The Role of Nutrition in Colic

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• Equine • Epidemiology • Dietary starch • Digestion • Probiotics

Equine digestive anatomy and physiology evolved over more than 50 million years for an animal that had significant behavioral and dietary dissimilarities with the modern equid domesticated over the last few millennia.[1,2] The dietary demands of modern equine activities frequently require marked quantitative and qualitative dietary changes and feeding patterns compared with the evolutionary model. The contrast is marked when considering the feral equid continually browsing for fiber-rich, low-starch grasses, sedges, and shrubs that might slowly and gradually change in quality and quantity with the seasons versus the modern competition horse that may have two or three high-starch bolus feeds daily interspersed by limited forage and the possibility of abrupt and marked dietary changes provided by inexpert caregivers. The summary of ideal feeding practice, "to efficiently supply dietary ingredients in amounts that will meet the horse's nutrient needs, while still retaining the horse's normal feeding behavior,"[2] is almost inevitably compromised under most modern management systems that frequently exceed certain needs, fall short of others, and rarely closely mimic feeding patterns of horses predomestication. The imposition of modern diets and dietary management strategies for which the equine gastrointestinal tract is not evolutionarily adapted may well lead to intolerance manifesting as colic. Many diet-related variables have subsequently been recognized as risk factors for colic, such as cereal feeding, restricted grazing, and forage quality, although abrupt dietary changes have generally been found to outweigh the negative impact of specific qualitative dietary factors.[3]

Although significant regional and population differences may occur,[4,5] typically around 5 cases of colic are expected per 100 horses each year,[3,6] representing one of the most frequent and potentially serious conditions encountered in equine practice. The welfare implications of a common, painful, and sometimes fatal condition, and inconvenient interruption of training and competition schedules and financial losses estimated 10 years ago at more than $115 million per annum in the United States,[5,6] have inevitably led to considerable epidemiologic research and advice intended to moderate the incidence and severity of this familiar equine affliction.[3,7–10] In addition to regional and population differences in horses studied,

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interpretation of epidemiologic data may be further hindered by possibly differing or opposing effects of certain epidemiologic factors on specific disease subtypes associated with colic signs. Furthermore, association of certain covariables may potentially confound results and their interpretation. For example, a particular age group of a particular breed or type of horse may undergo a particular form of exertional activity while receiving a particular diet at a particular time of year associated with a particular style of management, any or all of which may have a causal (additive or opposing) relationship with diseases manifesting with abdominal pain.

CEREALS AND STARCHES

Carbohydrates invariably represent the major digestible energy source for horses across a wide range of diets[11] and have the best documented influence on colic risk in horses.[12] Various classification systems exist for partitioning dietary carbohydrates in equine diets but the system described by Hoffman and colleagues[13] is perhaps the most relevant when considering nutrition and colic. Three major fractions of carbohydrates were described comprising hydrolysable carbohydrates (CHO-H), which are readily digestible and absorbable in the equine small intestine subject to physiologic limitations (eg, monosaccharides, disaccharides, and some oligosaccharides and starches); rapidly fermented carbohydrates (CHO-F_R), which are readily and extensively fermented by microbial populations in the hindgut (eg, fructans, pectins, β-glucans, some oligosaccharides, and resistant starches); and slowly fermented carbohydrates (CHO-F_S), which are more gradually and incompletely fermented, usually by different microbial species (eg, hemicellulose, cellulose, lignins).[13] A large, diverse, and dominant population of fibrolytic bacteria (eg, *Clostridiaceae*, *Fibrobacter*, *Spirochaetaceae*) are normally found in the forage-fed equine hindgut that ferment CHO-F_S to short-chain fatty acids primarily comprising acetate, propionate, and butyrate.[14,15] A smaller population of saccharolytic species (eg, *Bacillus*, *Lactobacillus*, *Streptococcus*) also exists that hydrolyze CHO-F_R, along with any CHO-H that has escaped small intestinal digestion, primarily producing lactate and propionate.[12–14] The short-chain fatty acids absorbed from the large bowel contribute the major source of energy to forage-fed horses with acetate representing the largest individual substrate for oxidation.[11] Propionate is largely used for hepatic gluconeogenesis and butyrate may have a more local role in maintaining colonic enterocyte health.[11] Lactate is normally found at very low levels within colonic contents and, when present in larger concentrations, is associated with adverse effects on pH, microbial populations, and short-chain fatty acid absorption by the colon.[12]

