Introduction

In the beginning, horses were grazers of forages, which were high in fiber and low in sugar and other soluble carbohydrates. They evolved with a large hindgut serving as a fermentative vat; this was accompanied by a weak small intestine. Then about 300 years ago intensive agriculture required the horse to do more work. The pastures often lacked the variety and sometimes were deficient in essential nutrients previously available. The extra energy and other nutrients needed were supplied as cereal grain supplements. These supplements contain large amounts of sugars and starches and provide twice the energy per gram than that of fiber. This new equine diet was alien to the horse's nutritional heritage, and occasionally caused digestive and metabolic upsets.

In more current management situations, pasture makes up all or part of the diet and habitat of horses. Ninety-four percent of horse farms in the United States allow their horses access to pasture (USDA, 1999). In most instances, pasture grasses and legumes alone can supply horses at maintenance with all of their required nutrients. However, for horses requiring extra energy and other nutrients (e.g., broodmares, young growing horses, performance horses), the pasture must be supplemented with energy and certain nutrients. For these horses, concentrates are commonly fed as a high carbohydrate meal two or three times per day to supplement the pasture and/or hay. A meal containing a large amount of non-structural carbohydrate has been associated with digestive, metabolic and hormonal changes (Clarke et al., 1990). Abrupt increases or high intakes of these non-structural carbohydrates may create a feeding-fasting cycle, which may cause large fluctuations in hormones and other metabolic substrates important in growth and bone formation (Kronfeld, 1998).

To avoid the feeding-fasting cycle and its consequences, our laboratory has been developing horse feeds in which sugar and starch are replaced with other energy sources – fiber and fat (Kronfeld, 1996; Hoffman and Kronfeld, 1999). Replacement of starch with fat in the feed has been used to prevent rhabdomyolysis (Kronfeld, 1990; Valentine et al.,

1998), to enhance aerobic (Meyers et al., 1989) and anaerobic exercise (Oldham et al., 1990), and to reduce excitability (Holland et al., 1996). The added fiber in the feed is intended to buffer the seasonal changes of the pasture grasses. Our laboratory has been developing horse feeds in which sugar and starch are replaced with multiple fiber sources and fat (Kronfeld, 1996; Hoffman and Kronfeld, 1999). The present study compares the glycemic and insulinemic responses of a typical concentrate feeds and a feed with fat as the energy source and sufficient fiber to take in account the seasonal variation of pastures.

Literature Review

Pasture Supplementation

When formulating a pasture supplement one first needs to decide which nutrients are present at low or deficient concentrations in the pasture. Seasonal variation of the pasture also needs to be taken into account when determining the nutritional contribution of the pasture. In general, as pasture grows early in its growing season the energy, nonstructural carbohydrates (NSC), and protein contents increase, then late in its growing season, the fiber and lignin contents start to increase (Ball et al., 1996). As plants shed their seeds, usually in late fall, they also lose their energy, protein, phosphorus and NSC content. These typical seasonal changes may be altered by unseasonable weather. The NSC content of the Virginia Tech MARE Center pastures' can range from about 8 to 11 % on a DM basis in the fall months and from 14 to 19 % in the spring months, depending on the weather and rain fall (Dairy One, DHI Forage Testing Laboratory, Ithaca, NY).

A survey of 12 farms has been conducted in northern Virginia (Kronfeld et al., 1996). Pastures were marginal or deficient in sodium, phosphorus, zinc, copper, and selenium when compared to the recommendations of the NRC (1989). Selenium concentration in mares and foals was found to be low in the blood serum, which reflected low concentrations in the pasture. However, there were no clinical signs of deficiency. Yearlings that were unsupplemented had hypophosphatemia, which indicated low phosphorus in the diet. In contrast, mares unsupplemented with phosphorus had inconsistent hyperphosphatemia, hyperphosphaturia and hypercalciuria (Greiwe-Crandell et al., 1992). The urinary findings suggested that mobilization of calcium and phosphorus from the bone ensured an adequate supply for the growing fetus. Foals maintained on pasture with a concentrate supplement were found to be less stressed during weaning than foals raised on pasture only (Hoffman et al., 1995). The zinc content in the concentrate supplemented total diet was seven times that of the pasture alone, and serum zinc concentration was lower in foals that only consumed pasture. Copper in the forages

marginally met minimal requirements (NRC, 1989). Supplementation of pregnant mares with two to three times the recommended copper intake has reduced the frequency of skeletal abnormalities in foals (Hurtig et al., 1990; Pearce et al., 1998). Carotenes are abundant in green forages and most horses that have daily access to pasture are able to meet their vitamin A requirements. However, there are seasonal differences in pasture β-carotene contents. During the winter months horses on pasture have been shown to have a depletion of vitamin A indicated by a relative dose response test for retinol (Greiwe-Crandell et al., 1995). When these horses were repleted in their vitamin A status by supplementation of retinal palmitate at twice the current recommended amount of vitamin A their status did improve, but not enough to eliminate the seasonal fluctuations (Greiwe-Crandell et al., 1997). When supplemented with a water-soluble form of β-carotene there was no improvement of vitamin A status. A previous study demonstrated that multiple variables in growing ponies reached optimal values when 1.5- to 5-times the recommended vitamin A intake was fed (Donoghue et al., 1981). Because of multiple interactions, tentative target ranges for all micronutrients should be kept in line with zinc, copper and vitamin A, that is, two to three times the minimum requirements (Hoffman and Kronfeld, 1999).

The NSC consists of hydrolyzable (e.g., starch) and non-hydrolyzable but rapidly fermentable carbohydrates (e.g., fructans, pectins). When NSC is consumed in large amounts (i.e. spring pastures or large grain meals), a portion escapes hydrolysis in the small intestine and moves to the large colon where it undergoes rapid fermentation. Rapid fermentation produces excessive gas and lactic acid, including D- as well as the usual L-lactate. D-lactic acid is not metabolized and tends to accumulate. The high concentration of lactic acid attracts water and lowers the luminal pH below 6, thereby increasing the risk of digestive disorders, such as osmotic diarrhea and colic associated with distension by gas and fluid (Clarke et al., 1990; Cohen et al., 1999).

