J Vet Intern Med 2012;26:1186–1191

Effect of Sea Buckthorn Berries and Pulp in a Liquid Emulsion on Gastric Ulcer Scores and Gastric Juice pH in Horses

N.K. Huff, A.D. Auer, F. Garza Jr., M.L. Keowen, M.T. Kearney, R.B. McMullin, and F.M. Andrews

Background: Sea buckthorn berries (*Hippophae rhamnoides*) are rich in vitamin C and E, carotenoids, flavonoids, fatty acids, plant sterols, lignans, and minerals. A feed supplement containing sea buckthorn berries might have efficacy in treatment and prevention of gastric ulcers in horses.

Objectives: To test the efficacy of a commercially available formulation of sea buckthorn berries and pulp (SeaBuck SBT Gastro-Plus) for treatment and prevention of gastric ulcers in stall-confined horses.

Animals: Eight Thoroughbred and Thoroughbred-cross horses (3–10 years of age, 5 geldings and 3 mares, 380–600 kg body weight).

Methods: This study was a 2-period crossover in which all horses received no treatment (untreated controls; n = 8) and treatment (SeaBuckSBT Gastro-Plus, 4 ounces [35.6 g berries and pulp], twice daily; n = 8) mixed with a pelleted complete feed (18% crude fiber; 9% starch; 14% crude protein). Horses were treated for 4 weeks followed by a 1-week (d28–d35) alternating feed-deprivation period to induce or worsen existing ulcers. Gastroscopic examinations were performed on days 0, 28, and 35. Gastric juice pH was measured and gastric ulcer number and severity scores were assigned by a masked investigator.

Results: Mean nonglandular gastric ulcer scores significantly ($P < .05$) increased in all horses after day 28, as a result of intermittent feed deprivation. Mean nonglandular gastric ulcer number ($P = .84$) and severity ($P = .51$) were not significantly different between SBT-treated and untreated control horses. However, mean glandular ulcer number ($P = .02$) and glandular ulcer severity ($P = .02$) were significantly lower in the SBT-treated horses compared with the untreated control at week 5.

Conclusions and Clinical Importance: SeaBuck SBT Gastro-Plus liquid fed to horses did not show efficacy in treatment or prevention of naturally occurring nonglandular ulcers in horses; however, glandular ulcer scores were significantly lower in SBT-treated horses after feed deprivation. Thus, SBT might have efficacy in prevention of glandular ulcers in horses housed in stalls and undergoing intermittent feeding.

Key words: Gastric ulcers; *Hippophae rhamnoides*; Horse; Sea buckthorn berry.

Equine gastric ulcer syndrome (EGUS) continues to be a conundrum for horse owners, trainers, and veterinarians.[1] The high prevalence of gastric ulcers in horses, vague clinical signs, and negative effect on performance make it a significant clinical and economic problem within the horse industry. Current approved pharmaceutical agents used to treat gastric ulcer disease in horses are effective, but are labeled for treatment for 28 days, and increase gastric juice pH.[2] Furthermore, these agents frequently have to be given continuously during the training and racing season to prevent ulcers from recurring or worsening. Also, preventative measures, such as increased pasture turnout, ad libitum hay feeding, reduced training levels, and stress reduction coupled with treatment, are not always possible or effective. Discovery of less expensive and easier to administer alternatives (ie, feed additives) to treat or prevent gastric ulcers would be desirable.

Abbreviatons:

EGUS	equine gastric ulcer syndrome
GN	glandular number
GS	glandular severity
NGN	nonglandular ulcer number
NGS	nonglandular ulcer severity
PCV	packed cell volume
SBT	SeaBuck SBT Gastro-Plus
TPP	total plasma protein

