Effects of dietary oils on the development of gastric ulcers in mares

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Objective—To assess antiulcerogenic properties of 3 dietary oils.

Animals—8 healthy adult mares.

Procedure—A protocol to induce gastric ulcers was used and included 240 mL of water plus corn oil, refined rice bran oil, or crude rice bran oil administered each day for 6 weeks according to a 4 × 4 Latin square randomized crossover design with 5-week washout intervals. A 7-day alternating feed deprivation period was included between weeks 5 and 6. Omeprazole was administered daily for the last 14 days of each washout interval. Endoscopic examinations of the stomach were performed at 0, 5, and 6 weeks, and the number (0 to 4 scale) and severity (0 to 5 scale) of ulcers were scored. Gastric fluid was collected at 0 and 5 weeks.

Results—Median body weight significantly increased by 29 kg (range, 10 to 50 kg). Mean ± SE gastric fluid pH significantly decreased from 4.9 ± 0.4 to 3.1 ± 0.3 over 5 weeks, and total volatile fatty acid concentration significantly decreased over time. Mean ± SE severity of nonglandular ulcers significantly increased from 0.4 ± 0.1 to 1.2 ± 0.2 over 5 weeks. Nonglandular ulcers significantly increased in number (mean ± SE, 1.3 ± 0.2 to 3.0 ± 0.2) and severity (mean ± SE, 1.2 ± 0.2 to 2.6 ± 0.2) during the 7-day alternating feed deprivation period. No effects of treatment were detected.

Conclusions and Clinical Relevance—In this model dietary oils did not prevent gastric ulcers from forming in the nonglandular portion of the stomach of horses. (*Am J Vet Res* 2005;66:2006–2011)

Gastric ulcers commonly occur in horses and affect both condition and performance. The prevalence of gastric ulcers has been reported to be 58% in show horses[1] and as high as 93% in Thoroughbred racehorses[2] in training. Horses with gastric ulcers can be treated with a variety of drugs including omeprazole, but these treatments are expensive and must be administered for several weeks.[3] Omeprazole may also be used on a daily basis to prevent gastric ulcer disease by reducing acid production,[4] but this effect does not persist after the drug is discontinued. An antiulcerogenic diet or feed additive would therefore be desirable to prevent gastric ulcers in horses that are at high risk.

Potential antiulcerogenic dietary factors have been examined in 2 studies.[5,6] In the first, higher gastric fluid pH values and lower nonglandular ulcer number and severity scores were detected when the same horses were fed a combination of alfalfa hay and grain instead of bromegrass hay alone.[5] Results suggested that higher concentrations of calcium and protein in alfalfa hay buffered acid produced in the stomach and therefore inhibited nonglandular ulcer formation.[5] In a second study,[6] acid output from the stomach significantly decreased when corn oil was administered by dose syringe to ponies fitted with gastric cannulas. Ponies were not evaluated for gastric ulcers, but results of in vitro studies[7,8] indicate that gastric mucosal injury is less likely if the environment is more alkaline.

Rice bran oil is extracted from the pericarp and germ of rice (*Oryza sativa*) seeds and can be found in refined and crude forms.[9] Refined rice bran oil has the same fatty acid profile as crude rice bran oil, but the unsaponifiable fraction that contains phytosterols has been removed.[9] Of the phytosterols contained in crude rice bran oil, γ-oryzanol has received the most attention because of its cholesterol-lowering properties.[9] Rice bran oil has also been reported to possess antiulcerogenic properties if it is stored appropriately to avoid rancidity.[10] Rats fed rice bran oil for 4 days had significantly lower gastric ulcer index values (based on the total area of ulceration) after being stressed, compared with untreated controls.[10]

The purpose of the study reported here was to evaluate whether corn oil, refined rice bran oil, or crude rice bran oil possess antiulcerogenic properties. We hypothesized that addition of dietary oils to the diet would significantly decrease gastric fluid **volatile fatty acid (VFA)** concentrations, increase the pH in the stomach, and prevent gastric ulcers.

