

# Fermaid®Ease187 - Equine Gastric Ulcer Syndrome



Tara Hembrooke, PhD, MS and Meri Stratton Phelps, DVM, MPVM, DACVIM (LAIM), DACVN

**Performance horses, pleasure horses, broodmares, weanlings and neonates all can develop gastric ulcers. The majority of these ulcers occur in the upper part of the stomach that contains the squamous mucosa.**

## Gastric Ulcers Defined

Gastric ulcers are a familiar problem to horse owners, as horses of all ages are susceptible to developing ulcers. Risk factors associated with the development of gastric squamous ulcers include exercise, fasting, transport, stabling as well as consumption of a low roughage and high grain ration.<sup>1</sup> The majority of these ulcers develop in the squamous mucosa, which is dorsal to the ridge of tissue that separates this region from the glandular region. Horses continually secrete hydrochloric acid produced in the glandular region of the stomach. The non-glandular squamous region of the stomach may have some protection from mucus glycoproteins (i.e., mucins),<sup>2</sup> however without cells that secrete protective substances including mucus and bicarbonate,<sup>3,4</sup> the squamous mucosa becomes damaged soon after exposure to hydrochloric acid. Although ulcers can occur in the glandular mucosa, this is far less common.

In horses, the parietal cells in the glandular region secrete hydrochloric acid throughout the day and night, which can promote the development of gastric ulcers in many breeds of horses and in horses that are athletes as well as those residing in pasture.<sup>1</sup> Other chemical substances that influence the development of ulcers include volatile fatty acids (VFA), lactic acid, and bile acids. Grains and other feeds that contain a high concentration of highly fermentable carbohydrates result in the production of VFA and lactic acid by the bacteria that inhabit the stomach.<sup>5</sup>

Horses with equine gastric ulcer syndrome (EGUS) may show signs of colic, may be reluctant to eat, and may have weight loss that is not explained by other health problems. The gold standard for the diagnosis of gastric ulcers is gastric video endoscopy, using a scoring system to classify the severity of the ulcers.

Ulcer prevention and treatment often includes changing the horse's husbandry to decrease stress and exercise intensity, decreasing the intake of grain and feeds high in non-structural carbohydrates, providing frequent but smaller meals throughout the day, and administering medications to decrease acid secretion and to protect the mucosa.

## An Innovative Nutritional Approach

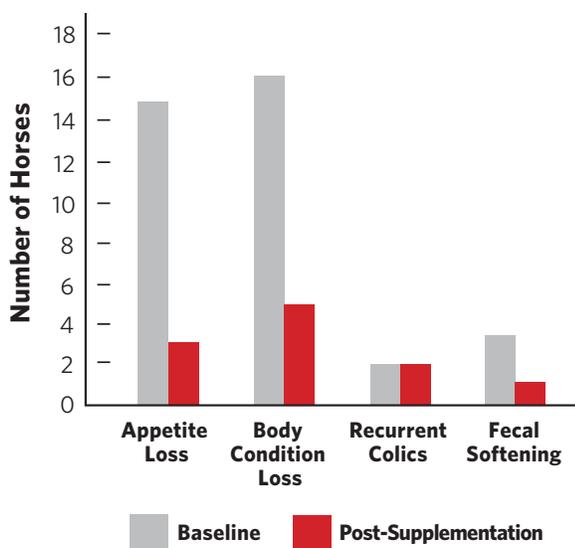
A new product recently introduced to the equine market takes advantage of the unique benefits provided by the bacterium *Lactobacillus delbrueckii* and its fermentation byproducts. Research in horses with clinical signs of mild to moderate gastric ulcers showed clinical improvements following supplementation with *Lactobacillus delbrueckii* and its fermentation byproducts (Figure 1).<sup>6</sup> The bacteria and fermentation products are reported to have beneficial effects on the gastric environment in the following three ways:

- 1. Anti-inflammatory-** *Lactobacillus delbrueckii* helps reduce inflammatory mediators that can play a role in the formation and exacerbation of gastric ulcers. Analysis of cytokine mRNA taken from gastric biopsies in horses with EGUS showed a significant increase in TNF- $\alpha$  compared to horses with normal gastric histology.<sup>7</sup> Reductions in the pro-inflammatory proteins IL-8, TNF- $\alpha$ , and RANTES have been reported following *in vitro* culturing of intestinal cells with *Lactobacillus delbrueckii*.<sup>8</sup> Upregulation of IL-6, an antibody-stimulating cytokine, has been reported in similar *in vitro* cultures.<sup>9</sup>
- 2. Anti-proteolytic-** Fermentation of soy by *Lactobacillus delbrueckii* results in the presence of several trypsin inhibitors, including Bowman-Birk,