Recently, there has been an increased interest in the use of botanicals (herbs and berries) because of their potential therapeutic properties and efficacy in relieving disease states. Berries and pulp from the sea buckthorn plant (*Hippophae rhamnoides*) are rich in nutrients and compounds such as vitamins, carotene, flavonoids, essential oil, carbohydrates, organic acids, amino acids, and minerals.[3,4] Sea buckthorn berries have been used successfully to treat gastric and duodenal ulcers in people and rats.[5–7] A commercially available product containing sea buckthorn berries and pulp was recently introduced as a feed additive to promote gastrointestinal health in horses and may be efficacious in the treatment or prevention of gastric ulcers. The purpose of this study was to determine the

From the Department of Veterinary Clinical Sciences, School of Veterinary Medicine, Equine Health Studies Program, Louisiana State University, Baton Rouge, LA (Huff, Garza, Keowen, Andrews); Purdue University School of Veterinary Medicine, West Lafayette, IN (Auer); and the Department of Pathobiological Sciences, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA (Kearney); and SeaBuck Equine, LLC, Midvale, UT (McMullin).

Corresponding author: Frank M. Andrews, LVMA Equine Committee Professor and Director, Equine Health Studies Program, Department of Veterinary Clinical Sciences, Louisiana State University, School of Veterinary Medicine, Skip Bertman Drive, Baton Rouge, LA 70803; e-mail: fandrews@lsu.edu

Submitted October 9, 2011; Revised April 28, 2012; Accepted June 15, 2012.

10.1111/j.1939-1676.2012.00975.x

efficacy of a feed supplement containing sea buckthorn berries and pulp in decreasing nonglandular and glandular gastric ulcer scores in stall-confined horses undergoing intermittent feeding. We hypothesize that SBT treatment will significantly decrease spontaneously occurring and intermittent feed-deprivation induced nonglandular and glandular gastric ulcers in horses without altering gastric juice pH.

Materials and Methods

All procedures performed on the horses were approved for ethical use of animals by the Louisiana State University Institutional Animal Care and Use Committee (LSU Protocol #10-008).

All horses used in this study were Thoroughbred or Thoroughbred-cross breeds (5 geldings and 3 mares, 3–10 years of age, weighing 380–600 kg body weight) from the resident herd at Louisiana State University School of Veterinary Medicine, Equine Health Studies Program. This study was performed in the summer, June through August, when horses were routinely maintained on native grass pastures and supplemented with native round-bale grass hay. Before the beginning of the study, a full physical examination was performed on all horses to exclude presence of clinical disease. Blood samples were submitted for CBC analysis and plasma biochemical panel 48 hours before initiation of the study. All horses were vaccinated in the spring for Eastern Equine Encephalitis, Western Equine Encephalitis, West Nile Virus Encephalitis, Tetanus, and Influenza and dewormed twice yearly with ivermectin (200 μg/kg body weight) as part of the routine herd care. Horses were stratified by nonglandular gastric ulcer number score (NGN) and then randomly assigned by a coin-flip to 2 groups: control (untreated) and SBT supplement (treatment).

Experimental Design

The experiment was performed as a 2-period, control and SBT-treatment crossover design. Horses were housed in stalls and fed a complete pelleted feed ([a]; minimum guaranteed analysis: crude protein: 14%; crude fiber: 18%; starch: 9%; digestible energy: 1225 cal/g; dry matter [DM]: 90%) at 1.35 kg DM (1.5 kg as fed)/100 kg body weight divided into 3 equal feedings per day, without hay. Horses served as their own controls. The treatment period was 5 weeks (35 days) in duration, with the last week (Week 5) consisting of an intermittent feed-deprivation period. The day before the beginning of each study period (Day-1) horses were put in covered nonenvironmentally controlled stalls. Horses in the treatment group received SBT liquid concentrate (4 ounces, 35.6 g berries and pulp, twice daily; SBT[b];) per product label mixed into the complete pelleted feed grain ration for 5 weeks. At the end of 4 weeks, horses were subjected to a modified intermittent feed deprivation protocol to cause ulcers or worsen existing ulcers.[8] Briefly, during this period, horses were muzzled and deprived of feed for 24 hours, then fed their normal ration for 24 hours until a total of 96 hours of cumulative feed deprivation was achieved. During the intermittent feed-deprivation period, horses in the SBT-treatment group continued to receive the daily dose of SBT, mixed with a small amount of complete feed (50 g/100 kg of body weight), whereas the control horses received the same amount of complete feed with no addition of SBT. Horses had free access to water at all times. Between treatment periods, horses were turned out into a pasture for 2 weeks to wash out any residual effects of SBT.

