactate was being used by lactic-acid-utilizing bacteria to produce VFA. The VFA are subsequently absorbed by the intestine and are metabolized as an energy source in the liver. These significant results suggest that EquiShure™ prevented the decrease in pH associated with rapid starch and fructan fermentation enabling lactate-utilizing bacteria to thrive and convert lactate into VFA. Some caution is warranted in trying to buffer the hindgut, because of the potential formation of enteroliths in an alkaline pH. Enteroliths (Struvite stones, magnesium ammonium phosphate ( $\text{NH}_3\text{MgPO}_4 \cdot 6\text{H}_2\text{O}$ )) are a very common cause of colic in California. Precipitation of struvite is generally attributed to  $\text{Mg}^{2+}$  supersaturation, presence of  $\text{NH}_4^+$  and  $\text{PO}_4^-$ , and an alkaline pH. These conditions, combined with the natural relative hypomotility within the right dorsal colon, likely contribute to the optimal environment for precipitation of struvite. Magnesium concentration and colonic pH in horses appear to be diet-dependent, with Mg-rich alfalfa hay being the most commonly implicated dietary factor. Despite its alkalinizing effects and high Mg content, alfalfa hay cannot be solely responsible for the formation of enteroliths. Other factors influencing intestinal pH or mineral content within the colon must also be considered, such as undetermined genetic factors, diet, bacterial flora, innate deficiencies, buffering capacity and pH of the water supply.

### **2.3. Improving hindgut microbial flora and fermentation.**

#### **2.3.1. Probiotic**

Probiotic has been defined as live microorganisms (bacteria or yeast) which when administered in adequate amounts confer a health benefit on the host. Today a large range of defined strains of probiotics belong to the group of lactic acid bacteria (*bifidobacterium*, *lactobacillus*, *streptococcus*, *enterococcus* and *lactococcus* sp.), *bacillus* sp., fungi (*aspergillus* sp.) and yeasts (*saccharomyces* sp., *kluveromyces* sp.). An effective probiotic organism must be resistant to destruction by gastric acid, pancreatic secretions, and bile salts, be able to colonize the intestinal tract, inhibit the growth of disease-causing (pathogenic) bacteria and improve fermentation in the hindgut. The amount of live microorganisms available in a product is expressed as colony forming units per gram (CFU/g). The effective dose is estimated to be 10 to 100 billion CFU/day of bacterial species or 45 billion CFU/day yeast.

Research is most supportive for yeast supplementation. One of the most significant effects of adding yeast to equine rations is the ability to help stabilize the hindgut (cecum and colon) bacterial populations and as a result, also increase nutrient digestibility. Components of the yeast cell wall (manno-oligosaccharides and beta-glucan) prevent the colonization of pathogenic microorganisms in the gut, improve the percentages of useful microorganisms in the intestinal tract, boost immune function and strengthen the structure of the gut wall. Yeast also stimulates the growth of bacteria that utilize lactic acid, which helps to lower lactic acid levels in the hindgut and increases the numbers of bacteria that are responsible for the fermentation of fiber.

Research studies also have shown that yeast improves the digestibility of phosphorus and magnesium and lowers ammonia concentration in the hindgut. This might be an indication that yeast supplementation can help in the prevention of enteroliths formation as the minerals that comprise most enteroliths are a combination of magnesium, phosphorus and ammonium (also called struvite). However, as mentioned before, enterolith formation is multifactorial, so while yeast supplementation may affect one parameter, other factors may be present in sufficient quantity to cause enterolith production. Some may also question the value of a product that has been shown to increase gut pH since current recommendations are to add vinegar and/or increase the amount of grain in the diet to help prevent enteroliths. This is because the above mentioned minerals tend to precipitate at alkaline pH values. However, further research discovered that in high fiber diets that tended to increase the alkalinity of the gastrointestinal tract, yeast supplemented horses had numerically (but not statistically) lower pH values. These data seem to indicate that moderating the pH of the intestinal tract is associated with yeast supplementation rather than providing alkalinity.

When we talk about yeast, we have to make a differentiation between yeast cultures and live-cell yeast products. True yeast cultures are complex fermented products containing the yeast (usually less than 10%) ("intracellular" yeast cell nutrients) and the metabolic by-products produced by the yeast during fermentation ("extracellular" metabolites of fermentation). Yeast cultures are not fed as a source of live or viable yeast cells, but as a nutrition supplement to provide undefined fermentation factors, which are recognized to stimulate bacterial growth in the digestive tract. These fermentation factors, sometimes referred to as "nutrilites", appear to be heat stable and are not significantly affected by high temperatures or pelleting. So yeast cultures are actually prebiotics and the minimum effective dosage appears to be 1% of the total daily feed by weight or +/-50g/day/500kg horse. Brewer's yeast however, is not a true yeast culture, it only contains de "intracellular" yeast cell nutrients.

