

## **Recent Advances in Carbohydrate Nutrition for Horses**

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The horse evolved primarily as a grazing and browsing, hind-gut fermenting herbivore, with a range of forage carbohydrates—hydrolyzable to fermentable—as its main source of energy. Pastures provide the main habitat and nutrition for most horses, and the remaining stall-confined horses have at least one-half of their nutrition supplied by conserved pasture, i.e. hay. Horse owners supplement a diet of pasture and hay with grain concentrates in order to meet energy demands of performance and to provide a carrier for micronutrients that are marginal or deficient in forages. Common experience has been supported by epidemiological and experimental studies that associate grain concentrates with several digestive and metabolic disorders, including colic (Clarke et al., 1990; Hudson et al., 2001), laminitis (Pass et al., 1998), gastric ulcers (Murray, 1994), developmental orthopedic disease (Kronfeld et al., 1990; Ralston, 1996), insulin resistance (Hoffman et al., 2003a; Treiber et al., 2005) and some forms of exertional rhabdomyolysis (Valentine et al., 2001). The abundant starch in grain concentrates has been implicated as the culprit, leading to development and marketing of “low starch” concentrates for horses. Corresponding trends in human nutrition towards “low carb diets” have fed wide consumer support of low starch feeds for horses, perhaps to excess.

While low starch grain concentrates provide an alternative energy source that is critical for horses with a history of digestive and metabolic disorders that are sensitive to starch, these concentrates are not a “one fits all” solution. Specifically, exercising horses require some dietary starch in order to appropriately fuel performance. Horses have an opportunity for small intestinal metabolism of starch and simple carbohydrates to glucose, which is more metabolically efficient than hindgut fermentation of fibers to volatile fatty acids. Compared to fatty acids, glucose (or its stored form, glycogen) is aerobically metabolized twice as fast to generate ATP for muscle contraction. As speed and exertion increases to the point of anaerobic work, glycogen is metabolically favored over fatty acids.

This paper will summarize carbohydrate nutrition for horses, beginning with digestion and absorption of hydrolyzable and fermentable fractions. The physical capacity of preileal starch digestibility and factors contributing to rapid fermentation, including fructans in the equine hind gut will be covered. Physiological effects of feed carbohydrates on glycemic response, microbial populations and acid production will be considered with respect to carbohydrate components in forages, grains and super fibers.

## Carbohydrate Digestion

Carbohydrates may be hydrolyzed or fermented in horses, depending on the linkage of their sugar molecules: carbohydrates with  $\alpha$ -1,4 linked molecules are subject to enzymatic hydrolysis, while  $\beta$ -1,4 linked molecules must be fermented. Hydrolyzable carbohydrates include disaccharides, some oligosaccharides (e.g. maltotriose) and starch. Fermentable carbohydrates include soluble fibers (e.g. gums, mucilages, pectins), some oligosaccharides (e.g. fructans, galactans), starches resistant to enzymatic hydrolysis, hemicellulose, cellulose, and lignocellulose.

### *Hydrolytic Digestion.*

Enzymes secreted in the small intestine specific to carbohydrate hydrolysis include  $\alpha$ -amylase,  $\alpha$ -glucosidases (sucrase, glucoamylase, maltase), and  $\beta$ -galactosidase (lactase). Relatively little  $\alpha$ -amylase is present in equine saliva, so limited hydrolysis occurs prior to arrival of carbohydrates in the stomach. In the stomach, gastric acid hydrolyzes carbohydrates to an extent, independent of enzymes.

In the small intestine, hydrolysis of carbohydrates is initiated primarily by pancreatic  $\alpha$ -amylase. In the luminal phase,  $\alpha$ -amylase cleaves  $\alpha$ -1,4 linkages but not  $\alpha$ -1,6 or terminal  $\alpha$ -1,4 linkages of starch molecules. Amylopectinase cleaves  $\alpha$ -1,6 linkages. The end products of the luminal phase are disaccharides and oligosaccharides—no free sugars are yielded. Sucrase, lactase and maltase are expressed along the length of the equine small intestine at the brush border mucosal cells (Dyer et al., 2002). Sucrase activity was higher in the duodenum and jejunum than the ileum, while maltase activity was similar in duodenum, jejunum and ileum (Dyer et al., 2002). Functional lactase was present in all portions of the small intestine of mature horses, higher in the duodenum and jejunum than the ileum. Although its activity was lower in mature than weaned horses, the presence of functional lactase suggests that mature horses can digest lactose (Dyer et al., 2002). The action of these disaccharidases at the brush border mucosal cells completes hydrolysis to yield free sugars, glucose, galactose and fructose.