Materials and Methods

Horses—Eight healthy mares of mixed-breed and Quarter Horse body type were selected for use in the study. Mares were chosen to reduce variability associated with differences in sex. Immature and older horses were also excluded. Median age was 9 years (range, 5 to 17 years), and median body weight at the beginning of the study was 433 kg (range, 368 to 494 kg). A 5-year-old mare weighing 385 kg was withdrawn from the study after the first treatment period when acute neurologic deficits were observed at the beginning of the first washout interval. This mare was replaced by a 5-year-old mare weighing 379 kg that had been housed on the same pasture. Each mare was housed in its own 3.7 × 3.7-m stall for 10 days prior to and during treatment periods. Mares were turned out onto pasture during washout intervals. When housed in stalls, mares were turned out into a round pen for approximately 2 hours of exercise every other day. The study protocol was approved by the

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University of Tennessee Institutional Animal Care and Use Committee.

Experimental design—Eight mares were randomly paired and allocated to 4 treatment groups. A 4 × 4 Latin square randomized crossover design was used, with four 6-week treatment periods separated by three 5-week washout intervals (39-week total). Six weeks was selected as the length of the treatment period because rice bran oil inhibited the formation of gastric ulcers in rats that were fed the oil for 2 weeks[11] and 3- to 10-week treatment periods have been used in studies[12,13] that examined the effects of soybean oil on blood lipid concentrations in horses. Six-week treatment periods were divided into a 5-week period with a protocol designed to be ulcerogenic, followed by 7 days when mares were deprived of feed for 24 hours (96 hours in total) or fed as normal on alternating days (intermittent feed deprivation period) according to the model developed by Murray and Eichorn.[14] Feed deprivation was achieved by placing a muzzle over each mare's nose. Washout intervals lasted 35 days (5 weeks) in total and consisted of mares being turned out on pasture for 25 days, followed by 10 days when mares were returned to their stalls. A 5-week washout interval was selected to avoid potential carryover effects. Fatty acids are incorporated into cell membranes, and several weeks may be necessary to allow the composition of membranes to adjust to a new diet.[15] Grass hay was available for ad libitum consumption at both locations during washout intervals. Omeprazole[a] (2.28 g, PO, q 24 h) was administered for 14 days before each treatment period with the aim of starting with low or zero gastric ulcer scores.

Treatments consisted of 240 mL of water, corn oil,[b] refined rice bran oil,[c] or crude rice bran oil.[d] This volume of oil was selected because gastric ulcers were prevented when 0.1 mL of rice bran oil was administered to rats that weighed 100 to 160 g in a previous study.[11] Assuming an approximate body weight of 400 kg, the calculated equivalent volume for a horse was approximately 250 mL of oil when the lowest dosage was used in calculations. This volume was reduced to 240 mL (approx 0.6 mL of oil/kg) because 8 fl oz is a common measure used by horse owners. Water or oil was mixed with the morning grain meal, and mares were observed to ensure that all of the oil and feed was consumed. Treatments were administered orally via dose syringe on days when feed was withheld. Endoscopic examinations were performed before (week 0) and 5 weeks after the ulcerogenic protocol was initiated. An endoscopic examination was also performed after 7 days of intermittent feed deprivation (week 6). Gastric fluid was collected at weeks 0 and 5. Mares were observed daily, and body weights were measured at weeks 0 and 5 of each treatment period.

Ulcerogenic protocol—A base diet was formulated with the aim of inducing gastric ulcers, and mares received this diet for the first 5 weeks of every treatment period. Mares were gradually introduced to the diet during the first 7 days of each treatment period by feeding 25% of the grain ration for 2 days, 50% for 2 days, and 75% for 2 days. The diet was formulated to provide 1.5 times the digestible energy requirement for maintenance.[16] Of this requirement, most of the calories were supplied by feeding grain[e] at 1% (by weight) of body weight with the balance met by feeding grass hay. Amounts of feed were calculated on the basis of body weight measured at the beginning of each treatment period and digestible energy content of feeds, which was determined by independent analysis.[f] Grain and grass hay contained a median of 3.56 (range, 3.56 to 3.63) and 1.86 (range, 1.80 to 1.96) Mcal of digestible energy/kg as fed, respectively. When fed at 1% of body weight, 3.7 to 5.1 kg of grain/d was fed to each mare. Housing and feeding practices were also adjusted with the aim of inducing gastric ulcers. Mares were confined to stalls and fed their total daily amount of grain as a single meal in the morning 1 hour before hay was fed. The daily allowance of hay was divided equally between morning and evening feedings.