Live-cell yeast products, on the other hand, consist of viable active dry yeast blended with a diluent to provide a specified number of live yeast cells. Active dry yeast is defined as pure dried yeast (without fillers or diluents) containing not less than 15 billion ( $1.5 \times 10^{10}$ ) live yeast cells per gram. Thus, a live-cell product claiming 5 billion cells per gram would consist of 20-25% active dry yeast, with the remainder being carrier ingredients like distillers, solubles or rice hulls. These definitions help differentiate active dry yeast and true yeast cultures; the first being a source of viable yeast cells and the second being a fermented culture.

Good products are:

- Equibios™ ([www.rrvp.com](http://www.rrvp.com)) contains brewer's dried yeast (*saccharomyces cerevisiae*) and *saccharomyces boulardii*
- Triad™ ([www.kerx.com](http://www.kerx.com)) contains a blend of yeast and yeast culture
- Florastor™ ([www.florastor.com](http://www.florastor.com)) contains *saccharomyces boulardii* 20 billion CFU/g => feed 5 tablets of 450mg daily)
- Live cell yeast 20X™ ([www.sbpequine.com](http://www.sbpequine.com)) contains *saccharomyces cerevisiae* 20 billion CFU/ounce. Feed a double dose (one ounce twice a day)
- Assure™ and Assure Plus™ ([www.arenus.com](http://www.arenus.com)) contains *saccharomyces cerevisiae*, live bacteria and psyllium. Feed 1 scoop assure in the morning and 2 scoops assure plus in the evening for a 500 kg horse.
- Equine-zyme™ or Equine-zyme Plus™ ([www.eartsongranch.com](http://www.eartsongranch.com)) contain exogenous enzymes, *saccharomyces cerevisiae* and live bacteria
- Equipride™ ([www.equipride.biz](http://www.equipride.biz)) contains exogenous enzymes and yeast culture

### 2.3.2. Prebiotic

Prebiotic has been defined as selectively fermented ingredients that beneficially affect the host by selectively stimulating the growth and/or activity of one or a limited number of bacteria in the colon, and thus improve host health. Therefore, prebiotics can enhance the effect of certain probiotics that contain beneficial bacteria such as bifidobacteria and *Lactobacillus*. A wider definition would include ingredients that are ultimately digested (due to the action of the target bacteria) and those that indirectly promote a healthy bacterial ecology.

Recent research has uncovered several new prebiotic ingredients that have great promise in the equine world. Some of these components act directly to feed beneficial bacteria and some act to inhibit the growth of pathogens. Others act indirectly by stimulating the immune system to help maintain the proper balance of hindgut flora and fauna. Here's a brief summary of these novel ingredients.

#### a) Fructo-oligosaccharide (FOS)

The fructans, inulin and fructo-oligosaccharides have received the most attention. Fructans are not digested in the small intestine but are fermented by bacteria in the large intestine.

In horses, supplementation of oligofructose (0.1kg/day) decreased the incidence of colic and increases the pH in the stomach, contributing to a decreased risk of stomach ulcers. This information should be taken cautiously, as fructans are rapidly fermented by the microflora of the hind gut. Excessive intakes of fructans (10kg/day) (the main dietary source for horses is grass) and rapid fermentation could lead to acidosis and laminitis.

#### b) Manno-oligosaccharides (MOS)

Manno-oligosaccharides (MOS), are generally derived from outer cell wall of *Saccharomyces cerevisiae*. MOS mimics the carbohydrates in the enterocyte membranes. Pathogens are fooled into binding with MOS instead of the enterocytes, and are subsequently flushed out of the digestive system. Along with the pathogens go the toxins they would have produced. In addition, MOS can be digested by the enzymes of certain beneficial bacteria (*Lactobacillus*, *Bifidobacterium*) and promote their growth. MOS also stimulates the immune system and encourages the growth of intestinal villi, showing improved digestion and absorption of nutrients in various animal studies.

### **c) Polar Lipids**

Lipids represent a large class of molecules that include fatty acids, phospholipids (lecithin), galactolipids and triglycerides. They play a key role in the structure and function of cellular membranes and are found in much of the plant material already in equine diets.

Oat oil is rich in polar lipids, particularly galactolipids. These dietary polar lipids are important in forming the tight junctions between the epithelial cells lining the gut. Cells connected in this fashion present a unified barrier against digestive juices, toxins and pathogens. Polar lipids are digested into galacto-oligosaccharides (GOS) which nourish the beneficial bacteria in the hindgut and discourage pathogenic species.

