How to Minimize Gastrointestinal Disease Associated With Carbohydrate Nutrition in Horses

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The feeding of high cereal grain rations and tendency to suppress natural foraging behavior are at odds with healthy function of the horse’s gastrointestinal tract and may increase risk of colic and gastric ulcer disease. Recommended feeding practices targeting reduced risk for development of diet-associated gastrointestinal problems included the provision of adequate forage (1.5% of body weight per day), limiting the size of high starch meals (<2.0 kg for a 500-kg horse), and increasing use of non-starch sources of energy (e.g., vegetable oils and fiber sources such as beet pulp and soya hulls). Authors’ addresses: Middleburg Agricultural Research and Extension Center, Virginia Tech, 5527 Sullivans Mill Road, Middleburg, VA (Geor); and Equine Studies Group, WALTHAM Centre for Pet Nutrition, Melton Mowbray, Leicestershire LE14 4RT, United Kingdom (Harris); e-mail: rgeor@vt.edu. © 2007 AAEP.

1. Introduction

Carbohydrates are the primary source of energy in the diet of horses. Horses, as non-ruminant herbivores, evolved to use forages high in structural carbohydrates through bacterial fermentation and the production of volatile fatty acids (VFAs) in a highly developed large intestine. However, the daily digestible energy (DE) needs of racehorses, endurance horses, and 3-day eventers are about double the requirements of horses not in training and typically forage (relatively low energy density and bulky) alone will not satisfy the energy demands of their athletic training and competition. To meet this increased energy requirement, horses are commonly fed more energy dense feedstuffs, especially concentrates rich in starch and sugar (non-structural carbohydrates, e.g., cereal grains), which in turn often results in decreased provision of forage compared with the non-working state. Survey studies have indicated that racehorses weighing 450–550 kg typically receive 3–6 kg of concentrate per day, with some horses receiving >8 kg/day.1-3 Such high grain intakes by horses (and/or a low forage-to-concentrate ratio, e.g., 30:70) have been implicated in the development of gastrointestinal problems, particularly gastric ulcer disease and colic associated with disturbances to hindgut function.

The objectives of this paper are to (1) review information concerning links between diet, feeding practices, and disturbances in gastrointestinal function (e.g., gastric ulcers, colic), especially in relation to carbohydrate nutrition, and (2) provide recommendations for feeding management that may help
to reduce the risk for development of gastrointestinal disease.

2. Feeding Management and Colic

Colic is caused by many conditions, each of which may be related to specific risk factors such as changes in diet, feeding practices, exercise patterns, and housing, or inappropriate parasite control programs. An association between feeding practices and disturbances in gastrointestinal function has long been hypothesized, but the mechanisms linking diet with the development of intestinal dysfunction are poorly understood. Indeed, the exact relationship between diet and colic is difficult to determine because of the variety of feeds and feeding practices used throughout the world, as well as differences in study populations. Furthermore, it is often difficult to separate the effects of diet and feeding schedule from other management practices, which often will depend on the horse’s breed and use. Nonetheless, the results of recent epidemiological studies have provided support for the proposition that diet composition and recent changes in diet are important risk factors for development of colic.

Tinker et al. prospectively examined the risk for colic on 31 horse farms over a 1-year period. Both a change in concentrate feeding (odds ratio [OR] = 3.6 relative to no colic) and the feeding of high levels of concentrate (>2.5 kg/day dry matter, OR = 4.8 and >5 kg/day dry matter, OR = 6.3, relative to feeding no concentrate) were identified as risk factors for colic. In addition, colic risk increased when processed feeds such as pellets were fed. Hudson et al. reported that a recent (within 2 wk) change in the type of grain or concentrate fed (OR = 2.6), the feeding of >2.7 kg of oats/day (OR = 5.9), and a change in the batch of hay fed (OR = 4.9) were significant risk factors for an episode of colic. In another prospective, case control study, neither the amount nor type of concentrate fed was associated with colic risk, although the researchers did conclude that horses at pasture may have a decreased risk of colic. On the other hand, a recent (within 2 wk) change in diet, in particular the type of hay fed (including hay from a different source or cutting of the same type of hay), was a significant risk factor for colic. In this study, feeding hay other than coastal/Bermuda or alfalfa significantly increased the colic risk, but this may have reflected hay quality and digestibility rather than type of hay per se. Changing to a poorer-quality, less-digestible hay or feeding wheat straw or cornstalks may predispose horses to large colon impaction. In a practitioner-based colic study in the United Kingdom, a recent change in management was associated with at least 45% of the cases of spasmodic or mild undiagnosed colic. The most common management change was turnout onto lush pasture in the spring.