Dietary oils—Corn oil, refined rice bran oil, and crude rice bran oil were stored at 21°C and protected from light until fed. Oils were measured out from bulk storage bottles each morning, and 240 mL of oil or water was added to the morning grain meal and mixed thoroughly.

Endoscopic examination—Mares were sedated by use of detomidine hydrochloride[g] (0.02 mg/kg, IV), and an endoscopic examination of the stomach was performed on each mare with a 3.4-m video endoscope.[h] To enable observation of the nonglandular squamous mucosa (fundus ventriculi), margo plicatus, and glandular mucosa (corpus ventriculi), the stomach was insufflated with air and the mucosa was rinsed with tap water flushed through the endoscope biopsy channel. Number (0 to 4 scale) and severity (0 to 5 scale) of nonglandular and glandular mucosal lesions were scored in accordance with an equine gastric lesion scoring system[17] by a researcher (FMA) who was unaware of the diet each mare was being fed. At the beginning and end of each 5-week treatment period, mares were fed at 3 PM the day before endoscopic examinations and then all feed was removed from stalls at 5 PM the night before endoscopic examinations and gastric fluid collections were performed. Water remained available for ad libitum intake. Procedures were performed in the morning between 7:30 AM and 9:30 AM after mares were deprived of feed overnight (water remained available). Week 6 endoscopic examinations were performed at the end of the 7-day alternating feed deprivation period.

Gastric fluid collection—Approximately 100 mL of gastric fluid was withdrawn via the biopsy channel of the endoscope with suction and collected into a glass flask. Gastric fluid was collected from mares at the beginning and end of each 5-week treatment period.

Analysis of gastric fluid—The pH of gastric fluid was measured with a pH electrode,[i] and the fluid was immediately transferred to precooled plastic tubes. Tubes were transported to the laboratory on ice and stored frozen at –20°C. Concentrations of volatile fatty acids (acetic, propionic, butyric, isobutyric, valeric, and isovaleric acids) were subsequently measured in aliquots of gastric fluid. Values were obtained by use of a gas chromatography method described by Playne[18] and modified by Mathew.[19] Reported VFA concentrations are from gastric fluid collected approximately 15 hours after feeding. Lactic acid concentrations were not measured because in a previous study, D- and L-lactate concentrations were less than detectable concentrations in gastric fluid collected from horses deprived of feed overnight.[3]

Statistical analyses—Body weight data were non-normally distributed and were therefore compared between weeks 0 and 5 by use of the Wilcoxon signed rank test. Gastric fluid pH, VFA concentrations, and gastric ulcer scores were compared between groups by use of ANOVA for repeated measures with a mixed procedure.[j] Effects of time, block, and treatment were examined. When significant main effects were detected, least squares means were compared by use of the Bonferroni test. Values of $P < 0.05$ were considered significant.

Results

Body weights at the beginning of each treatment period ranged from 368 to 512 kg. Median body weight significantly increased by a median of 29 kg (range, 10

Table 1—Pooled mean ± SE pH values and total volatile fatty acid (VFA) concentrations in gastric fluid and scores for gastric ulcers in the nonglandular portion of the stomach observed via endoscope in 8 mares fed experimental diets for 5 weeks, followed by a 7-day intermittent feeding period.

	Time (wk)		
Variable	**0**	**5**	**6**
Gastric fluid pH	4.9 ± 0.4[a]	3.1 ± 0.3[b]	–
Total VFA concentration (mmol/L)	1.50 ± 0.13[a]	1.12 ± 0.10[b]	–
Nonglandular ulcer score			
Number (0–4 scale)	0.8 ± 0.2[a]	1.3 ± 0.2[a]	3.0 ± 0.2[b]
Severity (0–5 scale)	0.4 ± 0.1[a]	1.2 ± 0.2[b]	2.6 ± 0.2[c]

[a-c]Across a row, mean values with different superscripts differ significantly ($P < 0.05$).
– = Not measured.