Microbial population dynamics are markedly influenced by available dietary substrates and also pH changes consequent to their fermentation. Cecocolonic delivery of undigested CHO-H and CHO-F_R rapidly promotes bacterial multiplication and overgrowth of acidophilic *Streptococci* and *Lactobacilli*. Lactic acidosis decreases luminal pH from nearly neutral to as low as 6.0, markedly impairing survival of normal fibrolytic bacterial species and potentially leading to decreased CHO-F_S fermentation; decreased acetate production; impairment of mucosal barrier function; increased absorption of lipopolysaccharide, amines, and other noxious substances; dehydration of digesta; dysmotility; gaseous distention; colon displacement; and volvulus.[12,14,16–26] Interestingly, intraluminal colonic lactic acidosis, decreased fibrolytic bacteria, and increased acidophilic microbial species have been demonstrated not only in response to cereal feeding of horses, but have also been found within the large intestinal contents of horses with colic.[12]

Incomplete prececal digestion of starch is among the most important causal factors to consider in the association between diet and colic. Starches from different cereal sources have different proportions of straight-chain amylose and branched-chain amylopectin and differing relative digestibility. Oat and sorghum starch is more highly digestible than that in barley, wheat, or maize, although thermal processing techniques, such as micronization and popping, may also have a marked further beneficial influence.[27,28] Starch digestion occurs in two phases. First, α-amylase hydrolyzes amylose and amylopectin to disaccharides (maltose), trisaccharides (maltotriose), and larger oligosaccharides (α-dextrins), which are then further degraded by brush border enzymes.[29] Compared with other species the equine small intestine may be somewhat deficient in α-amylase, although it seems to possess good levels of disaccharidases.[30,31] The absorptive capacity of glucose by the equine small intestine improves following adaptation to high-starch feeds by up-regulation of the enterocyte glucose transporter protein sodium-glucose cotransporter isoform 1.[12] Because it is believed that sodium-glucose cotransporter isoform 1 up-regulation is dependent on exposure to monosaccharides rather than polysaccharides, this implies that starch digestion is indeed enhanced following dietary adaptation in horses as suggested by previous studies.[27] With relevance to colic in horses, however, there seems to be significant interindividual variation in α-amylase levels and it seems that adaptation is both limited and slow to develop.[12,32] Accordingly, incomplete prececal starch digestion is likely to occur in most horses fed to perform frequent and intense bouts of exercise. A study of racing Thoroughbreds in Australia reported a mean cereal intake of 7.3 kg/day (range, 3.8–13.2 kg/day),[33] which even when divided into several feeds is still expected to result in around half of the ingested starch arriving in the cecum.[34] Fecal pH was less than 6.2 in more than a quarter of horses in this study[33] consistent with a lack of adaptation to the diet and probable adverse cecocolonic health and function, as described previously.