Feeding meals high in hydrolyzable carbohydrates can create a feeding-fasting cycle (Figure 1), which is alien to the evolutionary grazing heritage of the horse (Kronfeld, 1998). The interactions of glucose and free fatty acids following a meal were first emphasized by Randle in 1955 (cited in Randle, 1998). He compared the “Glucose-Fatty

Acid Cycle" following high carbohydrate and high fat meals. This feeding-fasting cycle consists of large fluctuations in hormones and certain metabolic products. During the feeding portions of the cycle, glucose and insulin increase while free fatty acids, growth hormone, insulin-like-growth-factor-1 (IGF-1) and glucagon decrease. During the fasting portion of the cycle the opposite occurs. These metabolic and endocrine fluctuations are risk factors in various metabolic diseases including laminitis, gastric ulcers, perhaps some forms of exertional rhabdomyolysis, and osteochondrosis (Clarke et al., 1990; Kronfeld, 1998; Murray, 1999).

A series of studies at the Virginia Tech MARE Center are involved with developing a supplement to complement the variable nutrient content of the pastures, improve the health and well being of the mares and foals, and manage the nutrition of the animals in ways that will enhance the environment. The pasture supplement currently under development includes multiple fiber sources intended to promote a diverse microbial population that can adapt readily to changes in pasture contents of carbohydrates (Kronfeld, 1996). The wide spectrum of fibers amounting to about 36 % NDF qualifies this feed as roughage. The supplement also includes a cereal byproduct that contains about 26 % fat and 18 % protein. The added fat restores energy normally provided by the carbohydrates; this qualifies the feed as a concentrate.

Added Fiber.

Multiple fiber sources are the key components in our laboratory's pasture supplement. During the springtime when the grass is rapidly growing the fiber content of the pasture is low and slowly fermentable fiber is needed to keep the digestive system healthy and functioning. The full spectrum of carbohydrates is used to promote a diverse microbial population, which can readily adapt to any abrupt seasonal change of pasture carbohydrate content (Hoffman and Kronfeld, 1999). There are three main classifications of carbohydrates (Van Soest et al., 1991). Metabolism of hydrolyzable carbohydrates yields mostly glucose and other simple sugars (typically found in a traditional sweet feed). Rapidly fermented carbohydrates and non-hydrolyzable carbohydrates consumed in excess yield mostly propionate and lactate, which are metabolized via glucose and pyruvate

(Figure 2). Slowly fermentable carbohydrates yield mainly acetate and butyrate, which are metabolized via acetyl-CoA (used in the fat and fiber pasture supplement). The higher and more variable fiber sources in the pasture supplement are intended to buffer the seasonal variation of nutrients in the pastures by promoting slow fermentation and yielding mostly acetate and butyrate in the gut (Hoffman and Kronfeld, 1999). Propionate can control plasma glucose concentration by acting as a precursor or acetate and butyrate (betahydroxybutyrate) can spare glucose oxidation. Such effects are likely to be smaller than that of direct glucose absorption (through starch digestion), which may explain the positive effects of feeding hay and other roughages (Argenzio and Hintz, 1971; Tisserand, 1992). Rapidly fermentable or hydrolyzable carbohydrates would increase lactate production, thus possibly increasing the risk of metabolic and digestive disorders created by the hydrolyzable carbohydrates and the feeding-fasting cycle.

Added Fat.

Incorporating fat in the pasture supplement is secondary to the addition of fiber; however, the removal of hydrolyzable carbohydrates requires the addition of an alternate source of energy. Using a higher level of fat (i.e. 10 % vs. 3 %) as the energy source in a horse's diet has recently become popular and has several benefits. Along with an improved haircoat, increasing the dietary intake of fat allows the horse to obtain more energy or calories without the hyperactivity associated with high carbohydrate diets (Holland et al., 1996). Fat is more energy dense when compared to carbohydrates (9 kcal/g of fat vs 4 kcal/g of hydrolyzable carbohydrate and about 2 kcal/g of fermented carbohydrate). Including about 10 % fat in the diet reduces the heat of fermentation by about 3 % and metabolic heat by 2 to 5 % (Kronfeld, 1996). In the performance horse, this increase in energetic efficiency should enhance thermoregulation, delay fatigue and reduce cooling and recovery times.

Replacement of starch with fat in the feed has been used to prevent rhabdomyolysis (Kronfeld, 1990; Valentine et al., 1998; De La Corte et al., 1999), enhance aerobic (Meyers et al., 1989) and anaerobic exercise (Oldham et al., 1990), smooth out the growth curve (Hoffman, 1997) and reduce excitability (Holland et al., 1996). The lowering of glucose-

equivalents in the feed and increase in the fat and fiber contents is likely to cause changes in the glycemic responses to a meal. Higher fat in the diet may delay or decrease the peak glycemic response by retarding gastric emptying in human subjects (Wolever, 1990). To our knowledge, the effect of a high fat meal on gastric emptying has not been studied in horses.

Many studies have experimented with various levels of high fat feeds compared to a control feed or traditional sweet feed affecting growth, production or performance. One such study measured the growth of Quarter Horse yearlings given treatments of 5 or 10 % added fat (Scott et al., 1989). Average daily gains were greater and total feed intake was lower for yearlings on the 10 % added fat feed compared to the control or no fat feed. No differences between treatment groups were observed for bone density, plasma glucose, insulin and lipid concentrations. A similar study compared growth of yearlings and digestibility of diets with or without 10 % added fat (Davidson et al., 1991). Concentrate intake was lower for the weanlings offered the added fat diet, most likely due to the increase in digestibility of NDF and ether extract. Average daily gain tended to be higher in the fat fed group and they had a lower feed:gain ratio then the control group. Both studies concluded that fat could be used in high-energy diets for rapidly growing horses to replace some of the carbohydrates that have been reported to cause skeletal abnormalities.

Added animal fat (10 % of concentrate) to a performance horse's diet has been shown to increase ($P < 0.05$) resting muscle glycogen when compared to a control diet of primarily corn and oats (Oldham et al., 1990). Glycogen utilization was also greater ($P < 0.05$) in the fat supplemented group than in the control group after completing four 600 m sprints. Post-exercise muscle glycogen concentrations were similar between the groups of horses. These data indicate that fat supplemented horses had greater stores of muscle glycogen available for use in glycogenolysis and glycolysis during the anaerobic exercise.

Another study showed that added dietary fat had a stimulatory effect on muscle glycogen synthesis or a sparing effect on muscle glycogen utilization when horses were subjected to slow submaximal exercise (Meyers et al., 1989). They found that the fat supplemented horses had a higher initial muscle glycogen concentration and a decrease in

glycogen utilization. Also a trend for lower blood lactate levels was found when horses were fed 10 % added fat.