Gastroscopic examinations were performed on all horses on Day 0, at the end of week 4 (Day 28), and after the feed-deprivation period, week 5 (Day 35). Endoscopy of the stomach was performed using a 3-m endoscope.[c] To improve visualization of the stomach, feed was withheld for 16–18 hours and water for 3–4 hours before gastroscopy. Horses were sedated with xylazine (0.4 mg/kg, IV[d]) before gastroscopic examination. To enable observation of the nonglandular squamous mucosa (fundus ventriculi), margo plicatus, and glandular mucosa (corpus ventriculi), the stomach was insufflated with air using an air compressor attached to the endoscope biopsy chamber (until the rugae or stomach folds were absent). Mucosa was rinsed of adherent food material and mucus with tap water flushed through the endoscope biopsy channel. Each horse's stomach, a number and severity score, nonglandular number (NGN), nonglandular ulcer severity (NGS), glandular number (GN), and glandular severity (GS), was assigned for each entire region of the stomach, based on a validated equine scoring system (Table 1).[9] Scores were assigned by one of the authors (FMA), masked to the SBT treatment. In addition, hyperkeratosis (yellow color and thickening), hyperemia (mucosal reddening), and desquamation (generalized "peeling" or shedding of the mucosa) were recorded.

Gastric fluid was aspirated before insufflation or addition of water to the stomach during gastroscopy and pH was measured using a handheld pH meter.[e]

All horses were weighed weekly before the morning feeding using a digital electronic scale housed in the Veterinary Teaching Hospital & Clinic at Louisiana State University. Packed cell volume (PCV) and total protein (TP) were also measured weekly before the morning feeding.

Table 1. Validated equine nonglandular and glandular ulcer scoring system used to score gastric ulcers in the study reported here.[10]

Lesion Number Score	Descriptions
0	No lesions
1	1–2 localized lesions
2	3–5 localized lesions
3	6–10 lesions
4	10 or more lesions or diffuse (very large) lesions

Lesion Severity Score	Descriptions
0	No lesions
1	Appears superficial (only mucosa missing)
2	Deeper structures involved (>depth than Number 1)
3	Multiple lesions and variable severity (1,2, and/or 4)
4	Deeper structure involved (>depth than Number 1) and has active appearance (hyperemic and/or darkened lesion crater)
5	Same as Number 4 plus hemorrhage or adherent blood clot

Scores reflect the entire area of each region of the stomach and each horse's stomach received 4 scores: nonglandular lesion number (NGN), nonglandular lesion severity (NGS), glandular lesion number (GN), and glandular lesion severity (GS).

Statistical Analyses

Data were pooled from both study periods and means and SD were calculated on all variables measured. A crossover design with 8 horses over 2 time periods was used to determine the effect of the SBT treatment on NGN, NGS, GN, and GS, and gastric juice pH. A repeated measures analysis of variance (ANOVA) of a mixed effects model was conducted with a MIXED procedure[f] for the ordered-metric data. When significant differences ($P < .05$) were observed, posthoc pair-wise comparisons of main effects were conducted using Tukey's test. For interaction effects, posthoc comparisons were made with pair-wise *t*-tests of least-squares means. With respect to categorical data, hyperemia, hyperkeratosis, and desquamation, a repeated measures analysis was conducted with the General Models procedure in SAS to fit Generalized Estimating Equations (GEE) models with binary logit link functions. Comparisons weeks back to baseline values were made with sets of orthogonal contrasts. Significance was set at $P < .05$.

Results

Physical examinations were within normal limits and there was no evidence of clinical disease in any of the horses before enrollment in the study. The CBC and plasma biochemical values were unremarkable. Feed quantities were adjusted so that body weight did not change significantly during the trial.