### *Fermentation.*

Fermentation occurs predominantly in the hind gut of horses but may occur in any area of the digestive tract where microorganism populations are sufficiently established as a result of favorable conditions, such as adequate retention time and pH greater than 5 (Van Soest, 1994). The presence of viable anaerobic bacteria as well as acetate, propionate, butyrate and lactate suggests that limited fermentation occurs in the equine stomach, particularly in the fundic region (Kern et al., 1974) and favors lactic acid (Argenzio et al., 1974). The brief retention time in the stomach and the dorsal to ventral pH gradient of the gastric mucosa likely supports only nominal fermentation (Murray and Grodinsky, 1989). Some fermentation occurs in the small intestine of horses (Zentek et al., 1992; Moore-Colyer et al., 2002), but it is not well known if small intestinal fermentation occurs independent of large bowel fermentation or is merely due to reflux of large bowel contents. Fermentative gases in breath exhalation indicate that microbial

fermentation in the stomach and small intestine partially degrades starch and fructans, but not pectin and cellulose (Coenen et al., 2006).

Carbohydrates fermented by intestinal microflora yield volatile fatty acids, mainly acetate, propionate, butyrate, and to a lesser extent, lactate and valerate. The relative proportions in volatile fatty acids produced are dependent on substrates, i.e. the proportions of dietary forage and concentrate (Hintz et al., 1971; Longland et al., 1997; de Fombelle et al., 2001). Increasing proportions of grain favored production of propionate and lactate at the expense of acetate (Hintz et al., 1971; Willard et al., 1977; de Fombelle et al., 2001). Feeding higher percentages of grain depressed the efficiency of fiber utilization by altering the microbial ecosystem in the equine cecum and colon (de Fombelle et al., 2001). Rapid fermentation favors proliferation of *Lactobacilli* spp and production of lactate, which is poorly absorbed (Argenzio et al., 1974; Garner et al., 1978).

### **Carbohydrate Absorption**

Two classes of glucose carrier proteins have been identified in mammalian cells (Shirazi-Beechey, 1995): the high affinity, low capacity, Na<sup>+</sup>/glucose cotransporter type I (SGLT1) and facilitative glucose transporters (GLUT). The SGLT1 is present on the intestinal luminal membrane and in kidney proximal tubule absorptive epithelial cells. It transports primarily D-glucose and D-galactose across the brush border membrane against the concentration gradient by active transport of Na<sup>+</sup> and the Na<sup>+</sup>/K<sup>+</sup>-ATPase (Dyer et al., 2002). The sugars accumulate within the enterocytes and are transported down gradient into systemic circulation via GLUT (Joost and Thorens, 2001). The major site of glucose absorption in horses is the proximal small intestine, with glucose transport highest in the duodenum, followed by jejunum and ileum (Dyer et al., 2002).

The lag time between an abrupt change in dietary hydrolyzable carbohydrate and the appearance of enhanced SGLT1 was 12 to 24 h in mice (Ferraris and Diamond, 1993). Equine SGLT1 has 85% homology with mouse SGLT1 and 92% similarity at the amino acid level (Dyer et al., 2002). In mice, dietary regulation of glucose transport involves increased transcription of SGLT1, mainly in crypt cells (Ferraris and Diamond, 1993). Comparatively in horses, expression of SGLT1 is regulated at the level of mRNA abundance (Dyer et al., 2002). The differences in length and function of horse and mouse digestive tracts may play a role in appearance of SGLT1 after changes in dietary hydrolyzable carbohydrate, so direct comparisons should be considered with caution. However, if a similar lag time for SGLT1 exists in horse, then in the event of an abrupt change in diet, sugar transport would be inadequate, thus exacerbating hydrolyzable carbohydrate overload to the hind gut.

Volatile fatty acids are absorbed down the transmucosal pH gradient across the large intestinal wall by passive diffusion, primarily in the form of free acids. The rate of absorption is inversely proportional to molecular weight, with absorption of acetate > propionate > butyrate > lactate (Argenzio et al., 1974). The absorption of volatile fatty

acids is integral to maintaining the pH of the colon above 6, which is required for optimal balance of bacterial populations (Radicke et al., 1991; Van Soest, 1994).