In addition, both of these studies observed that by adding fat to the diets of exercising horses the caloric density could be increased with a beneficial reduction in overall feed intake. A similar study demonstrated similar results when using fat as the energy source for exercising horses (Greiwe et al., 1989). Overall, fat alone has many benefits that can improve a horse's growth, production, performance and nutritional status.

Glycemic Response

Due to the sensitivity of the horse to changes in the diet, nutrient and hormonal response tests have been developed. Glycemic response is one such test, which describes the changes in plasma glucose concentration after a meal. The *glycemic index* (GI) is formed when the glycemic response of a reference food (i.e. glucose) is compared to the glycemic response of a test food (Jenkins et al., 1981). In contrast, the *glucose tolerance test*, which is similarly determined, provides information of the carbohydrate and insulin status of an individual, and is measured after giving an oral or intravenous dose of glucose (Roberts and Hill, 1973). The first oral glucose tolerance test (OGTT) in the horse was conducted by Roberts and Hill (1973). Horses were administered 1.0 g anhydrous glucose per kg body weight in a 20 % solution and plasma glucose was measured for 6 h after dosing. The shape of the tolerance curve was affected by the dose of glucose administered. The quantity and concentration of glucose dose was also important because gastric emptying was delayed with increasing concentrations of glucose. The OGTT results are also dependant on the rapid entry of the glucose into the lumen of the small intestine. It was concluded that the OGTT in the horse provides an aid to the diagnosis of pancreatic and small intestine dysfunction, even though there are many factors that influence the exact problem or disease.

More recent studies on the OGTT include testing the effect of altering the animal's diet days prior to the test (Jacobs and Bolton, 1982), studying insulin sensitivity and carbohydrate metabolism in fat and laminitic ponies (Jeffcott et al., 1986) and in horses

with exertional rhabdomyolysis (De La Corte et al., 1999), studying the difference in glucose tolerance among various equids (June et al., 1992) and testing the effects of age and diet on glucose tolerance (Murphy et al.,1997).

When conducting a GI study, the feed or food is classified as low-GI or high-GI. The use of low-GI diets to reduce the risk of non-insulin-dependant diabetes, coronary heart disease and obesity is widely accepted for affected individuals, but controversial in the public health field (Beebe, 1999; Brand-Miller and Foster-Powell, 1999).

Low-GI meals are beneficial to diabetic humans because they are digested and absorbed more slowly, and thus flatten the glucose curve and lower insulin levels. On the other hand, consuming a high-GI meal may exaggerate the insulin and glucose response, which could lead to metabolic problems in diabetics (Brand-Miller and Foster-Powell, 1999). In regard to exercise, high-GI meals consumed after exercise may increase glycogen storage. In contrast, low-GI meals consumed before endurance exercise may prolong glucose availability and enhance performance (Burke et al., 1998).

The GI of a food cannot be predicted by the type of carbohydrates present (i.e. simple or complex, soluble or insoluble, etc), with the exception of rapidly available glucose, $r^2 = 0.58$ (Englyst et al., 1996). Some factors that influence the GI include different plant components (i.e. lectins, phytate, tannins, enzyme inhibitors, etc.), fiber content, different macronutrients (i.e. fat and protein), and nature of the starch in the food (Wolever, 1990).

Human GI Studies.

One of the first studies to apply and assign foods a GI value offered 62 different foods in portions of 50 g of carbohydrates as glucose equivalents (Jenkins et al., 1981). Glucose was used as the reference carbohydrate (assigned a rank of 100) for each subject. The magnitude of the response was measured as the area under the curve of plasma glucose plotted against time and expressed as a percentage of the reference; this is the GI (Table 1). The highest GI foods included carrots, and parsnips (92 and 97, respectively) and the lowest GI foods included peanuts and soybeans (13 and 15, respectively). A

negative relationship was found between fat (r = -0.386, $P < 0.01$) and protein (r = -0.523, $P < 0.01$) content and the GI of the food.

Applications of the GI have focused on human athletic diets and the potential to improve performance. A recent study compared the glycemic, insulinemic and physiologic responses to a pre-exercise control meal (CON), low-GI meal, and a high-GI meal (DeMarco et al., 1999). Thirty min after consuming the meal, subjects performed a cycling test at 70 % VO_{2max} for 2 h (comparable to competitive cycling). Throughout exercise the low-GI group had a more gradual plasma glucose decline than the high-GI group, and by the end of the trial the low-GI group's plasma glucose levels were higher ($P < 0.05$) than the high-GI and CON. In the same study, after the endurance trial, subjects cycled at 100 % VO_{2max} to exhaustion and showed that time to exhaustion was longer for the low-GI group than the high-GI group (206.5 ± 43.5 and 129.5 ± 22.8 s, respectively, $P < 0.05$).

In a similar study, a low-GI meal consumed before endurance exercise resulted in higher levels of blood glucose and free fatty acids than did high-GI meals for 120 min of exercise (Thomas et al., 1994). Although no differences were found in time to exhaustion, the authors concluded that low-GI meals consumed before exercise favors endurance. Other explanations are feasible, given the variation in interpreting changes in plasma metabolite concentration. Differences in substrate utilization during exercise with low-GI and high-GI meals have also been observed, with no difference in performance (Sparks et al., 1998).

Equine GI Studies.

Intense athletic activity has raised concern for increased supplementation of energy. Traditionally the increased demands are met by feeding a grain concentrate rich in starch, two times a day. This meal feeding regime creates a feeding-fasting cycle (Figure 1). In horses, daily changes in insulin and counter-regulatory hormones may contribute to the development of certain metabolic disorders (Glade and Reimers, 1985; Clarke et al., 1990; Ralston, 1996). Feeding low-GI meals (fat and fiber) rather than high-GI meals (starch and sugar) may moderate or eliminate the fluctuations in the cycle and should identify the problematic foods (Ralston, 1992; Kronfeld, 1998).

When using the GI in the horse, meals are usually defined in terms of total weight; however, it may be more beneficial to measure in terms of energy equivalents. This affects the outcome of the GI, and makes it more useful in formulating supplements to minimize the feeding-fasting cycle changes and to plan appropriate feeding management (Kronfeld, 1998). Studies applying the GI in horses include determining feed intake, type and time (Arana et al., 1989; Pagan et al., 1999), maximizing equine performance (Arana et al., 1988; Stull and Rodiek, 1995), and management of osteochondritis dissecans (OCD) and growth in young horses (Ralston, 1996).