SeaBuck SBT Gastro-Plus mixed with complete feed and fed twice daily was readily consumed by all horses. One SBT-treated horse developed diarrhea of 3 days duration and 1 untreated control horse showed signs of colic during the 1st period of the study. Clinical signs resolved without treatment in both horses. Also, 1 horse experienced tibio-tarsal joint effusion and severe (grade 4/5) lameness attributable to preexisting chronic osteoarthritis. Flunixin meglumine (1.1 mg/kg, IV, q24h, 3 days) was administered during week 3 of the study and the horse was turned out in a paddock for 10 minutes daily. Its lameness improved, hence treatment was discontinued. Although flunixin meglumine was used to treat this horse, the authors feel that the treatment did not interfere with the results of the study as ulcers in this horse did not worsen after the treatment period.

Gastric Ulcer Scores

Mean ± SD for NGN, NGS, GN, and GS scores are listed in Table 2. Before treatment, 2 horses in the control group and 3 horses in the SBT-treated group did not have gastric ulcers. On day 0, gastric ulcer scores were not significantly different between SBT-treated and untreated controls. After 4 weeks of treatment, mean ± SD gastric ulcer scores did not differ significantly from ulcer scores on day 0 or between SBT-treated horses and untreated controls. After the feed-deprivation period on week 5, NGN and NGS scores increased significantly ($P = .03$) in SBT-treated and untreated control horses on week 5 compared to day 0 and week 4 (Table 2). There was no significant difference in the mean NGN ($P = .84$) and mean NGS ($P = .51$) scores in SBT-treated horses compared to untreated controls.

Table 2. Mean ± SD gastric ulcer scores in untreated controls (n = 8) and SeaBuck Gastro-Plus (SBT)-treated (n = 8) horses on day 0 (before treatment), week 4 (after stall confinement), and week 5 (after 7 days of intermittent feed deprivation) in a 2-period crossover design.

	NGN	NGS	GN	GS
Score – Control				
d-0	1.75 ± 1.39	1.75 ± 1.58	0	0
d-28	1.50 ± 1.69	1.75 ± 1.67	0.50 ± 1.41	0.38 ± 1.06
d-35	3.13 ± 1.64[b]	2.50 ± 1.69[b]	1.25 ± 1.83[a]	0.88 ± 0.99[a]
Score – Treatment				
d-0	1.75 ± 1.67	1.25 ± 1.16	0.13 ± 0.35	0.13 ± 0.35
d-28	1.75 ± 1.04	1.88 ± 1.25	0	0
d-35	2.63 ± 1.51[b]	2.00 ± 1.51[b]	0	0

NGN = nonglandular number score; NGS = nonglandular severity score, GN = glandular number; GS = glandular severity.

[a]denotes a significant ($P < .05$) difference between untreated controls and SBT-treated horses for the same week.

[b]denotes significant ($P < .05$) differences from day 0 and week 4 scores.

On the other hand, mean GN ($P = .02$) and mean GS ($P = .02$) were significantly lower in the SBT-treated horses compared to untreated controls at the end of week 5 (Table 2).

Hyperkeratosis, Hyperemia, and Desquamation

Hyperkeratosis of the nonglandular mucosa was observed in 30/48 gastroscopic examinations during the study. The SBT-treated horses were 4.85 times ($P = .01$) more likely to develop hyperkeratosis than untreated control horses. Also, hyperkeratosis was observed more often ($P = .01$) in horses after the feed-deprivation period, compared to the day 0 and the end of week 4. Hyperemia of the glandular mucosa and desquamation (peeling of the mucosa) nonglandular mucosa occurred in 16.1 and 14.3% of horses, respectively. However, there was no significant difference in hyperemia ($P = .38$) and desquamation ($P = .41$) in SBT-treated horses compared to untreated controls.