## **Equine Disorders Associated with Carbohydrates**

### *Carbohydrate Overload.*

Sugars and starches are hydrolyzed in the equine small intestine up to the point at which the enzymatic capacity becomes overloaded, and the excess is rapidly fermented in the hind gut. The critical capacity for starch overload appears to be in the range of 0.35 to 0.4% of body weight per feeding (Potter et al., 1992), but may be as little as 0.2%, depending on the source of starch (Radicke et al., 1991; Kienzle et al., 1992). Prececal digestion of corn starch increased from an intake of 0.1% to peak at approximately 0.35% of body weight, then decreased at starch intakes above 0.4% of body weight (Potter et al., 1992). Similarly, the presence of ileal starch remained at a plateau from intakes of 0.1% to approximately 0.25% of body weight then increased exponentially at intakes above 0.25% of body weight. Compared to oat starch, feeding corn starch resulted in lower cecal pH at all levels of starch intake (from 0.1% to 0.4%), and differences in cecal pH between the starch sources increased in proportion to starch intake (Radicke et al., 1991). Accumulation of lactic acid may overpower the buffering mechanism of the hind gut and lower pH, which under normal conditions, is 6.4 to 6.7 in grazing horses. A cecal pH of 6 was considered to represent sub-clinical acidosis (Radicke et al., 1991). A pH less than 6 favors production of lactic acid (Garner et al., 1978; Van Soest, 1994) and was associated with clinical conditions such as osmotic diarrhea, overgrowth of undesired bacterial populations and lysis of desired bacterial populations, thus increasing the risk of endotoxemia and laminitis (Sprouse et al., 1987; Bailey et al., 2003).

Aside from the rapid fermentation of excess hydrolyzable carbohydrates, other rapidly fermentable carbohydrates include resistant starches and oligosaccharides, especially fructans, which may comprise 5 to 50% of the dry matter in cool season grasses (Longland et al., 1999; Cuddeford, 2001). The  $\beta$ -2,6 glycosidic bonds in fructans are not hydrolyzed in mammalian small intestine, although they may be partially degraded by small intestinal microbes (Coenen et al., 2006). Fructans were used to initiate equine carbohydrate overload and laminitis (Pollitt et al., 2003; van Eps and Pollitt, 2006) and produced a more rapid fall in cecal pH than an equal amount of corn starch (Bailey et al., 2002).

### *Insulin Resistance.*

Insulin resistance has been generally defined as a abnormal metabolic state when normal concentrations of circulating insulin fail to elicit a normal physiologic response in target tissues (Kahn, 1978). More specifically, cells in muscle, adipose tissue and liver that become insulin resistant require larger concentrations of circulating insulin to stimulate glucose uptake. In humans, insulin resistance is fundamental in the pathology of type II diabetes and is a risk factor in obesity (Frayn, 2001), cardiovascular disease and hypertension (Reaven, 1988), polycystic ovaries (Legro et al., 1998; Legro, 2002),

pregnancy loss (Craig et al., 2002) and colorectal cancer (Kim, 1998; Sturmer et al., 2006).

Diets rich in simple sugars have been associated with insulin resistance in several animal and human studies (Storlien et al., 2000; Bessesen, 2001), and the common practice feeding starch-rich cereal grains with high glycemic indices may promote insulin resistance in horses (Hoffman et al., 2003a; Treiber et al., 2005). Insulin resistance has been observed in obese (Hoffman et al., 2003a, Frank et al., 2006) and sedentary (Powell et al., 2002) horses. Similar to humans, mares became insulin resistant during late pregnancy and recovered to normal sensitivity during early lactation (Hoffman et al., 2003b; Hoffman, unpublished data). Insulin resistance may be a risk factor in horses with hyperlipaemia (Jeffcott and Field, 1985; Jeffcott et al., 1986), colic (Hudson et al., 2001), laminitis (Pass et al., 1998, Treiber et al., 2006, Hoffman, unpublished data), osteochondrosis (Ralston, 1996), Cushing's disease (Garcia and Beech, 1986; Johnson, 2003), and some types of exertional rhabdomyolysis (Valentine et al., 2001). Dietary therapy alone may not be sufficient to reverse insulin resistance (Hoffman et al., 2003; Frank et al., 2005). Exercise is beneficial, as both obese and lean mares had improved insulin sensitivity after seven days of moderate exercise training (Powell et al., 2002). In human athletes, insulin sensitivity was positively correlated with maximal aerobic capacity ( $VO_{2max}$ ) and proportion of type I muscle fibers (Goedecke et al., 2001).