The glycemic response to different feed types was measured using six different feeds in addition to mixed grass hay (Pagan et al., 1999). Whole oats fed at the medium intake served as the reference meal. Sweet feed and oats had the greatest GI, followed by cracked corn, a fiber mix, alfalfa and sweet feed plus corn oil. As the feeding level increased, the glycemic response increased for all of the feeds except whole oats and cracked corn. A similar study failed to find a difference in the glycemic and insulinemic response of four different grains (barley, corn, oats, and sweet feed) and four forms of alfalfa (chopped, cubed, long, and pelleted) (Arana et al., 1989).

The application of the GI in relation to the endurance horse would be beneficial to assess foods that maintain blood glucose concentration and promote the use of fatty acids. In a study to test the effect of different glycemic meals during exercise in horses, three diets were used: cracked corn, alfalfa, or fasting (Stull and Rodiek, 1995). One or 4 h after consuming the meal, a standard exercise test was performed. During exercise, plasma glucose concentrations fell in horses fed corn in both protocols, but increased after exercise completion. In the 4-h protocol, a higher plasma glucose concentration was observed in the fasted horses than in the corn fed horses. Thus, feeding corn 4 h before exercise was detrimental because the blood glucose concentrations fell drastically when exercise began and remained lower than when in a fasted state. The decrease in blood glucose after consuming a corn meal was attributed to a greater utilization of glucose by the muscles compared to a meal of alfalfa, which was interpreted as an increased utilization of free fatty acids. The decreased use of free fatty acids for energy in the corn fed horses may be due to the elevated insulin inhibiting lipolysis.

A similar study used horses that were fasted or fed alfalfa, corn or corn plus corn oil, then performed a standard exercise test 2 h after the meal (Arana et al., 1988). Before the cantering phase (after the warm up section), glucose levels were highest in horses fed corn or corn plus corn oil. These groups, however, utilized more glucose during the exercise than the fasting horses; thus the high glucose response from the meal was of no benefit in the end. During the recovery period after exercise the corn and corn plus corn oil diets had a larger increase in glucose and higher peak in insulin ($P < 0.05$). These results suggest that the fasted and alfalfa fed horses maintained their glucose levels during the exercise, perhaps by an increased utilization of free fatty acids or mobilization of liver glycogen stores to maintain a plasma glucose balance.

Insulin Resistance.

The amount, type and rate of digestion of ingested hydrolyzable carbohydrates are the determining factors of glucose entry into the blood, hence the plasma glucose and insulin response after a meal. Amounts of other macronutrients (i.e. fat and protein) also have an effect on these responses (Wolever, 2000). When considering starchy meals, decreasing the amount of carbohydrate in the diet or can reduce the response by reducing the rate of absorption. Neutral fat and free fatty acids may also have their effect on insulin sensitivity. Carbohydrates in the diet could mediate insulin secretion through altering plasma free fatty acid concentration because high concentrations of free fatty acids are associated with insulin resistance. Figure 3 illustrates the theory that reducing postprandial glucose and insulin responses may interrupt the progression of insulin resistance and related diseases. Free fatty acids raise plasma glucose by reducing insulin-stimulated glucose uptake through GLUT-4 receptors and by reducing glucose-induced insulin secretion (Wolever, 2000). A high rise in insulin causes the blood glucose to undershoot, which is then followed by a rebound in plasma free fatty acids.

Dietary fat has its effect on insulin secretion through the secretion of gastric inhibitory polypeptide (GIP). Insulin release is stimulated by GIP along with and amplification of insulin on target tissues (Yip and Wolfe, 2000). In adipose tissue, GIP stimulates fatty acid synthesis, enhances insulin-stimulated synthesis of triglycerides from

fatty acids, increases insulin receptor affinity, and increases the sensitivity of insulin-stimulated glucose transport. The secretion of GIP is very sensitive to changes in diet. In humans, roughly 40 % of the energy in the diet is provided by fat, which is a stimulator of GIP release. However, in rats or pigs (and possibly the horse) that consume a diet with < 10 % fat, carbohydrates account for more of a GIP stimulating effect. The increased GIP response would increase insulin release resulting in greater and prolonged hyperinsulinemia and also increase the affinity of the insulin receptor for insulin as well as increase the insulin-like effects in fat cells (Yip and Wolfe, 2000).

Insulin resistance is characterized by specific changes of the proportions of the fatty acid pattern of the serum lipids, indicating possible changes and elongation in the body. Diets higher in unsaturated fatty acids have been associated with better insulin sensitivity than in diets with a high saturated fat content (Vessby, 2000). Insulin-stimulated glucose uptake was measured in incubated muscle tissue and then measured for triglyceride concentration after stripping away all interstitial fat and connective tissue. A highly significant inverse relationship was found between intramyocyte triglyceride and insulin-stimulated glucose metabolism. Therefore, impaired insulin action has been closely associated with high muscle triglyceride levels (Storlien et al., 2000).

Foods higher in glucose or hydrolyzable carbohydrates are usually lower in fiber and lead to an increased glycemic and insulinemic response. This hyperinsulinemia may result in many glucose and insulin complications, including insulin resistance. In order to break this cycle, highly viscous fiber foods can cause the small intestine to slowly release glucose molecules into portal circulation (Jenkins et al., 2000). When viscous fiber guar was given to healthy men, insulin sensitivity, triglyceride levels, clotting factors, and blood pressure improved. Overall, whole-grain foods and low-GI foods are becoming dietary components, which can reduce the complications of certain insulin-resistant related diseases in humans. This is based on the evidence that the rate of absorption is slowed by creating a reservoir in the small intestine.

Many of these concepts have been well developed in the human and may be adapted to the horse; however, studies are lacking to provide sufficient scientific evidence.

Studies have been conducted investigating glucose and insulin metabolism in horses diagnosed with osteochondrosis (Glade and Reimers, 1985; Ralston, 1996) and rhabdomyolysis (Valentine et al., 1998; De La Corte et al., 1999)

Young horses offered large amounts of high energy carbohydrates are believed to be more susceptible to osteochondrosis (OCD), a disease resulting from failure of the endochondral ossification, which causes a thickening of the cartilage and cracks or lesions extending into the joint (Jeffcott, 1991). In a study by Ralston (1996), it was proposed that hyperglycemia/hyperinsulinemia caused by a high carbohydrate diet would be greater in horses with OCD. Young horses were fed a diet of 50 % sweet feed (Omolene 300, Purina Mills, St. Louis, MO, which contains abundant starch and sugar) and 50 % alfalfa/grass mixed hay. Results showed that horses diagnosed with OCD had greater ($P < 0.02$) changes in plasma glucose and insulin when fed sweet feed. The OCD horses also had a faster rate of appearance of glucose ($P < 0.02$). This glucose intolerance and apparent insulin resistance may correlate with the development of OCD in young horses. Insulin affects cartilage growth by influencing growth hormone and IGF-1 release (Glade, 1986).