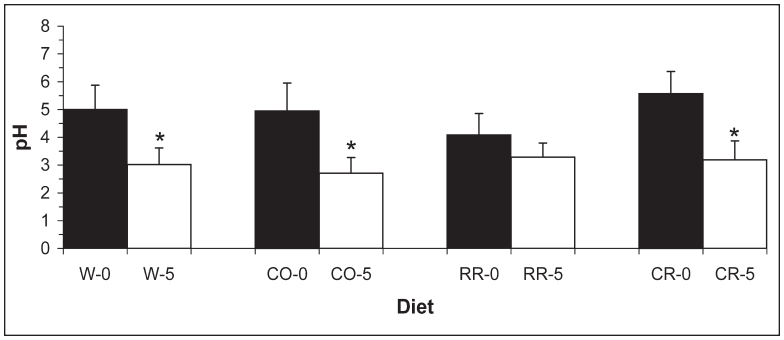

Figure 1—Mean ± SE pH in gastric fluid collected from 8 mares fed a base diet supplemented with 240 mL of water (W), corn oil (CO), refined rice bran oil (RR), or crude rice bran oil (CR) per day for 5 weeks (week 0 to week 5). Significant ($P < 0.05$) time and period × time effects were detected, but no significant differences were detected among treatment groups. *Significant ($P < 0.05$) difference between weeks 0 and 5 within a treatment group.

to 50 kg) overall, with median increases of 25, 29, 31, and 30 kg detected for water, corn oil, refined rice bran oil, and crude rice bran oil treatment groups, respectively. However, there were no significant differences among treatment groups.

Mean ± SE gastric fluid pH significantly decreased during 5 weeks (**Table 1**), but differences among treatment groups were not identified (**Figure 1**). Mean ± SE total VFA, acetic acid, and isobutyric acid concentrations significantly decreased during 5 weeks (Tables 1 and 2), but VFA concentrations did not differ among treatment groups. Volatile fatty acid concentrations were also influenced by block because concentrations increased during 5 weeks during the third block.

Percentage VFA composition of gastric fluid did not vary significantly with time or treatment. Gastric fluid VFAs were composed of 85.0% acetic acid, 8.6% propionic acid, 2.2% butyric acid, 2.2% isobutyric acid, and 2.1% isovaleric acid. Valeric acid was only detectable in 1 gastric fluid sample. Significant block × time effects were also detected. Gastric fluid pH and VFA concentrations decreased during 5 weeks in blocks 1, 2, and 4 but increased in the third block.

Mean number and severity ulcer scores were < 1 at the beginning of each treatment period after 14 days of administration of omeprazole. Significantly ($P = 0.01$) higher nonglandular ulcer severity scores were detected after 5 weeks in mares that consumed experimental diets (Table 1), but the number or severity of nonglandular ulcers

Table 2—Mean ± SE VFA concentrations in gastric fluid collected via endoscope approximately 15 hours after feeding in 8 mares fed a base diet plus 240 mL of water (W), corn oil (CO), refined rice bran oil (RR), or crude rice bran oil (CR) per day for 5 weeks. Time and period × time effects were significant ($P < 0.05$) for acetic and isobutyric acid concentrations. Valeric acid was only detectable in 1 sample.