The combination of relatively poor prececal starch digestibility and the dysfermentative effects of starches delivered to the large bowel suggest that equids are poorly suited to high-starch diets as frequently provided to competition horses.[12,34] Unsurprisingly, several studies have associated increased cereal feeding with colic risk. In one study comparing cereal-fed horses with grazing horses, those consuming only moderate quantities of concentrates (2.5–5 kg/day) were found to have an almost five times increased risk of colic (odds ratio [OR], 4.8; 95% confidence interval [CI], 1.4–16.6; $P = .01$) and the risk was increased further at higher levels of concentrate feeding (>5 kg/day: OR, 6.3; 95% CI, 1.8–22; $P<.01$).[35] Similarly, an approximately six times increased risk of colic was found in horses consuming more than 2.7 kg oats per day in another study (OR, 5.9; 95% CI, 1.6–22; $P<.01$).[36] A recent study of impaction colic in donkeys indicated that the risk of colic was more than doubled in those donkeys fed extra concentrate rations (OR, 2.2; 95% CI, 1.1–4.5; $P = .03$).[37] Pelleted feeds have been proposed to represent a particular colic risk in some studies,[7,35] although this has not been a consistent finding in all reports.[8,38]

Cereal feeding may have different effects on different causes of colic. One study found that concentrate intakes were significantly higher in horses diagnosed with duodenitis-proximal jejunitis than those with colic from other causes.[39] In contrast, enterolithiasis is one cause of colic that might actually benefit from modest colonic acidification because formation of enteroliths is suspected to be favored in an alkaline colonic environment enriched by protein-derived nitrogen and sulfur and also in minerals including calcium, magnesium, phosphorus, potassium, and sodium.[40–42]

Given the potential consequences of the limited capacity of the equine small intestine to hydrolyze starches, the practice of dividing cereal-based feeds into frequent,

small meals is generally recommended as far as is reasonably practical.[43] Starch boluses fed at greater than 300 g/100 kg bodyweight have a marked effect on microbial populations with perhaps half of the ingested starch reaching the cecum,[23,28,34] and a guideline of no more than 200 g/100 kg bodyweight starch per meal (eg, approximately 2 kg of concentrate feed for a 500-kg horse) is frequently proposed.[24,28,44,45] In horses prone to colic, far lower starch boluses may be advisable because even when fed at 100 to 200 g/100 kg bodyweight per meal there is still frequently 40 g starch/100 kg bodyweight reaching the cecum.[28,34] The possible benefits of exogenous α-amylase added to high-starch equine diets is worthy of further investigation. In one study an increased glycemic response was seen in horses following incorporation of α-amylase (with or without further enzymes) to a high-starch diet suggesting increased prececal hydrolysis.[31] This effect was short-lived, however, and was not associated with significantly improved fecal pH or short-chain fatty acid concentrations as might have been expected.

When it is considered unlikely that a grass-forage diet modestly supplemented by cereal can provide enough energy for a working horse, then fiber-rich feeds, such as sugar beet pulp or alfalfa cubes, might be provided that contain similar digestible energy content to many cereal-based feeds but far less starch. Fat may then be supplemented to increase the energy density of the diet without the undesirable consequences of starch on the hindgut. Although vegetable oils have also been found to adversely affect cellulolysis in the equine hindgut, presumably as a consequence of bactericidal fatty acids,[46] this effect is far less disruptive than similar energy levels provided by starch-rich feeds and fat feeding has not yet been associated with colic in the horse.

GRAZING

Leguminous and subtropical grasses may store significant quantities of starch in their leaves in contrast to most temperate pasture grasses that store carbohydrate as fructans in the stems.[47] Although it is recognized that there is some prececal hydrolysis and fermentation of fructans,[11,47] this is quite limited compared with starches and fructans fit mainly into the CHO-F_R category.[13] Logical extrapolation of the dysfermentative effects of excess dietary starch might then lead to the assumption that grass might also contribute to colic risk in the horse, although additional important factors to consider are possibly differing rates of exposure of grass-fructan (or grass-starch) versus cereal-starch to the hindgut bacteria and also stable adaptation of fermentative bacterial populations to a particular continuous carbohydrate source. There is a marked contrast in probable bacterial population dynamics between trickle-feeding grass-fructans (or grass-starches) over perhaps 14 to 18 hours of grazing daily compared with bolus-feeding of concentrated cereal-starch in two meals perhaps lasting 10 minutes each during a 24-hour period. Nevertheless fructan can be consumed rapidly and in large quantities under certain climatic and management conditions leading to similar dysfermentative effects to undigested starch that reaches the cecum.[47]