Glucose uptake was studied in horses with polysaccharide storage myopathy (PSSM, a form of recurrent exertional rhabdomyolysis). Oral and intravenous glucose tolerance tests, and insulin tolerance tests were given to horses with a history of PSSM; healthy adult horses served as a control (De La Corte et al., 1999). The intravenous glucose tolerance test had an increased rate of glucose clearance of 1.5-times that in control horses. Blood glucose concentrations in horses given the oral glucose tolerance test were lower throughout the test for horses with PSSM. The insulin tolerance test resulted in decreased glucose concentrations in both groups of horses. However, horses with PSSM continued to have low blood glucose concentrations for more than 3 hours after the insulin injection, where normal horses returned to baseline within 2 hours of the injection. This study concluded that horses with PSSM had enhanced uptake of glucose, which may be due to and increase in insulin sensitivity.

Literature Cited

USDA, Animal and Plant Health Inspection Service. 1999. Info Sheet: Veterinary Services, Highlights of equine '98 study results.

Arana, M.J., A.V. Rodiek, and C.L. Stull. 1988. Effects during rest and exercise of four dietary treatments on plasma glucose, insulin, cortisol, and lactic acid. Proc. Am. Soc. Anim. Sci. 39:165-169.

Arana, M.J., A.V. Rodiek, and C.L. Stull. 1989. Blood glucose and insulin responses to four different grains and four different forms of alfalfa hay fed to horses. Proc. Equine Nutr. Physiol. Soc. 11:160-161.

Argenzio, R.A., and H.F. Hintz. 1971. Volatile fatty acid tolerance and the effect of glucose and VFA on plasma insulin levels in ponies. J. Nutr. 101:723-730.

Ball, D.M., C.S. Hoveland, and G.D. Lacefield. 1996. Forage Quality. In: Southern Forages. 2nd Edition. Potash & Phosphate Institute. Norcross, GA. p. 124-132.

Beebe, C.B. 1999. Diets with a low glycemic index: not ready for practice yet! Nutr. Today. 34:82-86.

Brand-Miller, J., and K. Foster-Powell. 1999. Diets with a low glycemic index: from theory to practice. Nutr. Today. 34:64-72.

Burke, L.M., G.R. Collier, and M. Hargreaves. 1998. Glycemic index - a new tool in sport nutrition? Int. J. Sport Nutr. 8:401-415.

Clarke, L.L., M.C. Roberts, and R.A. Argenzio. 1990. Feeding and digestive problems in horses. Vet. Clin. N. Am: Equine Pract. 6:433-450.

Cohen, N.D., P.G. Gibbs, and A.M. Woods. 1999. Dietary and other management factors associated with colic in horses. J. Am. Vet. Med. Assoc. 215:53-60.

Davidson, K.E., G.D. Potter, J.W. Evans, L.W. Greene, P.S. Hargis, C.D, Corn, and S.P. Webb. 1991. Growth, nutrient utilization, radiographic bone characteristics and postprandial thyroid hormone concentrations in weanling horses fed added dietary fat. Equine Vet. Sci. 11:119-125.

De La Corte, F.D., S.J. Valberg, J.M. MacLeay, S. E. Williamson, and J.R. Mickelson. 1999. Glucose uptake in horses with polysaccharide storage myopathy. Am. J. Vet. Res. 60:458-462.

DeMarco, H.M., K.P. Sucher, C.J. Cisar, and G.E. Butterfield. 1999. Pre-exercise carbohydrate meals: application of glycemic index. Med. Sci. Sports Exerc. 31:164-170.

Donoghue, S., D.S. Kronfeld, S.J. Berkowitz, and R.L. Copp. 1981. Vitamin A nutrition of the equine: growth, serum biochemistry and hematology. J. Nutr. 111:365-374.

Englyst, H.N., J. Veenstra, and G.J. Hudson. 1996. Measurement of rapidly available glucose (RAG) in plant foods: a potential in vivo predictor of the glycaemic response. Br. J. Nutr. 75:327-337.

Glade, M.J., and T.J. Reimers. 1985. Effects of dietary energy supply on serum thyroxine, triiodothyroine and insulin concentrations in young horses. J. Endocrinol. 104:93-105.

Glade, M.J. 1986. The control of cartilage growth in osteochondrosis: a review. J. Equine Vet. Sci. 6:175-187.

Greiwe, K.M., T.N. Meachem, and J.P. Fontenot. 1989. Effect of added dietary fat on exercising horses. Proc. Equine Nut. Phys. Soc. 11:101-106.

Greiwe-Crandell, K.M., G.A. Morrow and D.S. Kronfeld. 1992. Phosphorus and selenium depletion in Thoroughbred mares and weanlings. Pferdeheilkunde. 1:96-98.

Greiwe-Crandell, K.M., D.S. Kronfeld, L.S. Gay, and D. Sklan. 1995. Seasonal vitamin A depletion in grazing horses is assessed better by the relative dose response test than serum retinol concentration. J. Nutr. 125:2711-2716.

Greiwe-Crandell, K.M., D.S. Kronfeld, L.S. Gay, D. Sklan, W. Tiegs, and P.A. Harris. 1997. Vitamin A repletion in Thoroughbred mares with retinyl palmitate or β-carotene. J. Anim. Sci. 75:2684-2690.

Hoffman, R.M. and D.S. Kronfeld. 1999. Nutrient requirements for grazing horses: development of an optimal pasture supplement. In: Garnsworthy, P.C. and J. Wiseman (Eds). Recent Advances in Animal Nutrition. Nottingham University Press, Nottingham, U.K.

Hoffman, R.M. 1997. Carbohydrate and fat supplementation in grazing mares and foals. Ph.D. dissertation. Virginia Polytechnic Institute and State University, Blacksburg.

Hoffman, R.M., D.S. Kronfeld, L.A. Lawrence, W.L. Cooper, J.J. Dascanio and P.A. Harris. 1996. Dietary starch and sugar versus fat and fiber: growth and development of foals. Pferdeheilkunde. 12(3):312–316.