Gastric Juice pH

Gastric juice pH was low and varied from 1.68 to 6.24 during the study. On day 0, mean ± SD gastric juice pH was 3.76 ± 2.02 (range: 1.70–6.24) in untreated controls and 2.79 ± 0.83 (range: 1.68–3.01) in the SBT-treated group. At the end of week 4, mean ± SD gastric juice pH was 3.42 ± 1.10 (range: 2.11–5.41) in untreated controls and 3.43 ± 0.99 (range: 2.69–5.83) in SBT-treated horses. After week 5, mean ± SD gastric juice pH was 3.97 ± 1.05 (range: 2.18–4.73) in untreated controls and 3.01 ± 1.17 (range: 1.87–5.68) in SBT-treated horses. There was no significant difference in gastric juice pH ($P = .06$)

between the SBT-treated and untreated control horses at any time during the study period.

Total Plasma Protein (TPP) and Packed Cell Volume (PCV)

Total plasma protein (TPP) values were within reference range (5.2–7.9 g/dL) in all of the horses in this study. There was no significant difference between SBT-treated and control horses ($P = .14$) at any time point during the study. However, there was a significant decrease in TP ($P < .01$) in both groups on week 4, compared to day 0 and week 5. Also, PCV did not differ between SBT-treated and control horses ($P = .63$) at any week during the study, however, PCV was decreased significantly ($P = .03$) in horses during week 4 of the study. PCV values were never below the reference range (32–48%) for our laboratory.

Discussion

The intermittent feed deprivation model was successful in inducing and making existing gastric ulcers more severe, as mean nonglandular and glandular gastric ulcer scores significantly ($P = .03$) increased in both groups of horses. SeaBuck Gastro-Plus containing sea buckthorn berries and pulp added to a complete feed ration resulted in preventing glandular ulcer formation after intermittent feed deprivation, without a significant increase in gastric juice pH. Glandular ulcer number and severity was significantly lower in the SBT-treated horses. However, this product did not have a significant effect on nonglandular ulcer number or severity.

Sea buckthorn berries and pulp are rich in lipophilic and hydrophilic bioactive compounds such as vitamins C and E, carotenoids, flavonoids, fatty acids, plant sterols, lignans, and minerals.[4,10] These compounds have antioxidant and immunomodulary properties, which are important in mucosal healing.[5] Numerous in vitro and in vivo studies demonstrate that the gastroprotective effects of seabuckthorn berries are mainly explained by its strong antioxidant effects, which is likely caused by high concentrations of flavonols, vitamin C, and vitamin E.[6] Furthermore, a recent study in rats undergoing a cold-stress model for inducting gastric ulcers showed an increase in gastric mucosal lipid peroxidation and superoxide dismutase. The results of that study indicated a positive correlation between free-radical-induced oxidative-stress and gastric ulcers.[11] Thus, sea buckthorn berries might act to attenuate oxidative stress that may initiate and aggravate glandular gastric ulcers in horses, which has been shown in the cardiovascular system of rats; whereas sea buckthorn berries have been shown to attenuate cardiac dysfunction and oxidative stress in isoproterenol-induced cardiotoxicity in rats.[12]

Procyanidins in sea buckthorn berries have been shown to accelerate mucosal repair and healing of acetic acid-induced gastric ulcers in rats.[5] This recent study showed that a procyanidin-rich extract from sea buckthorn berries increased expression of epidermal growth factor receptor (EGFR) and proliferating cell nuclear antigen (PCNA) in acetic acid-induced gastric ulcers in rats. In a study in horses undergoing intermittent feed deprivation, epithelial growth factor receptor was isolated from the stomach and receptor area and density increased around and adjacent to ulcers in the nonglandular mucosa.[13] It is possible that SBT treatment increased expression of EGFR and PCNA in the nonglandular and glandular mucosa of the horses in the study reported here and facilitated epithelialization of the glandular mucosa. It is likely that the procyanidins exert their effect locally as they are not hydrolyzed in the stomach and are poorly absorbed throughout the gastrointestinal tract. It must be emphasized that EGFR and PCNA expressions were not measured in horses in the study reported here.