	Treatment			
VFA	**W**	**CO**	**RR**	**CR**
Acetic (mmol/L)*				
Baseline	1.38 ± 0.25	1.19 ± 0.28	1.25 ± 0.19	1.20 ± 0.13
5 wk	1.00 ± 0.17	1.12 ± 0.27	0.89 ± 0.08	0.80 ± 0.12
Difference	–0.38 ± 0.31	–0.07 ± 0.19	–0.36 ± 0.25	–0.41 ± 0.16
Propionic (mmol/L)				
Baseline	0.16 ± 0.04	0.13 ± 0.04	0.15 ± 0.05	0.12 ± 0.02
5 wk	0.10 ± 0.03	0.15 ± 0.06	0.08 ± 0.01	0.09 ± 0.03
Difference	–0.06 ± 0.05	0.02 ± 0.03	–0.07 ± 0.05	–0.03 ± 0.02
Isobutyric (mmol/L)*				
Baseline	0.04 ± 0.01	0.04 ± 0.01	0.02 ± 0.01	0.03 ± 0.00
5 wk	0.03 ± 0.00	0.03 ± 0.01	0.02 ± 0.00	0.02 ± 0.00
Difference	–0.01 ± 0.01	0.00 ± 0.01	0.00 ± 0.01	–0.01 ± 0.01
Butyric (mmol/L)				
Baseline	0.04 ± 0.01	0.05 ± 0.01	0.02 ± 0.00	0.04 ± 0.01
5 wk	0.03 ± 0.01	0.04 ± 0.02	0.02 ± 0.01	0.02 ± 0.01
Difference	–0.01 ± 0.02	0.00 ± 0.01	0.00 ± 0.01	–0.02 ± 0.01
Isovaleric (mmol/L)				
Baseline	0.04 ± 0.01	0.03 ± 0.01	0.02 ± 0.00	0.03 ± 0.01
5 wk	0.03 ± 0.00	0.03 ± 0.01	0.02 ± 0.00	0.02 ± 0.01
Difference	–0.01 ± 0.01	0.00 ± 0.01	0.00 ± 0.01	–0.01 ± 0.01

*Variable significantly ($P < 0.05$) affected by time.

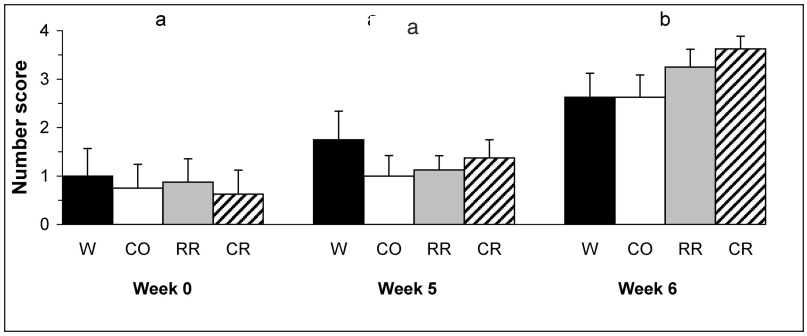

Figure 2—Mean ± SE number scores (0 to 4 scale) for gastric ulcers in the nonglandular portion of the stomach observed via endoscope in 8 mares fed a base diet supplemented with 240 mL of W, CO, RR, or CR per day for 5 weeks, followed by a 7-day intermittent feeding period. Mean number scores across all treatment groups were significantly ($P = 0.01$) higher at week 6, compared with week 5 and baseline values. Significant differences were not detected among treatment groups. [a,b]Mean number scores for each week (all treatment groups combined) with different letters are significantly ($P < 0.05$) different.