Hoffman, R.M., D.S. Kronfeld, J.H. Herbein, W.S. Swecker, W.L. Cooper, and P.A. Harris. 1998. Dietary carbohydrates and fat influence milk composition and fatty acid profile of mare's milk. J. Nutr. (in press).

Hoffman, R.M., D.S. Kronfeld, J.L. Holland and K.M. Greiwe-Crandell. 1995. Preweaning diet and stall weaning method influences on stress response in foals. J. An. Sci. 73:2922-2930.

Holland, J.L., D.S. Kronfeld, and T.N. Meacham. 1996. Behavior of horses is affected by soy lecithin and corn oil in the diet. J. Anim. Sci. 74:1252-1255.

Hurtig, M.B., S.L. Green, H. Dobson, and J. Burton. 1990. Defective bone and cartilage in foals fed a low-copper diet. Proc. Am. Assoc. Equine Pract. 36:637-643.

Jacobs, K.A., and J.R. Bolton. 1982. Effect of diet on the oral glucose tolerance test in the horse. J. Am. Vet. Med. Assoc. 180:884-886.

Jeffcott, L.B. 1991. Osteochondrosis in the horse - searching for the key to pathogenesis. Equine Vet. J. 23:331-338.

Jeffcott, L.B., J.R. Field, J.G. McLean, and K. O'Dea. 1986. Glucose tolerance and insulin sensitivity in ponies and Standardbreds. Equine Vet. J. 18:97-101.

Jenkins, D.J.A., M. Axelsen, C.W.C. Kendal, L.S.A. Augustin, V. Vuksan and U. Smith. 2000. Dietary fiber, lente carbohydrates and the insulin-resistant diseases. British J. Nutr. Suppl. 83:S157-S163.

Jenkins, D.J.A., T.M.S. Wolever, R.H. Taylor, H. Barker, H. Fielden, J.M. Baldwin, A.C. Bowling, H.C. Newman, A.L. Jenkins, and D.V. Goff. 1981. Glycemic index of foods: a physiological basis for carbohydrate exchange. Am. J. Clin. Nutr. 34:362-366.

June, V., V. Soderholm, H.F. Hintz, and W.R. Butler. 1992. Glucose tolerance in the horse, pony and donkey. Equine Vet. Sci. 12:103-105.

Kronfeld, D.S. 1998. Clinical Assessment of Nutritional Status of the Horse. In: T. Watson (Ed.) Metabolic and Endocrine Problems of the Horse. pp 185-217. W.B. Saunders Company, London.

Kronfeld D.S., W.L. Cooper, K.M. Crandell, L.A. Gay, R.M. Hoffman, J.L. Holland, J.A. Wilson, D Sklan, and P.A. Harris. 1996. Supplementation of pasture for growth. Pferdeheilkunde. 12(3):317–319.

Kronfeld, D.S. 1996. Dietary fat affects heat production and other variables of equine performance, under hot and humid conditions. Equine Vet. J. Suppl. 22:24-34.

Kronfeld, D.S. 1990. Dietary aspects of developmental orthopedic disease in young horses. Clin. Nutr. 6:451-465.

Mayes, P.A. 1996. Gluconeogenesis and Control of the Blood Glucose. In: J. Dolan and C. Langan (Ed.) Harper's Biochemistry. pp 194-204. Appleton and Lange, Stamford, Conn.

Meyers, M.C., G.D. Potter, J.W. Evans, L.W. Green, and S.F. Crouse. 1989. Physiologic and metabolic response of exercising horses to added dietary fat. Equine Vet. Sci. 9:218-223.

Murphy, D., S.W. Reid, S. Love. 1997. The effect of age and diet on the oral glucose tolerance test in ponies. Equine Vet. J. 29:467-470.

Murray, M.J. 1999. Gastroduodenal ulceration in foals. Equine Vet. Edu. 11:199-207.

NRC. 1989. Nutrient Requirements of Horses (5th Ed.). National Academy Press, Washington, D.C.

Oldham, S., G.D. Potter, J.W. Evans, S.B. Smith, T.S. Taylor, and W.S. Barnes. 1990. Storage and mobilization of muscle glycogen in exercising horses fed a fat supplemented diet. J. Equine Vet. Sci. 10:353-359.

Pagan, J.D., P.A. Harris, M.A.P. Kennedy, N. Davidson, and K.E. Hoekstra. 1999. Feed type and intake affect glycemic response in Thoroughbred horses. Proc. Equine Nutr. Physiol. Soc. 16:149-150.

Pearce, S.G., N.D. Grace, E.C. Firth, and P.F. Fennessy. 1998b. Effect of copper supplementation on the evidence of developmental orthopaedic disease in pasture-fed New Zealand Thoroughbreds. Equine Vet. J. 30:211-218.

Ralston, S.L. 1996. Hyperglycemia/hyperinsulinemia after feeding a meal of grain to young horses with osteochondritis dissecans (OCD) lesions. Pferdeheilkunde. 12:320-322.

Ralston, S.L. 1992. Effect of soluble carbohydrate content of pelleted diets on postprandial glucose and insulin profiles in horses. Pferdeheilkunde. 8:112-115.

Randle, P.J. 1998. Regulatory interactions between lipids and carbohydrates: the glucose fatty acid cycle after 35 years. Diabetes Metab. Reb. 14:263-283.

Roberts, M.C. and F.W.G. Hill. 1973. The oral glucose tolerance test in the horse. Equine Vet. J. 5:171-173

Scott, B.D., Potter, G.D., Evans, J.W., Reagor, J.C., Webb, G.W., and Webb, S.P. 1989. Growth and feed utilization by yearling horses fed added dietary fat. Equine Vet. Sci. 9:210-214.

Sparks, M.J., S.S. Selig, and M.A. Febbraio. 1998. Pre-exercise carbohydrate ingestion: effect of the glycemic index on endurance exercise performance. Med. Sci. Sports Exerc. 30:844-849.

Storlien, L.H., J.A. Higgins, T.C. Thomas, M.A. Brown, H.Q. Wang, X.F. Huang and P.L. Else. 2000. Diet composition and insulin action in animal models. British J. Nutr. Suppl. 83:S85-S90.

Stull, C.L., and A.V. Rodiek. 1995. Stress and glycemic responses to postprandial interval and feed components in exercising horses. J. Equine Vet. Sci. 15:382-386.

Thomas, D.E., J.R. Brotherhood, and J. Brand-Miller. 1994. Plasma glucose levels after prolonged strenuous exercise correlate inversely with glycemic response to food consumed before exercise. Int. J. Sport Nutr. 4:361-373.