Recent research suggests that an increase in toxic oxidant parameters can induce gastric damage.[14–16] In light of the potent antioxidant effects of SBT, there was lack of its effect on nonglandular mucosal healing in the study reported here. However, a recent study showed similar results when an equine supplement containing sea buckthorn berries[g] was fed to horses.[17] In that study, the sea buckthorn berry supplement was fed for 67 days, compared with 35 days in the study reported here. In that study, gastric ulcer severity scores stayed the same or decreased in 7/8 of the horses after the intermittent feed-deprivation period, compared with only 2/8 in the untreated controls. In the study reported here, gastric ulcer severity scores decreased or stayed the same in only 4/8 SBT-treated horses and was similar in control horses. The difference between the 2 studies may be related to the duration of feeding of the supplement. Horses may need to be fed this sea buckthorn berry supplement for a longer (>60 days) period to observe an effect on nonglandular gastric ulcer scores. Furthermore, a higher dose may be needed to effect ulcer healing, as procyanidin has been shown to increase expression of EGFR in a dose-dependent manner in rats.[5]

Another reason SBT treatment was not effective in improving nonglandular ulcer scores in the horses in this study might have been caused by a dose-response effect. Horses in this study varied in weight from 380 to 600 kg body weight. All horses were fed the same amount of SBT supplement (35.6 g berries and pulp/4 oz, twice daily); therefore, dose ranged from approximately 60 to 90 mg/kg body weight. If that were the case, one would expect the lighter horses to have less severe gastric ulcer scores when compared with the heavier horses, but that seems less likely because body weight did not correlate with ulcer scores in this study. However, a higher dose, more frequent administration, or administration for a longer time could have made the SBT supplement used in this study more effective.

One additional reason that SBT treatment was not effective in improving nonglandular ulcer scores in the horses in this study may have been caused by pelleted diet fed to the horses. Horses in this study were fed a pelleted complete feed diet, whereas horses in the

previous study were fed a grass hay and grain diet.[17] Recent research suggests that feeding alfalfa hay may buffer gastric acid production, thus reducing the severity of gastric squamous mucosal ulceration.[18,19] Typically, horses on a hay diet spend more time chewing and thus produce saliva that is rich in bicarbonate. Increased salivary bicarbonate buffers stomach contents and may reduce acid exposure.[20] Furthermore, horses typically eat pelleted diets faster and because of smaller particle size, gastric emptying is shortened, leaving longer periods when the stomach is empty and the mucosa is exposed to acids. Diets with reduced amounts of hay have been shown to produce gastric ulcers.[21] One additional consideration is that horses on a pelleted diet produce less saliva, which would result in less dilution and the delivery of a more concentrated SBT supplement and bioactive compounds to the stomach. However, it is difficult to predict the in vivo effects of these bioactive compounds based on concentration within the supplement, attributable to the volume of saliva production, amount hydrolyzed by bacterial in the stomach and gastric emptying.[22,23]

Hyperkeratosis (thickening and yellow appearance) of the nonglandular mucosa was more frequently seen in the SBT-treated horses when compared to the control horses. However, after the intermittent feed-deprivation period, all SBT-treated and 7/8 untreated control horses had hyperkeratosis in the nonglandular mucosa. Hyperkeratosis was reported in a previous study after horses were intermittently fed.[21] Acid exposure to the nonglandular squamous mucosa is thought to stimulate the cornified layer to thicken and change color. However, SBT-treated horses had significantly more hyperkeratosis than control horses on week 4. The reason for the increased incidence of hyperkeratosis in the SBT-treated horses remains unknown, but perhaps the phytochemicals, like procyanidins, and especially ß-carotene in the SBT, stimulate proliferation of the nonglandular mucosa or incorporation into the mucosa during proliferation as the mucosa imparting the yellow appearance to the mucosa. Also, procyanidins have been shown to stimulate EGFR and PCNA in rats.[5] Hyperkeratosis may be an adaptive mechanism to protect the stomach from the damaging effects of acid or to facilitate ulcer healing once an ulcer is formed.