In reviewing the results of available epidemiologic studies, Cohen estimated that approximately one third of colic cases had a history of a recent change in diet. The ingestion of high concentrate and low forage diets has also been implicated in the development of gastric ulcers, which in turn may result in signs of colic.

These observations raise several questions concerning the effects of diet composition and dietary change on gastrointestinal function, including the capacity of the equine digestive tract for grain (starch) digestion, possible reasons for increased colic risk with high levels of grain feeding, and the effect of a sudden change in diet (grain or forage) on gastrointestinal function.

3. Carbohydrate Digestion and Hindgut Function

Limited Capacity for Starch Hydrolysis in the Small Intestine

From a digestive viewpoint, carbohydrates in horse feedstuffs can be divided into three main fractions: (1) hydrolyzable carbohydrates (CHO-H), which can be digested in the small intestine by mammalian enzymes (or if they escape digestion in the small intestine and reach the hindgut, can be rapidly fermented in the hindgut); (2) rapidly fermentable carbohydrates (CHO-FR), which cannot be broken down by mammalian digestive enzymes but are readily available for microbial fermentation; and (3) slowly fermentable carbohydrates (CHO-FS). The hydrolyzable fraction included hexoses, disaccharides, some oligosaccharides, and the non-resistant starches. Although some fermentation of these compounds may occur in the stomach, the primary products of digestion of these compounds are monosaccharides that can be absorbed in the small intestine, with a relatively high energy yield. The rapidly fermentable fraction included pectin, fructan, and some oligosaccharides not digested in the small intestine. Resistant starch and neutral detergent hemicellulose could also be included in the rapidly fermented fraction. The slowly fermented carbohydrate fraction includes cellulose, hemicellulose, and ligno-cellulose that result primarily in the production of acetate in the large intestine.

In non-ruminant species, there are three primary steps in the hydrolysis of starch: (1) hydrolysis of α-1,4 glycosidic bonds by pancreatic α-amylase and intestinal glucoamylase, yielding primarily maltose and maltotriose (which is further hydrolyzed to maltose and d-glucose); (2) hydrolysis of maltose by the brush-border membrane disaccharidase maltase, yielding d-glucose; and (3) transport of d-glucose across the enterocyte brush-border membrane by the Na+/glucose co-transport protein, SGLT1. There is some evidence that horses have a limited capacity for complete digestion of starch in the small intestine (i.e., pre-cecal starch digestion). At low levels of starch intake (130–140 g/100 kg body weight [BW] from oats, barley or corn as a single meal), ~80% of the starch was digested in the small intestine. When starch feeding was doubled (250–270 g/100 kg BW), pre-cecal starch digestibility decreased to 50–55%. Grain type
and processing affect the efficiency of starch hydrolysis in the small intestine, and thus, the amount of starch that can be tolerated (see Recommendations for Minimizing Digestive Disturbances). Based on the results of their studies, Potter et al. recommended that the maximum amount of starch that should be fed at one meal is 3.5–4.0 g/kg BW. Feeding <300 g starch per 100 kg BW was recommended by another author, and recently, it has been suggested that, even at this level, there may be concerns depending on the nature of the feed. This recent work suggested that, whereas at 300 g starch per 100 kg BW, all the oat starch was digested in the small intestine, 20% of the barley starch and 34% of the corn starch escaped the pre-cecal digestion and reached the large intestine. This was supported by work by Cuddeford et al., which showed that 2.1 g starch/kg BW/meal from a hay cube:rolled barley diet (50:50) was sufficient to elicit unfavorable changes in intra-cecal fermentation in ponies, suggesting a maximum value of 2.0 g starch/kg BW/meal. For example, if a grain concentrate is 50% starch, no more than 4 g/kg BW or ~2 kg for a 500-kg horse should be fed. At higher intakes in a single meal, particularly when unprocessed corn or barley is fed, there is risk of substantial starch overflow into the hindgut, where it will undergo rapid fermentation.