Tisserand, J.L. 1992. Fermentation in the hindgut of the horse – possibilities of disorders. Pferdeheilkunde. 8:197-200.

Valentine, B.A., H.F. Hintz, K.M. Freels, A.J. Reynolds, and K.N. Thompson. 1998. Dietary control of exertional rhabdomyolysis in horses. J. Am. Vet. Med. Assoc. 212:1588-1593.

Van Soest, P.J., J.B. Robertson and B.A. Lewis. 1991. Methods for dietary fiber, neutral detergent fiber, and nonstarch polysaccharides in relation to animal nutrition. J. Dairy Sci. 74:3582-3597.

Vessby, B. 2000. Dietary fat and insulin action in humans. British J. Nutr. Suppl. 83:S91-S96.

Wolever, T.M.S. 2000. Dietary carbohydrates and insulin action in humans. British J. Nutr. Suppl. 83:S97-S102.

Wolever, T.M.S. 1990. The glycemic index. World Rev. Nutr. Diet. 62:120-185.

Yip, R.G.C. and M.M. Wolfe. 2000. GIP biology and fat metabolism. Life Sci. 66:91-103.

Table 1. The glycemic index (GI) of food based on glucose as the reference (i.e. 100)[a]

High GI foods	GI #
Parsnips	97
French Bread	95
Carrots	92
Honey	87
Cornflakes	84
Jelly beans	80
Potatoes	70-80
White rice	72
White bread	70
Banana	62

Low GI	GI #
Mixed grain bread	30-45
All-Bran cereal	42
Baked Beans	40
Apple	39
Tomato soup	38
Skim Milk	32
M&M's	33
Peach	28
Soybeans	15
Peanuts	13

[a]From Brand-Miller and Foster-Powell (1999); Jenkins et al. (1981)

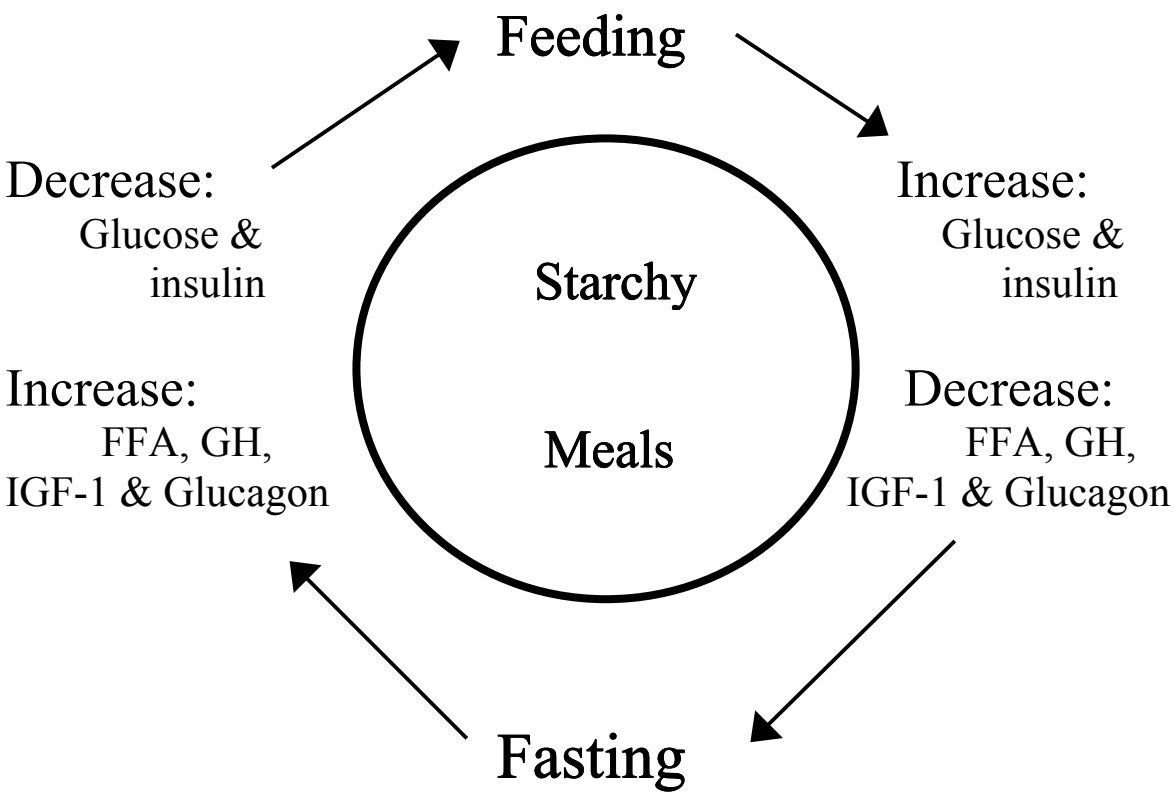

Figure 1. Feeding-fasting cycle (adapted from Kronfeld, 1996). During the feeding portions of the cycle, glucose and insulin increase while free fatty acids (FFA), growth hormone (GH), insulin-like-growth-factor-1 (IGF-1) and glucagon decrease. During the fasting portion of the cycle, the opposite occurs. These metabolic and endocrine fluctuations are risk factors in various digestive diseases (Clarke et al., 1990)