Hyperemia in the glandular mucosa and desquamation in the nonglandular mucosa were observed in 16.1 and 14.3% of the gastroscopies performed, respectively. Hyperemia was observed around the pyloric opening and may be caused by irritation from hydrochloric acid from the stomach or reflux of bile acids from the proximal duodenum.

Desquamation, on the other hand, is a process of mucosal shedding. The reason for the presence of desquamation in the horses in this study remains unknown, but may represent normal mucosal turnover as a result of exposure to gastric acids and abrasive feed materials.

Packed cell volume and TPP were not significantly ($P = .63$, $P = .14$, respectively) different in SBT-treated horses when compared with untreated controls. However, mean PCV and TPP was decreased in horses on week 4 of the study presented here, when compared to day 0 and week 5 values. A previous report suggested that lower RBC counts and hemoglobin concentrations might be attributable to gastric ulceration and blood loss from those ulcers.[24] However, the PCV and TPP increased from week 4 to week 5 in both SBT-treated and untreated control horses. The increase in PCV and TPP by week 5 may have been caused by stress from feed deprivation and associated splenic contraction or dehydration.

Gastric juice pH was variable but not significantly ($P = .055$) different in SBT-treated horses when compared to untreated controls. A single gastric juice sample can show a variable pH and may not be as accurate as a continuous monitoring with an in situ 24-hour pH probe. All samples were taken before distending the stomach in fasted horses. Gastric juice pH in these fasted horses was consistent with previous pH values in fasted horses.[21] This feed supplement containing sea buckthorn berries and pulp did not increase gastric juice pH, as is seen with the use of proton pump inhibitors or histamine type-2 blockers. Increased pH in the stomach may affect digestion of feed material, enzyme conversion, or allow overgrowth of aberrant flora, or passage of flora normally killed by gastric acid into the small intestine.

The results reported here confirm that stall-confined horses undergoing intermittent feed deprivation have a significant increase in number and severity of glandular and nonglandular gastric ulcers. SeaBuck Gastro-Plus supplement prevented ulcers from worsening in the glandular mucosa in horses intermittently fed, when compared with untreated controls, where ulcer severity increased significantly. However, Seabuck Gastro-Plus treatment had no effect on treatment or prevention of nonglandular gastric ulcers in these horses.

Conflict of Interest Declaration

Authors disclose no conflict of interest.

Footnotes

[a] Equine Senior, Purina Mills, Gray Summit, MO
[b] SeaBuck SBT Gastro-Plus, SeaBuck Equine, LLC, Midvale, UT
[c] Karl Storz, El Segundo, CA
[d] AnaSed, Lloyd, Shenandoah, IA
[e] Chek-mite, pH-20, NOVA Analytics, Woburn, MA
[f] SAS Inst. Inc., Cary, NC
[g] SeaBuck Complete; SeaBuck Equine

Acknowledgment

The sea buckthorn berry supplement (SeaBuck SBT Gastro Plus) used in this study was supplied by SeaBuck Equine, LLC, Midvale, UT.

References

1. Nadeau JA, Andrews FM. Equine gastric ulcer syndrome: The continuing conundrum. Equine Vet J 2009;41:611–615.

2. Andrews FM, Sifferman R, Bernard W. Efficacy of omeprazole paste in the treatment and prevention of gastric ulcers in horses. Equine Vet J 1999;29(Suppl):81–86.

3. Sabir SM, Maqsood H, Hayat I, et al. Elemental and nutritional analysis of seabuckthorn (*Hippophae rhamnoides* ssp. turkestanica) berries of Pakistani origin. J Med Food 2005;8:518–522.

4. Yang BR, Kallio H. Composition and physiological effects of sea buckthorn (Hippophae). Trends Food Sci Technol 2002;13:160–167.

5. Xu X, Xie B, Pan S, et al. Effects of sea buckthorn procyanidins on healing of acetic acid-induced lesions in rat stomach. Asia Pac J Clin Nutri 2007;16(Suppl 1):234–238.