It should be noted that the data on pre-cecal starch digestibility were derived from studies in which horses or ponies were adapted to grain feeding for a 3- to 4-wk period. The upper limit of pre-cecal starch digestibility may be lower in horses not accommodated to grain or concentrate feeding. In this regard, it is possible that the increased risk of colic after a change in the amount or type of grain fed reflects slow or inadequate adaptation in mechanisms for starch digestion in the small intestine. In omnivorous species, there is a rapid increase in the expression and activity of SGLT1 in response to increased dietary load of starch or sugar, and therefore, an increase in glucose absorptive capacity. Currently, there are no data in horses on the speed and extent of any adaptations in small intestinal carbohydrate digestive functions in response to increased grain feeding. A substantial lag in the up-regulation of small intestinal carbohydrate digestive and absorptive mechanisms could provide one explanation for colic events subsequent to an abrupt change in diet.

**Hindgut Fermentation: Effects of Starch and Other Rapidly Fermentable Substrate**

The equine large intestine (cecum and colon) is an enlarged fermentative chamber that contains an extremely abundant and highly complex community of microorganisms. Although some fermentation of feedstuffs occurs in the stomach and small intestine, most fermentation occurs in the hindgut. The microbial hydrolysis of dietary plant fiber within the large intestine leads to the release of soluble sugars that are subsequently fermented to the VFAs acetate, propionate, and butyrate, which are an important source of energy. In addition, the VFAs (particularly butyrate) regulate the expression of genes controlling proliferation, apoptosis, and differentiation of gut epithelial cells.

The rate of fermentation and the microbial and biochemical contents of the large intestine are affected by diet composition and feeding pattern (i.e., continuous “grazing” or small, frequent meals vs. large meals administered twice daily). An abrupt change from forage only to a forage/concentrate diet will result in an increased rate of fermentation and marked changes in the microbial population, luminal pH, and the contents of VFA and lactate. The extent of these changes is likely dependent on the nature and abruptness of the dietary change. With a sudden increase in grain (i.e., starch) feeding, a portion of the ingested starch passes into the cecum undigested, where it undergoes rapid fermentation with increased production of lactate and gas and a decrease in cecal/colonic pH. Increasing proportions of grain result in decreased acetate and increased propionate and lactate contents of the cecum and colon. Other biochemical changes with the rapid fermentation of starch (or other CHO-FR such as fructan) in the hindgut may include an increase in the production of vasoactive monoamines (e.g., tyramine, tryptamine), endotoxins, and exotoxins, all of which have been implicated in the pathogenesis of laminitis. Studies have shown that lactate inhibits the transport of butyrate across the colonic luminal membrane by the monocarboxylate transporter 1, MCT1. Given the important role of butyrate in the regulation of gene expression at the level of the gut epithelial cells, the inhibition of butyrate transport by lactate may alter tissue homeostasis and function. An increase in lactic acid production will also cause a decrease in hindgut pH, which may increase intestinal permeability and favor the absorption of endotoxin.

After an increase in starch feeding, the numbers of lactobacilli and total anaerobic bacteria increase, whereas the numbers of xylanolytic and pectinolytic bacteria decreases. Overall, there is an increase in amylolytic, lactic acid–producing bacteria and decreases in the proportions of acid-using (particularly lactate) and cellulolytic (i.e., fiber degrading) bacteria. Reduced efficiency of fiber use and decreased energy yield may result from the decline in cellulolytic bacteria with high grain feeding. In horses fed forage only, pH within the cecum and colon is in the range of 6.7–7.0. The feeding of increasing amounts of corn or barley starch is associated with proportionate decreases in cecal pH, with values approaching 6.0 when 3–4 g/kg BW is fed as a single meal. A similar dose of oat starch was not associated with a significant decrease in pH, consistent with other data showing higher pre-cecal digestibility of oat starch vs. barley and corn starch. Radicke et al. suggested that a cecal pH of 6.0 represented sub-clinical acidosis and that risk
of development of clinically apparent intestinal dysfunction (e.g., increased permeability) is substantially increased when hindgut pH is <6.0.