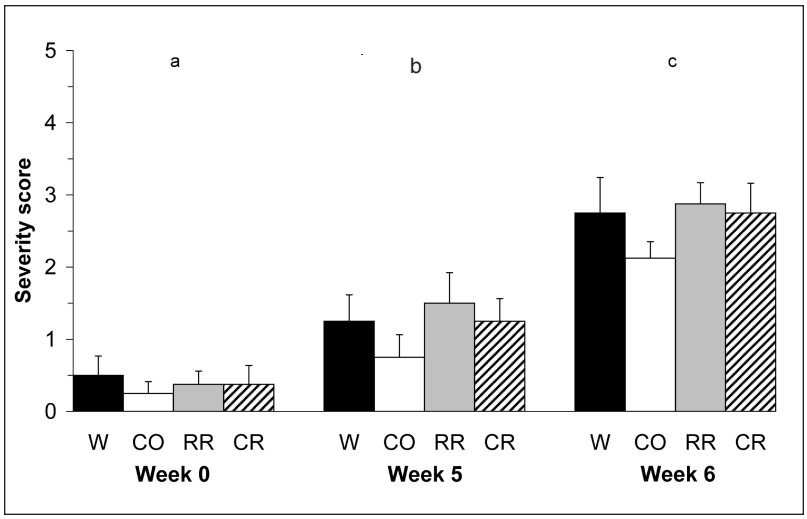

Figure 3—Mean ± SE severity scores (0 to 5 scale) for gastric ulcers in the nonglandular portion of the stomach observed via endoscope in the same mares as Figure 2. [a-c]Mean severity scores across all treatment groups were significantly ($P = 0.01$) higher at weeks 5 and 6, compared with baseline values, and increased significantly ($P < 0.05$) between weeks 5 and 6. Significant ($P < 0.05$) differences were not detected among treatment groups.

did not differ significantly among treatment groups (**Figures 2 and 3**). After 7 days of alternating feed deprivation, the number and severity of nonglandular ulcer scores were significantly ($P = 0.01$) higher than week 5 and baseline values (Table 1), but these scores also remained unaffected by treatment (Figures 2 and 3). Glandular mucosal ulcers were only detected on 4 occasions during the study. A single grade 1 glandular mucosal ulcer was detected in 2 mares at week 0, and 1 of the mares treated with refined rice bran oil developed a grade 2 glandular mucosal ulcer that was first detected at week 5 and then observed again at week 6.

Discussion

Mares were confined in stalls, placed on a high-grain diet, and fed grain before hay in order to mimic husbandry practices used in many show barns and racing stables. These practices were selected because show horses and racehorses are at highest risk for developing gastric ulcers.[1,2] We also postulated that a high-grain diet would induce gastric ulcers by raising VFA concentrations within the stomach. Higher VFA concentrations have been detected in gastric fluid collected from horses fed a combination of grain and alfalfa hay, compared with bromegrass hay alone, although both feeds may have contributed to this effect.[5] As a consequence of these dietary manipulations, all of the mares in this study gained weight during the 5-week period because caloric intake exceeded energy demand. Mares that received oils received approximately 1.6 Mcal of additional digestible energy/d[20] and gained more weight than those that received water, although differences were not significant.

Addition of grain to the diet significantly lowered gastric fluid pH at 15 hours after feeding in this study, but supplementation with dietary oils did not affect this response. In a previous study,[6] ponies treated with 45 mL of corn oil/d orally for 5 weeks had a significant reduction in gastric acid production, but grass hay was the only feed provided during control and treatment periods. Grain may therefore exert an effect on gastric pH that cannot be attenuated by dietary oils. Further studies are required to assess whether dietary oils have antiulcerogenic properties if they are administered at higher dosages or at a higher frequency. Multiple samples should also be collected in future studies because gastric pH fluctuates widely throughout the day.[21]

An alternative explanation for the reduction in gastric fluid pH with time was that week 0 pH values were increased because omeprazole was administered for 14 days prior to testing. Gastric acid production decreases[22] and gastric fluid pH increases to > 5.0[23] when omeprazole is administered to horses. Omeprazole may therefore have influenced week 0 gastric fluid pH values in this study. A mean gastric fluid pH of 4.9 was detected approximately 24 hours after the last dose of omeprazole in this study, which was much higher than the pH of 2.0 detected in gastric fluid collected from feed-deprived horses in a previous study.[22]

Volatile fatty acid concentrations in gastric fluid collected approximately 15 hours after feeding were similar to 12- and 24-hour postfeeding concentrations measured in a previous study.[5] In that study, acetic acid comprised 78% of gastric VFA and was found at concentrations < 5 mmol/L when sampled 12 hours after feeding.[5] When gastric fluid was collected from horses euthanatized shortly after consuming grass hay, acetic acid represented 88% of measured VFA and a maximum concentration of 16.23 mmol/L was detected.[7] In

contrast, acetic acid comprised 85% of gastric VFA in our study and ranged in concentration from 0.43 to 2.95 mmol/L. Acetic, propionic, butyric, and valeric acids have been implicated in the pathogenesis of nonglandular gastric ulcers.[7,8] Gastric mucosa maintained at a pH ≤ 4 sustains more damage if it is exposed to higher VFA concentrations.[7,8]