Access to grazing has generally been regarded as offering some protection against colic, although this observation might be biased or confounded by associated factors, such as reduced observation of pastured horses, increased ambulatory activity, psychologic comfort, or climatic, seasonal, or other factors associated with a management decision to increase stabling. Nevertheless, a large study in Texas found that horses that were fully stabled or had recently reduced pasture access were at three times the risk of suffering colic (95% CI, 1.4–6.6; $P<.01$) after controlling for many other

variables.[36] An even greater protective effect of grazing has also been proposed for specific colic subtypes, such as simple colon obstruction; epiploic foramen entrapment (EFE); and enterolithiasis cases, which are far more frequently reported in horses that spend little or no time at grass.[10,41,42,48,49] Although it might be argued that simple colon obstructions arise following stabling because of several other associated factors, such as the introduction of relatively dry forage rather than the withdrawal of grazing per se, multivariable analysis confirms the protective effect of access to pasture against enterolith formation and EFE even after controlling for alternative preserved forage intake.[42,49]

That is not to say, however, that grazing horses are at low risk of all types of colic. Fructan content of grasses and herbage can be markedly variable between different grasses, exacerbated by variable temperature and light exposure,[47,50] creating an increased likelihood of hindgut disturbance when grazing is changed or weather is changeable. Interestingly, equine grass sickness (EGS) and duodenitis-proximal jejunitis, two specific causes of colic that are unusual in being associated with increased grazing,[39,51,52] have both been etiologically linked with putatively altered enteric toxicoinfectious clostridial populations,[53,54] although the precise causal factors in these conditions remain uncertain. The further association between weather, season, and recent change of fields with risk of EGS also is consistent with a possible dietary influence on enteric bacterial populations.[51,52]

Grazing should generally be encouraged in preference to preserved forage for the benefit of gastrointestinal health but consideration should be given to introducing access to new grazing gradually and to allowing free access eventually to limit variable rates of consumption. Clearly, the balancing of other health and management issues is also a key to successful grazing policy and this has to be finally formulated on the basis of local conditions with respect to the quality and quantity of pasture and the characteristics of the individual horses concerned. Obese, laminitis-susceptible individuals or those considered to be at high risk of EGS, sand impactions, or duodenitis-proximal jejunitis may gain net health benefit from grazing restriction.

PRESERVED FORAGE

When grazing is restricted by availability or management requirements, then preserved forages should be regarded as the staple of equine diets. The quality within batches of preserved forage is likely to be less variable than grazing, although abrupt changes in forage type or even a change in batch of the same type of preserved forage still carries a significant risk of colic.[8,10,36] Particular types of forages have been found to be risk factors for particular types of colic. Poor quality hays that are especially high in less fermentable components of CHO-F_S increase the risk of ileal and colonic impactions,[36,38,55] whereas alfalfa hays higher in crude protein and minerals are associated with increased risk of enteroliths (alfalfa hay $\geq$50% of diet: OR, 4.7; 95% CI, 1.4–15.6; $P = .01$)[41,42] and fecoliths.[7] Colonic alkalinization resulting from alfalfa feeding has long been suspected to explain the significant association with enterolith formation exacerbated by the relatively high mineral content of alfalfa hay.[40] In contrast, oat and grass hays have been shown to reduce the risk of enterolith formation significantly over and above the associated benefit of decreased alfalfa feeding (oat hay $\geq$50% of diet: OR, 0.20; 95% CI, 0.07–0.62; $P<.01$; grass hay $\geq$50% of diet: OR, 0.22; 95% CI, 0.08–0.61; $P<.01$).[42] The buffering effect of the increased quantities of saliva consumed with prolonged mastication of preserved forage perhaps augmented by the greater buffering properties of alfalfa hays might also offer benefits to the health of the proximal gastrointestinal tract and may partly explain

apparent protective effects against gastric ulceration and small intestinal strangulations.[7,56,57]

There is little evidence on which to base recommendations for fiber feeding in horses.[11] Where grazing is limited, a minimum of 1% to 1.5% bodyweight of preserved forage (as dry matter) is advisable.[2] Following gradual access to new forage supplies, free access should be considered in preference to intermittent provision of forage, and physical limitation of ingestion with narrow-weave haynets, double haynets, or ''haybags'' may be useful to simulate a natural trickle-feeding behavior.