Similar disruptions to the hindgut environment probably occur with the delivery of other rapidly fermentable substrates, such as the oligosaccharide fructan, which can comprise 5–50% of grass dry matter (DM), particularly temperature species including perennial rye grass and timothy. Fructans are water-soluble polymers of fructose with either β-2,1 or β-2,6 linkages, all bonded to a terminal glucose moiety. Consequently, digestion of dietary fructan is reliant on microbial hydrolysis and fermentation. Although some hydrolysis may occur in the stomach and small intestine, a substantial load of this rapidly fermentable substrate may be presented to the hindgut under grazing conditions. In vitro experiments have shown that fructan induces a more rapid decrease in the pH of cecal contents compared with corn starch, and one type of fructan (raffilose) has been used to induce carbohydrate overload and laminitis.

These disturbances to the hindgut environment put the horse at greater risk for digestive disturbances such as colic, osmotic diarrhea, and laminitis. Scenarios favoring the presentation of large loads of rapidly fermentable substrate to the hindgut include (1) a sudden introduction to grain feeding or an abrupt increase in the amount of grain concentrate; (2) the feeding of large grain meals that, even in horses adapted to such feeds, overwhelm the hydrolytic and/or absorptive capacity of the small intestine; and (3) the grazing of lush pasture or first-cut forage with high contents of rapidly fermentable substrate such as fructan and simple sugars. It is therefore apparent that feeding strategies for avoidance of hindgut disturbances must focus on minimizing the flow of rapidly fermentable substrate to the cecum and large colon.

4. Squamous Gastric Ulcer Disease

Squamous mucosal ulceration is common in performance horses, with prevalence ranging from ~40% to 90%. The prevalence and severity of gastric ulceration seems to be dependent on several factors, including feeding and housing management and the form and level of physical activity. However, intense exercise seems to play a major role in the development of squamous gastric ulcers. In an epidemiologic study of Thoroughbreds, the prevalence of gastric ulcers was 100% in actively racing horses and 91% for horses in race training. A high prevalence (67%) was also reported in one study of endurance horses after 50- and 80-km races. In an experimental study, gastric ulceration developed soon after the start of simulated race training and was maintained during the period of active training. In contrast, there is low prevalence of gastric lesions in horses given limited controlled exercise and kept at pasture. In a study of 275 Standardbreds, horses in race training were nine times more likely to have gastric ulcers than horses not in training.

Exposure of the squamous mucosa to gastric acid is thought to be the primary cause of ulceration, although other acids (short-chain or volatile fatty acids produced by fermentation in the stomach or reflux of bile acids from the duodenum) and pepsin also may play a role. Therefore, factors that alter gastric acid secretion, production of volatile fatty acids in the stomach, and exposure of the squamous mucosa to these organic acids may alter risk for development of mucosal injury and ulceration.

Repeated oral administration of hypertonic electrolyte solutions, a common practice in horses during endurance competitions, may be another risk factor for ulceration of the squamous mucosa in horses. In a study of 14 horses, oral administration of 56.7 g of commercial electrolyte supplement mixed with 60 ml of water once an hour for 8 h (~11 g sodium, 24 g chloride, 7.5 g potassium, 1.5 g calcium, and 300 mg magnesium per dose) resulted in a significant increase in mean ulcer number and severity scores.