## **Carbohydrates in Horse Forages and Feeds**

### *Forages.*

During photosynthesis, green plants produce glucose and other simple sugars, with oxygen as a by-product, from water and atmospheric carbon dioxide in the presence of light:



When the production of sugars exceeds the energy requirements of the plant, they are converted to storage carbohydrates, most commonly starch or fructans. Cool season pasture grasses accumulate fructans, while warm season grasses and legumes accumulate starch. The accumulation of storage carbohydrates in plants is affected by temperature, light intensity and plant growth rate (Longland et al., 1999; Hoffman et al., 2001). While plants that accumulate starch are limited to maximum storage when their chloroplasts are saturated, plants that accumulate fructans have no self-limiting mechanism, so high concentrations may accumulate.

Abrupt changes in fructan concentrations were observed from day to day in rapidly growing pastures and diurnally as plant composition changed from night to day or from shade to sunlight (Longland et al., 1999; Longland and Byrd, 2006). Fructan concentrations usually rose during the morning, peaked in the afternoon, and declined to a low overnight until the early morning hours. Horses grazing in the afternoon, as compared to morning, may ingest between two to four times as much fructans (Longland et al., 1999).

An association between an abrupt increase in pasture plant fructans and the incidence of laminitis has been suggested. Laminitis has been clinically induced with 3.75 kg of fructan (Pollit et al., 2003; van Eps and Pollit, 2006), thus establishing a link between pasture fructans and laminitis. Considering pasture intake and cool season pasture fructan concentrations, a horse grazing in the summer potentially could ingest 5 kg or more of fructans per day (Longland et al., 1999; Longland and Byrd, 2006). Although the amount of fructans ingested while grazing can be as much as that used to clinically induce laminitis, it is relevant to consider that the gradual dose encountered over time during grazing likely has a far different impact than the entire dose in a single bolus during clinical induction of laminitis.

### *Grains.*

Oat starch appears to be the most digestible of the cereal starches fed to horses, followed by sorghum starch and barley starch (Kienzle et al., 1992; Meyer et al., 1993). Starch digestion is impeded when the physical form of the food limits contact with pancreatic amylase, so the higher digestibility of oat starch over other grain starches may be explained on the basis of its relatively small starch granules. Digestion is hindered when starch is contained within whole grain or waxy seed coats, such as rice or corn, or entrapped within rigid cell walls that hinder swelling and dispersion of the starch, such as soybeans. Milling and grinding increased susceptibility to hydrolysis *in vitro* by breaking seed coats and cell walls, and abrading the microscopic surface of the starch, which is naturally smooth (Gallant et al., 1992). Preileal digestibility of starch was improved in horses when oats or corn were ground, but rolling and breaking did not improve preileal digestibility over that of whole grain (Kienzle et al., 1992). Processing of corn improved preileal digestibility: compared to whole or crushed corn at 30%, grinding improved preileal digestibility to 51% and popping to 90% (Meyer et al., 1995).

### *“Super Fibers.”*

So called “super fibers” are those feeds that contain a large component of digestible fiber and low lignin, hence more fiber available for microbial digestion. In the horse industry, this class of feeds most commonly includes beet pulp, rice bran, soybean hulls, almond hulls, oat hulls, and sometimes includes rice by-product, cereal by-product and citrus pulp. Super fibers are purported to contain digestible energy equivalent to oats and barley while not producing symptoms of grain overload.

Table 1. A comparison of digestible energy (mean  $\pm$  SE) in equine forages and feeds (Hoffman, 2004).

<b>Feed</b>	<b>DE, Mcal/kg</b>
Hay	2.15 $\pm$ 0.14 <sup>c</sup>
Pasture	2.49 $\pm$ 0.13 <sup>bc</sup>
Super fibers	2.62 $\pm$ 0.14 <sup>b</sup>
Grains	3.71 $\pm$ 0.14 <sup>a</sup>

<sup>a,b</sup> Means with different superscripts differ,  $P < 0.0001$ .

<sup>b,c</sup> Means with different superscripts differ,  $P = 0.028$ .