Good dental care intuitively forms an integrated part of the dietary strategy offered to the horse with recurrent colic, although studies have not consistently found any beneficial effect of routine dental care on digestion, assimilation, or colic risk.[10,58–61] A recent study in donkeys with impaction colic, however, did identify dental disease as a very strong and highly significant risk factor for disease (OR, 29.7; 95% CI, 4–223.7; $P<.01$)[37] and dental examination and appropriate corrections seems to be advisable when presented with a case of impaction or recurrent colic.

DIETARY CHANGES

Whatever the exact dietary quality and quantity supplied to a horse, it seems that recent changes in diet represent an especially high risk for colic of various types,[8,10,35,36,49,51,55,58,62] an association that is most probably mediated by altered diet-adapted microbial populations. In an experimental study conducted in ponies, significant changes in hindgut bacterial flora and short-chain fatty acids concentrations promptly followed addition of 30% rolled barley to a hay-only diet.[18] Changes in diet have also been found to predispose to *Salmonella* shedding in hospitalized horses, further emphasizing a destabilizing effect on intestinal microflora.[63] In addition to the markedly increased risk of EGS in horses that have recently changed grazing, other studies have identified risk factors for general colic incidence including changing the batch or type of hay, changing the quantity or frequency of feeding, and erring from usual feeding times.[8,35,36] Although it is well accepted that abrupt dietary changes may lead to colic in horses, the common practice of feeding a few daily cereal-based meals interspersed by long periods of high-fiber forage intake (or no intake) of markedly differing nutritional quality represents marked intraday dietary change and probable significant diurnal variability in hindgut pH and bacterial populations and perhaps promotion of colic.[45]

The duration of the colic risk following dietary change varies between types of colic. In a study of horses with colic caused by simple colon obstruction and distention,[10] the risks of colic were greatest within a week of a change in forage (OR, 22; 95% CI, 2.8–170.4; $P<.01$) or concentrate (OR, 12; 95% CI, 2.7–54.4; $P<.01$), with the risks being reduced but still significant between 8 and 15 days of a dietary change (forage change OR, 4.9; 95% CI, 1.3–18.6; $P = .02$; concentrate change OR, 3; 95% CI, 1.0–8.9; $P<.05$). Diet changes more than 15 days previously were not significantly associated with colic risk.[10] A 2-week duration of colic risk following dietary change is also supported by several further studies of general colic incidence,[8,36,55,58] although other reports examining the temporal relationship between dietary change and risk of specific types of colic indicate that significantly increased risk of disease may exist for even longer after the change of diet. The risk of EFE was significantly increased for a period of 28 days following increased stabling (OR, 3.7; 95% CI, 1.4–9.7; $P<.01$).[49] Although the risk of EGS was found to be greatest within 2 weeks of a change of grazing (OR, 29.7; 95% CI, 66.7–130; $P<.01$), the increased risk of disease was still significant as long as 2 to 3 months following a field change (OR, 4.1; 95% CI,

1.0–16.6; $P<.05$).[51] Interestingly, several studies have found that a recent change in hay or forage is associated with higher risk of colic than a recent change in grain or concentrate.[8,10,36]