Possible dietary influences include the effects of diet composition, meal size, and feeding frequency on saliva production; the rate and extent of intragastric fermentation; and gastric emptying rate. Saliva production during the consumption of a hay meal is approximately double that produced during intake of the equivalent DM as grain, in part because of the longer period of mastication needed for ingestion of forage. Because the flow of salivary fluid and masticated feed into the stomach may buffer the acidity of gastric contents, the lower volume of salivary fluid with ingestion of grain may favor mucosal injury because of exposure to hydrochloric acid. Another consideration is the potentially ulcerogenic effects of volatile fatty acids produced by the fermentation of starch and other readily fermentable substrate in the non-glandular region of the stomach. In studies of harvested equine gastric squamous mucosa, the addition of 60 nmol/l VFAs (butyric, propionic, acetic, and valeric acids) resulted in decreased chloride-dependent sodium transport, cell swelling, and tissue damage. In the acidic conditions of the equine stomach (pH ≤ 4.0), these organic acids will be predominantly in non-ionized forms and therefore able to penetrate and damage squamous epithelial cells. In a previous study, this research group compared the effects of a grass hay (brome grass) diet vs. a combination of legume hay (alfalfa) and grain on gastric squamous epithelial ulceration and the pH and VFA contents of gastric juice in horses with surgically implanted gastric cannulae. Surprisingly, the number and severity of squamous ulcers were greater in horses that received the grass hay-only diet. In this group, postprandial pH was lower and butyric acid concentration higher compared with the alfalfa/grain diet, whereas acetic, propionic, valeric, and isovaleric acid concentrations were higher in the alfalfa/grain diet group, consistent with a higher rate
of intragastric fermentation in grain-fed horses. In a subsequent analysis of these data, the presence of VFAs (butyric, propionic, and valeric acids) and low stomach pH (gastric acidity) were found to be significant predictors of ulcer severity.34

The size of grain meals may affect the extent of intragastric fermentation and thus VFA production.40 Métayer et al.40 compared gastric emptying rate in horses fed a small (300 g/100 BW) vs. large (700 g/100 kg BW) high-starch concentrate. Although the calculated rate of gastric emptying (g/min) was higher with the large meal, intragastric emptying in terms of percent of the total meal was much slower. Thus, with large starch-rich meals, intragastric fermentation and volatile fatty production may be favored because of the large load of fermentable substrate and longer residence time in the stomach.

Feeding frequency also may affect the risk of gastric squamous ulceration.41 In grazing horses, the continuous flow of saliva and ingesta may provide a buffering effect such that gastric pH remains >4.0 for most of the day. On the other hand, given that horses are continuous secretors of gastric acid, stomach pH falls when feed is withheld and the non-glandular mucosa is exposed to an acid environment. In healthy horses, squamous ulceration was induced by alternating 24-h periods of feed deprivation and ad libitum access to hay over an 8-day period.41 Ulceration developed after 24 h of cumulative feed deprivation. The median intragastric pH during a 24-h period with ad libitum access to grass hay was 3.1, whereas median pH was 1.6 during feed deprivation. These findings confirm that gastric acidity is the primary mechanism of squamous mucosal ulceration and suggest that the typical practice of twice daily meal feeding may be a contributing factor.

In summary, there is some support for the hypothesis that low forage, high grain diets are permissive to development of gastric squamous mucosal ulcers. However, further studies are needed to better elucidate the role of diet composition in ulcer development and to identify feeds and feeding methods that reduce risk of gastric ulcers. For example, it has been suggested that the feeding of lower starch, higher oil and fiber concentrates is beneficial, but this hypothesis remains unproven. Interestingly, corn oil supplementation (45 ml/day) in ponies was associated with a significant decrease in gastric acid production and increased prostaglandin E2 concentration in gastric juice.42 Recent studies have shown abundant microbial colonization of the stomach of horses,43 but the potential role of bacteria (e.g., Helicobacter pylori) in the pathogenesis of gastric ulcer disease is unknown.

5. Recommendations for Minimizing Digestive Disturbances

Feeding Frequency
In an ideal world, feeding strategies for horses kept under intensive conditions would mimic the pattern of a grazing animal, i.e., an almost continuous feeding pattern that minimizes fluctuations in the rate of delivery of substrate to the large intestine and, when forage comprises the bulk of the diet, assures some stability of the hindgut ecosystem. A more continuous feeding pattern also may minimize fluctuations in gastric acidity and therefore be of benefit in horses at risk for squamous mucosal ulcer disease. For stabled horses fed two large meals per day, foraging behavior should be encouraged by increasing the availability of hay (or even a variety of different forages44) and pasture or dry lot turnout (with forage available). Provision of more frequent (e.g., 3 times/day rather than 2 times/day), smaller concentrate meals throughout the day is also recommended to minimize delivery of undigested hydrolyzable carbohydrate to the hindgut. Extending eating time by diluting the energy density of the meal (e.g., chopped hay mixed with concentrates) or feeding forage before grain or concentrate may be helpful. For some greedy eaters, placement of several large stones in the feeder trough may slow the rate of intake.