Gastric fluid VFA concentrations were low at the time of sampling and did not differ significantly among treatment groups. However, significantly lower total, acetic, and isobutyric acid concentrations were detected after 5 weeks, which suggests that switching mares to a diet containing grain lowered gastric fluid VFA concentrations. This is the opposite finding to the one anticipated, but the 15-hour postfeeding sampling time must be considered when interpreting results. Feeding grain may have markedly raised gastric fluid VFA concentrations in the hours immediately following feeding, yet this increase might not be detected when samples are collected 15 hours after feeding. Attempts were made to collect gastric fluid in the postprandial period, but ingesta blocked passage of the endoscope into the ventral portion of the stomach. This limitation greatly restricted assessment of the effects of diet on gastric fluid VFA concentrations in this study. Grain was expected to increase gastric fluid VFA concentrations because this feed is rich in nonfiber carbohydrates, including sugars and starches that are readily fermentable. Sweet feeds contain as much as 57.8% nonfiber carbohydrates on a dry-matter basis, compared with 20.7% in alfalfa pellets.[24] Alternative methods of extracting gastric fluid must be used in the future if postprandial gastric fluid VFA concentrations are to be assessed.

Gastric fluid pH and VFA concentrations were both influenced by block in this study, but an explanation for these discrepancies was not apparent. Nutrient composition of feeds may have influenced results, but pH values and VFA concentrations did not vary according to changes in nutrient or digestible energy concentrations. Substitution of 1 mare for another was also discounted as a source of variability because this event occurred during the first washout interval. One possible explanation for this effect was that water intake may have varied between sampling times and resulted in dilution of gastric fluid. All horses were deprived of feed overnight prior to gastric fluid collection, but water intake was not restricted. Higher gastric fluid VFA concentrations detected at the end of the third block may have resulted from a reduction in water intake.

The order in which feeds were provided was also reversed in our experimental model, with the aim of inducing gastric ulcers. Feeding hay prior to grain is commonly recommended as a strategy to inhibit gastric ulcers by introducing a feed substrate that buffers acid.[3,21] However, only the severity of gastric ulcers increased during this phase of the model. Number score did not increase significantly, and few glandular ulcers developed. This phase of the model might therefore be improved by selecting different subjects, housing horses in a more stressful environment, and eliminating the afternoon feeding in order to create a daily feed deprivation period. Response to this model should also be evaluated in horses of different sex because it has been suggested that geldings are more likely to develop gastric ulcers.[25]

Supplementation with 240 mL of corn oil, refined rice bran oil, or crude rice bran oil (approx 0.5 to 0.6 mL/kg of body weight) did not significantly alter the development of gastric ulcers in this study, so our hypothesis was rejected. These findings contrast with results of a recent study[6] in which ponies fitted with gastric cannulas had a significant reduction in acid output when they received 45 mL of corn oil/d (approx 0.3 to 0.4 mL/kg of body weight) for 5 weeks. However, gastric ulcer scores were not reported in these ponies, and horses were not evaluated. Further studies are therefore required to determine whether dietary oils can inhibit gastric ulcer formation when administered under different conditions. For instance, dietary oils may have to be administered more frequently to prevent gastric ulcers in horses when experimental models are used.

a. Gastrogard, Merial, Duluth, Ga.
b. Corn oil, Ventura Foods, Brea, Calif.
c. Refined rice bran oil, RITO Inc, Stuttgart, Ark.
d. Rice bran oil, McCauley Bros Inc, Versailles, Ky.
e. Co-op 13% Supreme Performance, Tennessee Farmers Cooperative, Lavergne, Tenn.
f. Dairy One DHIA Forage Testing Laboratory, Ithaca, NY.
g. Dormosedan, Pfizer Inc Animal Health Group, New York, NY.
h. Endoscope, ETM-Endotech, München, Germany.
i. AP62, Accumet AP61 portable pH meter, Fischer Scientific, Pittsburgh, Pa.
j. SAS, version 9.0, SAS Institute Inc, Cary, NC.

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