FURTHER FEED ADDITIVES

Many prebiotic and probiotic products are marketed for horses with gastrointestinal diseases and may have a reasonable evidence basis in some other species. The quality of commercially available probiotic products was scrutinized and questioned in one study that found that most products contained few, if any, viable or potentially beneficial organisms and sometimes potential pathogens were encountered.[64] In a further examination of a commercially available equine probiotic product claiming to contain lactobacilli, a pure growth of *Enterococcus gallinarum*, an occasional cause of antibiotic-resistant nosocomial infections in humans was obtained (A.E. Durham, unpublished data, 2008). An evidence-based approach to the design of appropriate equine bacterial probiotic products has thus far been unsuccessful.[65,66] In contrast, the investigation of yeast-containing probiotics including *Saccharomyces cerevisiae* and *Saccharomyces boulardii* has generally provided better quality supportive evidence in horses with gastrointestinal disease and they have been shown to protect against the adverse effects of starch overload[23,67] and also to reduce significantly the duration of diarrhea in clinical enterocolitis cases.[68]

In a recent study of risk factors for EFE it was found after multivariable analysis that access to a mineral-salt lick significantly reduced the risk of disease (OR, 0.3; 95% CI, 0.1–0.9; $P = .03$).[49] Given the triangular association between EFE, stereotypical behaviors, and gastrointestinal pH, the authors speculated that access to the salt lick stimulated increased salivation and the consequent gastrointestinal buffer perhaps offered some protection against EFE. Enterolithiasis, however, a condition that is unlikely to benefit from increased salivation (because colonic alkalinity seems to be a risk factor),[40] has also been found to be less likely in horses that are supplemented with minerals or vitamins (including mineral-salt licks or in-feed supplementation), although this finding did not reach statistical significance (enterolithiasis: OR, 0.5; 95% CI, 0.2–1.2; $P = .11$).[42] In the latter study, a further currently unexplained finding was that the only specific dietary variable other than alfalfa feeding that was associated with increased risk of enterolithiasis was the feeding of carrots (OR, 2.7; 95% CI, 1.0–7.6; $P = .05$).[42]

Psyllium is often fed to horses considered to be affected by or at risk from sand enteropathy.[69–71] One experimental study found that fecal output of sand that had been deposited surgically within the cecum of healthy ponies was not increased by 1 g/kg bodyweight psyllium when compared with an untreated control group.[72] Another study also failed to detect a beneficial effect of 0.5 g/kg bodyweight psyllium given daily when measuring fecal sand output following nasogastric dosing with sand.[73] In contrast, two more recent studies have provided support for the clinical use of psyllium in sand enteropathy cases. One study administered 1 kg of sand to healthy horses by nasogastric tube daily for 5 days and then administered either 1 g/kg psyllium combined with 2 L mineral oil, or 2 L mineral oil alone, from days 6 to 10. Fecal sand output was significantly greater in the group receiving the combined treatment from days 2 to 4 of administration.[74] A further study of a group of healthy horses with access to dry pasture or feedlot monitored fecal sand output for 7 days before and 35 days during administration of a psyllium-prebiotic-probiotic mixture containing approximately 0.5 g/kg bodyweight psyllium daily. Fecal sand output

was significantly increased from 4 days after commencing treatment until the end of the study.[75]

SUMMARY

Equine gastrointestinal health seems best served by a slow and constant intake of a high-fiber, low-starch diet of even quality that maintains stability in fermentative fibrolytic bacterial species in the hindgut. Disruption and adverse dynamism of microbial populations may be especially promoted by dietary change when major feed ingredients are replaced or simply provided intermittently through the day. The demands of competition horses in particular often dictate that forage availability is restricted, energy-dense high-starch or high-fat boluses are ingested intermittently, and abnormally prolonged periods of total feed deprivation are common. The relative rarity of colic in domesticated horses (typically 95 in every 100 do not suffer colic during the course of a year) is a tribute to the adaptive capability of the equine gastrointestinal tract to the imposition of a diet that is frequently alien in respect of quality, quantity, and patterns of ingestion. Those individuals that do suffer from colic, however, especially if on a recurrent basis, should have their diet reviewed and adjusted toward that of their feral ancestors wherever and as far as possible.

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