Adequate Forage/Fiber
For hard working horses with high DE requirements, the provision of roughage is often restricted in favor of grain concentrates to ensure adequate DE intake within limits of typical dry matter consumption. However, there is considerable circumstantial evidence associating low-roughage diets with digestive disturbances (hindgut acidosis, colic, gastric ulcers) and behavioral problems. There also is evidence that the adverse effects of high starch intake on hindgut function are mitigated when the ration is at least 50% neutral detergent fiber (NDF).45 Accordingly, there is rationale for feeding programs that promote higher roughage/fiber intake. An absolute fiber requirement has not been defined, but a minimum of 1.0 kg long-stem forage per 100 kg BW (i.e., 5.0 kg for a 500-kg horse, as fed basis) has been recommended. Some nutritionists have suggested that a rate of 1.5 kg per 100 kg BW is more ideal. Alternatively, fiber intake can be increased by feeding other sources such as sugar beet pulp or soya hulls, both of which are highly digestible (i.e., the DE yield is higher compared with hay) and now commonly added to energy supplements for horses. This approach also facilitates a decrease in reliance on grain or sweet feed for energy, thereby decreasing risk of digestive disturbances associated with high starch intake.

Forage quality is another important consideration. Immature (i.e., first-cut) forages have higher DE content and digestibility compared with later cuttings and are preferred for hard working horses with high energy needs. On the other hand, the exclusive feeding of highly lignified fiber sources (e.g., straw), which are poorly degraded in the large intestine, may increase risk of impaction colic. High intake of straw is possible when it is used for...
bedding, particularly when an inadequate amount of hay (or roughage with low palatability) is offered. An increase in provision of palatable forage and/or a change in bedding can be helpful in these situations. Moldy forage should not be fed to equids.

Limiting Delivery of Rapidly Fermentable Substrate to the Hindgut

Size of Grain-Concentrate Meals
The feeding of large meals rich in starch and sugar can overwhelm the digestive capacity of the small intestine and destabilize the hindgut because of rapid fermentation of these substrates. No more than 2 g starch/kg BW should be fed in a single meal. For a grain or sweet feed mix that is 40–50% starch, this upper limit equates to ~2.0 kg per meal for a 500-kg horse.

Feed Starch Sources With High Pre-Cecal Digestibility
Pre-cecal starch digestibility varies with the type of grain and the nature of any mechanical or thermal processing. For example, whereas oat starch has a pre-cecal digestibility of ~80–90%, ~35% of equivalent doses of barley or corn starch (from unprocessed grains) reaches the cecum undigested.16,24,46 The higher pre-cecal digestibility of oat starch may relate to the small size of the starch granules compared with other grains, providing a large surface area for exposure to intestinal amylase. Milling, grinding, and various heat treatments (e.g., steam flaking, micronization, extrusion) improves the pre-cecal starch digestibility of oats, barley, and corn. In one study, the pre-ileal digestibility of ground oats was 97% compared with 83% for whole oats. Rolling or breaking did not improve the pre-ileal digestibility of oats.14 For corn and barley, pre-ileal starch digestibility is substantially increased after heat (e.g., steam-flaked corn, micronized barley) but not mechanical treatment. Overall, oats seem to be the safest source of starch for horses, although barley and corn are acceptable if they are subjected to some form of heat treatment.

Use Alternative Sources of Energy
The most obvious approach to avoidance of gastrointestinal disorders associated with grain feeding is to not feed grain! It is now apparent that energy demands of growth, lactation, and performance can be readily met by provision of alternative energy sources such as vegetable oil (fat) and non-starch carbohydrates (e.g., sugar beet pulp, soya hulls). Commercial concentrates made with these ingredients will contain varying amounts of starch and sugar, but in general amounts will be substantially lower compared with straight cereals or sweet feed mixes.