6. Geetha S, Ram MS, Singh V, et al. Anti-oxidant and immunomodulatory properties of seabuckthorn (*Hippophae rhamnoides*) – An in vitro study. J Ethnopharmacol 2002;79:373–378.

7. Xing J, Yang B, Dong Y, et al. Effects of sea buckthorn (*Hippophaë rhamnoides* L.) seed and pulp oils on experimental models of gastric ulcers in rats. Fitoterapia 2002;73:644–650.

8. Murray MJ. Equine model of inducing ulceration in alimentary squamous epithelial mucosa. Dig Dis Sci 1994;39:2530–2535.

9. MacAllister CG, Andrews FM, Deegan E, et al. A scoring system for gastric ulcers in the horse. Equine Vet J 1997;6:430–433.

10. Erkkola R, Yang B. Sea Buckthorn oils: Towards healthy mucous membranes. AgroFood Industry hi-tech 2003;3:53–57.

11. Tandon R, Khanna HD, Dorababu M, Goel RK. Oxidative stress and antioxidant status in peptic ulcer and gastric carcinoma. Indian J Physiol Pharmacol 2004;48:115–118.

12. Malik S, Goyal S, Ojha SK, et al. Seabuckthorn attenuates cardiac dysfunction and oxidative stress in isoproterenol-induced cardiotoxicity in rats. Int J Toxicol 2011;30:671–680.

13. Jeffrey SC, Murray MJ, Eichorn ES. Distribution of epidermal growth factor receptor (EGFr) in normal and acute peptic-injured equine gastric squamous epithelium. Equine Vet J 2001;33:562–569.

14. Suleyman H, Albayrak A, Bilici M, et al. Different mechanisms in formation and prevention of indomethacin-induced gastric ulcers. Inflammation 2010;33:224–234.

15. Swarnakar S, Ganguly K, Kundu P, et al. Curcumin regulates expression and activity of matrix metalloproteinases 9 and 2 during prevention and healing of indomethacin-induced gastric ulcers. J Biol Chem 2005;280:9409–9415.

16. Dursun H, Bilici M, Albayrak F, et al. Antiulcer activity of fluvoxamine in rats and its effect on oxidant and antioxidant parameters in stomach tissue. BMC Gastro 2009;9:36.

17. Reese RE, Andrews F, Elliott S, et al. Effects of seabuckthorn berry extract (Seabuck Complete) on prevention and treatment of gastric ulcers in horses. In: White NA, ed. Proceedings 9th International Equine Colic Research Symposium. Liverpool, England: British Equine Veterinary Association (BEVA); 2008: 125–126.

18. Lybbert T, Gibbs P, Cohen N, et al. Feeding alfalfa hay to exercising horses reduces the severity of gastric squamous mucosal ulceration. Proc Am Assoc Equine Pract 2007; 53:525–526.

19. Nadeau JA, Andrews FM, Mathew AG, et al. Evaluation of diet as a cause of gastric ulcers in horses. Am J Vet Res 2000;7:784–790.

20. Reese RE, Andrews FM. Nutrition and dietary management of equine gastric ulcer syndrome. Vet Clin N Amer Eq Pract 2009;25:79–92.

21. Murray MJ, Schusser GF. Measurement of 24-h gastric pH using an indwelling pH electrode in horses unfed, fed and treated with ranitidine. Equine Vet J 1993;5:417–421.

22. Larmo P. The Health Effects of Seabuckthorn Berries and Oil. Turku, Finland: Department of Biochemistry and Food Chemistry, University of Turku; 2011. PhD Thesis.

23. Manach C, Scalbert A, Morand C, et al. Polyphenols: Food sources and bioavailability. Am J Clin Nutr 2004;79:727–747.

24. McClure SR, Glickman GT, Glickman NW. Prevalence of gastric ulcers in show horses. J Am Vet Med Assoc 1999;216: 1130–1133.