Trypsin Inhibitor subtype A, and Kunitz Trypsin Inhibitor. Trypsin inhibitors may help prevent gastric ulcers by protecting the mucosa from the degradative actions of trypsin. Trypsin inhibitors have resulted in improvements in reflux esophagitis and peptic ulcers following oral dosing in humans.<sup>10,11</sup>

**3. Anti-acidic-** *Lactobacillus delbrueckii* can reduce stomach acidity, as noted by one trial that demonstrated transient increases in stomach pH following administration in humans.<sup>12</sup> Anti-acidic benefits may result from a direct buffering effect by the fermentation byproducts, competitive inhibition of histamine activity, and an increase in bicarbonate secretion.

Figure 1. **Clinical signs associated with gastric ulcers in 17 horses before and after 1 month of supplementation with *Lactobacillus delbrueckii* and its fermentation byproducts<sup>6</sup>**



### Effects of Supplementation

In an additional study, a gastric ulcer support product\* containing *Lactobacillus delbrueckii* and its fermentation products was administered twice daily to five Thoroughbred horses (4-10 years of age) with gastric

\*Fermaid®Ease 187

ulcers induced using an established EGUS model.<sup>13</sup> Four horses in the same study served as untreated controls. All horses were maintained on an oat hay diet, housed in individual stalls, and walked 20-30 minutes 4 days each week. The severity of gastric ulceration was determined by a veterinarian blinded to the treatment grouping using the MacAllister Gastric Ulcer Scoring System<sup>14</sup> with photographs obtained through a gastroscope. At baseline, the supplemented group had an average ulcer score of 2.8 (+/- 0.2), and the control group had an average score of 3.0 (+/- 0.0). Within four days, the average ulcer score in the supplemented group decreased by 36% to 1.8 (+/- 0.2), while the control group maintained a similar average ulcer score of 2.8 (+/-0.25).

### Literature Cited

1. Sykes B. Disorders of the stomach, 669-674. In Smith BP (ed), Large animal internal medicine, ed 5. Elsevier Mosby, St. Louis, MO.
2. Bullimore S, Corfield A, Hicks S, et al. Surface mucus in the non-glandular region of the equine stomach. *Res Vet Sci* 2001;70:149-155.
3. Merritt A. Normal equine gastroduodenal secretion and motility. *Equine Vet J Suppl* 1999:7-13.
4. Murray M, Mahaffey E. Age-related characteristics of gastric squamous epithelial mucosa in foals. *Equine Vet J* 1993;25:514-517.
5. Reese R, Andrews F. Nutrition and dietary management of equine gastric ulcer syndrome. *Vet Clin North Am Equine Pract* 2009;25:79-92.
6. Sacy A, Le Treut Y, Benoit P. Gastric ulcers in adult horses: current situation, prevention and improvements observed with the use of fermented soya (Fermaid®Ease 187). 37th Equine Research Day 2011.
7. Pietra M, Morini M, Perfetti G et al. Comparison of endoscopy, histology, and cytokine mRNA of the equine gastric mucosa. *Vet Res Commun* 2010;34:Suppl 1:S121-4.
8. Wallace T, Bradley S, Buckley N, et al. Interactions of lactic acid bacteria with human intestinal epithelial cells: Effects on cytokine production. *J of Food Protection* 2003;66:466-472.
9. Wallace T, Tompkins T, Green-Johnson J. Effects of lactic acid bacteria on cytokine production by a human intestinal epithelial cell line: Institut Rosell - Lallemand Inc, Human Nutrition, 2000.
10. Kono K, Takahashi A, Sugai H, et al. Oral trypsin inhibitor can improve reflux esophagitis after distal gastrectomy concomitant with decreased trypsin activity. *Am J Surg* 2005;190:412-417.
11. Moriya S, Omura N, Kashiwagi H, et al. Treatment of peptic ulcer and reflux esophagitis with enzyme inhibitors [Article in Japanese]. *Nippon Rinsho* 1991;49:2115-2119.
12. Litinskaia E. Experience with the therapeutic use of Gastropharm in peptic ulcer. *Vrach Delo* 1982:70-73.
13. Murray M. Equine model of inducing ulceration in alimentary squamous epithelial mucosa. *Digestive Diseases and Sciences* 1994;39:2530-2535.
14. MacAllister C, Andrews F, Deegan E, et al. A scoring system for gastric ulcers in the horse. *Equine Vet J* 1997;29:430-433.