What About Pasture Forage?
An unresolved problem is management of the intake of rapidly fermentable substrate (e.g., fructan) by horses at pasture, particularly animals with a history of pasture-associated laminitis or those with recognized risk factors for this disease (i.e., obesity, insulin resistance). Recommendations for dietary management horses and ponies at high risk for pasture laminitis are provided elsewhere in these proceedings. In brief, the most obvious avoidance strategy is to prevent access to pasture and feed preserved forage with low non-structural carbohydrate content (e.g., <10–12% non-structural carbohydrates). Alternative approaches are to restrict access to pasture at certain times of the day, avoiding peaks in forage non-structural carbohydrate content that may increase risk of laminitis, or application of a grazing muzzle that limits forage intake (but allows water intake). Several factors affect the accumulation of fructans and other forms of non-structural carbohydrates in pasture plants, including plant growth rate, temperature, and light intensity. There also is marked diurnal variation with peak concentrations in the afternoon and a nadir during the night/early morning, and it has been suggested that horses grazing in the afternoon may ingest two- to four-fold higher quantities of non-structural carbohydrates compared with night or early morning grazing.45–47 These observations are the basis for the recommendation to restrict grazing to late night and early morning, with removal of the horse or pony from pasture by mid-morning.48

Gradual Dietary Changes
The increased risk of colic in the 2-wk period after a change in hay or grain feeding7,12 suggests that all changes in diet and pattern of feeding should be gradual. This will include feeding a blend of old and new hays during the transition between hay batches (e.g. over a 7-day period, with gradual increase in the proportion of the new forage) and a conservative introduction to concentrate feeding or changes in type of grain or concentrate. One suggestion is to start at ~0.5 kg/day (split into two feedings) for a 450- to 500-kg horse, with daily increments of no more than 0.5 kg until attainment of the target feeding rate.

What About Probiotics and Prebiotics?
There is considerable interest in the use of feed additives such as live yeast culture, probiotics (bacterial species), and prebiotics (e.g., short-chain fructo-oligosaccharides) as a strategy to minimize the negative effects of cereal-based diets. There is some evidence that yeast cultures might be beneficial for stabilization of the hindgut environment in the face of high cereal feeding. In horses fed high starch meals (3.4 g/kg BW/meal, such as barley), supplementation with 10 g/day of a live yeast (Saccharomyces cerevisiae) culture preparation attenuated post-feeding decreases in cecal and colonic pH and alterations in hindgut microbial populations.49 Thus, supplementation with live yeast culture may
be beneficial in horses fed a high grain ration, but from a practical standpoint, it may be more important to ensure adequate fiber in the diet, decrease the quantity of grain, and emphasize use of non-starch energy sources that do not adversely affect the hindgut environment.

Probiotics have been defined as live microorganisms that, when ingested or administered orally, provide a beneficial effect beyond that of their nutritional value. Many probiotics are marketed for use in horses, the primary rationale being the treatment or prevention of gastrointestinal diseases, for example, as an adjunct therapy in the management of acute or chronic diarrhea or prophylactic administration for prevention of colic associated with disturbances to gut microflora, e.g., for horses fed a high cereal ration. Anecdotally, there is widespread use of probiotics in horses; however, as with many nutritional supplements, there is a dearth of scientific data concerning safety and efficacy. An effective probiotic organism must be resistant to destruction by gastric acid, pancreatic secretions, and bile salts and be able to colonize the intestinal tract. Weese et al. have screened equine intestinal microflora for organisms meeting these criteria and initially identified Lactobacillus pentosus (WE7) as a potential equine probiotic. However, in a randomized controlled clinical trial of 153 neonatal foals (24–48 h old), the administration of freeze-dried L. pentosus WE7 for 7 days was significantly associated with development of signs of depression, anorexia, and colic and more days with diarrhea compared with the placebo treatment. It is possible that other probiotic organisms are effective in horses. However, as discussed by Weese and Rousseau, the use of commercial probiotics for treatment or prevention of gastrointestinal disease in horses may be ill advised in the absence of data on safety and efficacy.

References