Of the super fibers individually, mean DE ranged from 1.52 Mcal/kg for oat hulls to 3.02 Mcal/kg for rice bran, while in comparison, DE of oats and barley was 3.37 and 3.66, respectively. In some super fibers, sugar and starch concentrations approach that of oats (Equi-Analytical Forage Library, <http://www.equi-analytical.com>), so it would seem that grain overload could be a possibility if overfed.

## **Glycemic Index**

The glycemic index is a reflection of plasma glucose and insulin responses to a meal, an *in vivo* estimate, rather than a chemical analysis of the hydrolyzable carbohydrates in a feed. The glycemic index provides information about the food but not necessarily the animal. It has been applied primarily in human nutrition for diabetics in order to formulate diets with a low glycemic impact, with glycemic index calculated as a percentage of the response to a standardized reference: an oral glucose dose or white bread (Jenkins et al., 1981; Englyst et al., 1996; Wolever and Mehling, 2002). In horse nutrition, meal-related responses of blood glucose and insulin to different diets have been quantified in several reports (Stull and Rodiek, 1988; Rodiek et al., 1991; Pagan et al., 1999; Williams et al., 2001). Most studies compared ingestion of different feeds as either equal-weight or isocaloric meals and did not calculate glycemic index as a percentage of a standardized reference. More recently, glycemic indices were quantified in a series of studies by Rodiek (2003) and reported using whole oats as a standardized reference feed. This work is summarized in Table 2. Several factors may affect glycemic response including meal size, amount of hydrolyzable carbohydrates in the meals, fiber and fat content of the feed, processing, intake time, gastric emptying, digestibility and rate of absorption. One such example (Hoekstra et al., 1999), the effect of processing on the glycemic index of corn (reported relative to oats) is also shown in Table 2.

Table 2. Glycemic index relative to whole oats in different feeds and with varying degrees of processing.

<i>Feed</i>	<i>Glycemic Index</i>	<i>Source</i>
Corn	117	(Rodiek, 2003)
Oats and molasses	105	
Barley	101	
<b>Oats</b>	<b>100</b>	
Oats and oil	86	
Alfalfa and molasses	85	
Wheat	71	
Vetch blend hay	53	
Carrots	51	
Wheat bran	37	
Timothy hay	32	
Alfalfa cubes	30	
Alfalfa hay	26	
Bermudagrass hay	23	
Rice bran	22	
Beet pulp	1	
Cracked corn	108	(Hoekstra et al., 1999)
Ground corn	118	
Steam flaked corn	156	

In human nutrition, the glycemic index provides a physiological classification of foods useful in developing nutritional programs for patients with insulin resistance or non-insulin dependent diabetes. These data may be useful in developing feeds and nutritional programs for horses.

### Summary

Horses evolved consuming primarily fermentable forage carbohydrates, but forage diets have been traditionally supplemented with grain meals rich in starch and sugar. Starch and sugar provide fuel for performance that is metabolically more efficient than fiber carbohydrates. This is important for performance horses; however, the consumption starch-rich meals may exacerbate equine digestive and metabolic disorders associated with carbohydrate metabolism. The critical capacity for preileal starch digestibility appears to be around 0.35 to 0.4% but may be as little as 0.2% of body weight per feeding, depending on the source of starch. Small intestinal absorption of simple sugars is limited by the activity and expression of two classes of glucose carrier proteins, which are affected by chronic intake of hydrolyzable carbohydrate but may be sluggish to respond to abrupt changes in diet, further exacerbating the risk of overload. Rate of hind gut fermentation and production of acids is dependent on the proportions of dietary forages and concentrates. The most rapid fermentation occurs during starch

overload or in the presence of fructans. Rapid fermentation perturbs the microbial and pH balance of the cecum and colon, favoring proliferation of *Lactobacillus spp* and acid production, and it increases the risk of colic and laminitis. In addition to digestive disturbances, feeding grain concentrates rich in hydrolyzable carbohydrate may increase the risk of insulin resistance, which has been associated with obesity, colic, laminitis, developmental orthopedic disease, and some forms of exertional rhabdomyolysis in horses. In order to provide optimal recommendations for carbohydrate nutrition of horses, the evaluation of horse feed carbohydrates may be improved by including a physiological classification that considers effects on glucose–insulin dynamics, such as the glycemic index.

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