Lecithin has been researched in its use as an anti-ulcer supplement. Studies in horses showed that a pectin-lecithin complex can have a beneficial effect on the healing of gastric ulcers.

### **d) Beta-glucan**

Beta-glucan is a polysaccharide derived from yeast (dried brewer's yeast), barley and oats (oat bran) that has several profound effects on typical animal systems. Beta-glucan serves an important role here as the most potent known stimulator of the immune system. It arouses macrophages, which have a specific beta-glucan receptor, to mount a full-blown immune system response to pathogenic microbes and helping to heal damaged tissue. Beta-glucan also creates a gel, slowing the transit of digesta through the gut and allowing starches to be digested earlier in the system, thereby reducing the negative effects of starch in the hind gut, particularly due to the growth of pathogens. Effective dose is estimated to be between 500 mg and 3000 mg/day.

### **e) Glutamine**

Glutamine has been shown to have strong trophic effects on enterocytes, enhancing their normal function, as well as being necessary in normal turnover of healthy mucosa. In addition, glutamine functions as a source of energy for white blood cells and other immune cells.

### **f) Threonine**

Threonine is especially useful for wound healing and for treating stress, but it is also an essential link in the production of immunoglobulins, which help to control the balance of bacteria in the hindgut.

Good Prebiotic products are:

- Ration Plus™ ([www.rationplus.com](http://www.rationplus.com)) is a lactobacillus fermentation product
- Succeed™ ([www.succeeddcp.com](http://www.succeeddcp.com)) is a source of MOS, glutamine, threonine and oat oil. Feed a double dose (one ounce twice a day) for 30 days, then, after one month, you can continue with a maintenance dose.
- Equibios™ is a source of *Saccharomyces boulardii* and MOS.
- Kombat boots™ ([www.kombatboots.com](http://www.kombatboots.com)) is a dried brewer's yeast which serves as a source of MOS and beta-glucan (The cell wall of brewer's yeast (*Saccharomyces cerevisiae*) cells contains both MOS (25%) and B-(1, 3)-(1, 6)D-glucans (30%))
- Restore™ ([www.biovance.com](http://www.biovance.com)) is a combination of specific enzymes with amino acids, peptides and soluble nitrogen sources.

#### **2.4. Intestinal protectants and absorbents**

Intestinal protectants and absorbents are used to protect the mucosal layer of the intestines and prevent adsorption of toxins, viruses and bacteria. Products that come to mind are kaolin-pectin, activated charcoal and bismuth subsalicylate.

Although kaolin-pectin is claimed to act as a demulcent and adsorbent in the treatment of diarrhea (related to the binding of bacterial toxins in the intestinal tract), clinical studies have not demonstrated any benefit from its administration. Activated charcoal is very effective for adsorbing drugs and toxins, so it is a common nonspecific treatment for intoxications. Activated charcoal is not absorbed, so overdose is not a problem. Bismuth subsalicylate is considered by many human gastroenterologists to be the symptomatic treatment of choice for acute diarrhea and also seems superior in horses to treat diarrhea compared to kaolin-pectin and activated charcoal. Bismuth adsorbs bacterial toxins and has a gastro-intestinal protective effect. The salicylate component has antiprostaglandin activity. A newer and even more superior product is di-tri-octahedral smectite (Bio-Sponge™). Bio-Sponge™ has substantial capacity to adsorb toxins, viruses, bacteria and free radicals.

Good intestinal protectants and absorbents are:

- Bio-Sponge™ ([www.platinumperformance.com](http://www.platinumperformance.com))
- Bismu-Kote™ ([www.milburnequine.com](http://www.milburnequine.com)): Bismuth-subsalicylate can be given at 0.5-2ml/kg orally every 4-6hrs in horses with acute diarrhea.

### **3. When to consider medication or supplementation**

The use of one or more medications or supplements to support the intestinal tract should be considered for:

- Horses having trouble holding weight
- Older horses that need help with digestion and absorption
- Foal and young horse up to 1 year, as their diet is changing constantly and they are still establishing their intestinal microbial flora
- Horses with a history of intestinal difficulties, including diarrhea, gas, colic
- Horses with hay bellies
- Horses on high doses of antibiotics
- Horses on high starch diet
- Horses that show signs of bruxism, yawning or other behavioral vices

Please contact your veterinarian before trying any of these products mentioned above or when your horse shows any signs of gastro-intestinal disease. Your veterinarian can discuss the gastro-intestinal problem of your horse and guide you in using a product that will meet the needs of your horse best.

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