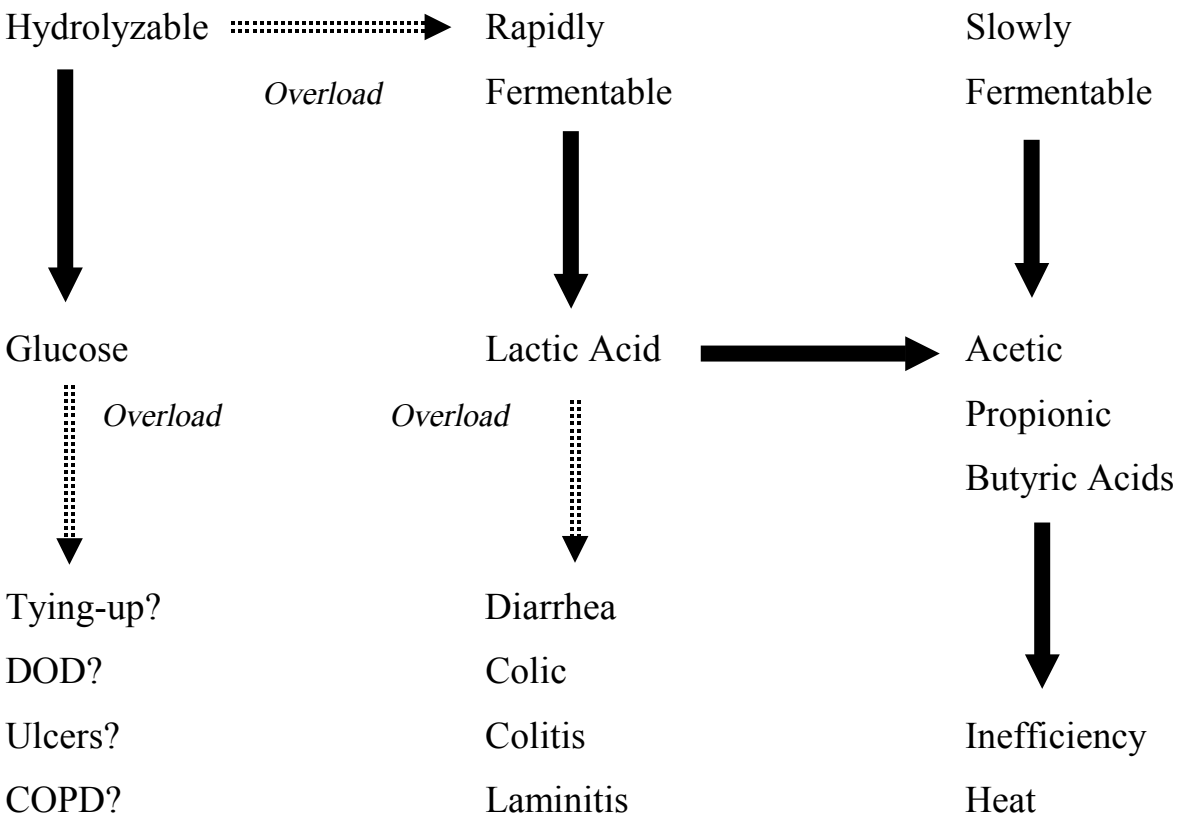

Figure 2. Carbohydrate fractions and their metabolism (adapted from Kronfeld, 1996). An overload of hydrolyzable and rapidly fermentable carbohydrates has been suggested to increase the risk factor of certain equine digestive and metabolic disorders. The slowly fermentable carbohydrates will help sustain healthy gut function, however, they are less efficient than the other carbohydrates requiring the need for added fat as the energy source

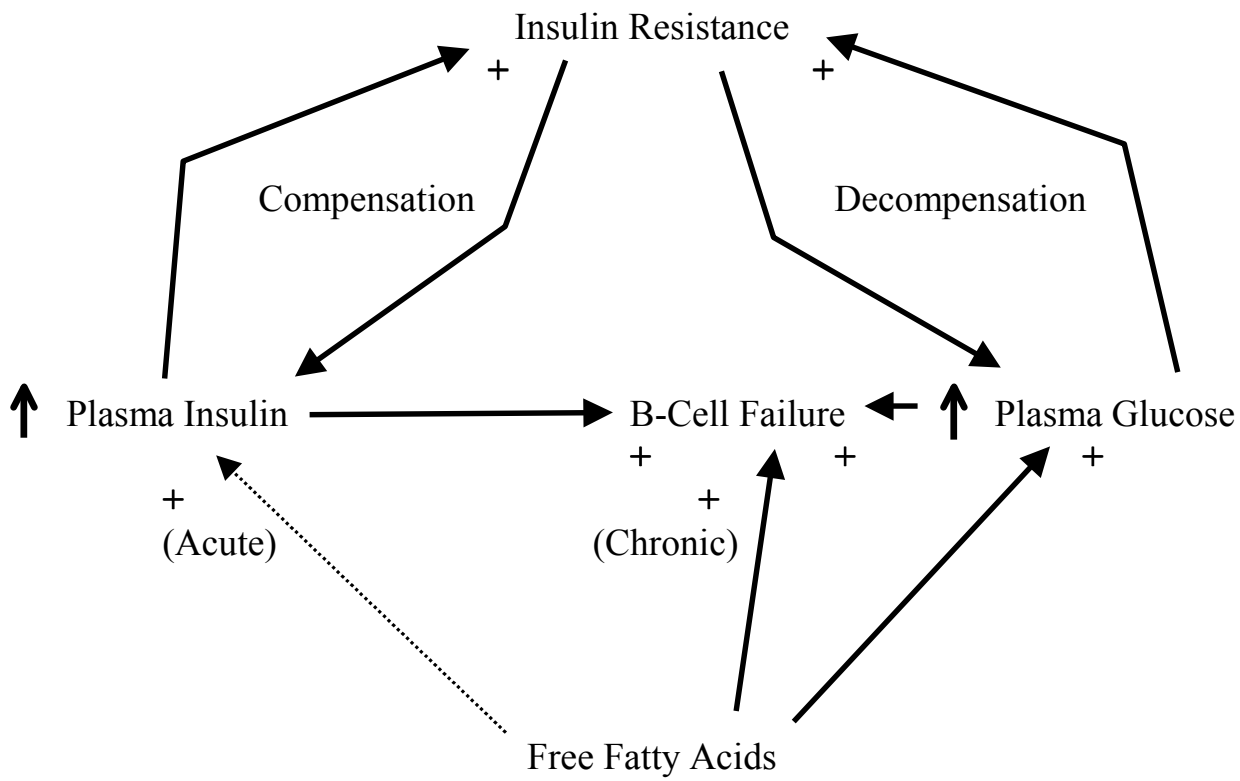

Figure 3. Proposed model for the role of insulin resistance, pancreatic B-cell function and plasma free fatty acids (adapted from Wolever, 2000)

Objective

This study will continue to determine if fat as an energy source, and fiber in a pasture supplement will be beneficial when compared to a typical concentrate high in sugar and starch. The present study compares the glycemic and insulinemic responses of typical concentrates and a supplement formulated with sufficient fat to achieve the same energy density of a typical concentrate and sufficient fiber to allow its use as a complete feed.

1. To test the difference in glycemic and insulinemic response between a typical pelleted concentrate diet, a textured sweet feed high in sugar and starch and a feed high in fat and fiber in Thoroughbred mares.

2. To test the effects of a high sugar and starch feed and a high fat and fiber feed on glucose and insulin in Thoroughbred mares during mid and late gestation, early lactation, and non-pregnancy.