

Disorders caused by feeding practices

Understanding evolution of equine metabolism provides insight into managing disorders

by Amy Gill, Ph.D.

TODAY'S RACEHORSES are selectively bred from a narrow gene pool, asked to perform unnatural exercise regimes, and then fed excessive amounts of unnatural, concentrated grain rations and cultivated, nutrient-dense forages in order to meet their digestible energy and nutrient requirements.

Contemporary management practices of these horses require confinement to stables, training and feeding schedules convenient to caretakers, and a sedentary lifestyle. As a rule, most racehorses remain confined to a stall for nearly the entire day.

All these factors have produced horses that have become susceptible to metabolic- and exercise-related disorders. A lifestyle such as the one described above is in complete contrast to that of pastured or feral horses, who spend 14 to 18 hours per day grazing and walking.

Feral and free-ranging horses consume only natural feedstuffs, such as twigs, leaves, sparse fibrous grasses, dirt, bark, wood, herbage, and flowers, always taking a few bites and then moving along. Horses in the wild or in pastures thrive in this type of environment and rarely suffer from metabolic disorders because all horses, as hindgut fermenters, can digest and metabolize fibrous feedstuffs much more efficiently than grains.

The difference between the two equine lifestyles recently has raised the question as to why some performance horses have developed metabolic disorders that appear to be linked to the relationship between the metabolism of the horse and current feeding and management practices.

Horses have evolved to be metabolically adaptable

Many contemporary breeds of horses are selected for specific genetic and physical traits. Intensive line breeding and inbreeding have been used for centuries to achieve ideal breed characteristics.

And nothing we do with these high-level performance horses can be considered natural, from selective breeding, putting a person on the horse's back, asking them to run for 1¼ miles in two minutes, eat starch, and live in what is essentially a cage. In general, high-level performance horses must be fed diets well beyond what is considered natural for an herbivore to achieve the growth and performance levels expected of them.

However, horses have evolved by nature

to efficiently store energy and nutrients in times when feedstuffs are in abundance and, therefore, survive when food is sparse. This is a normal and natural adaptive response that occurs as the growing season in various geographic locations starts and stops.

Horses have evolved to be metabolically efficient as a means of protecting their ability to survive under conditions of feast and famine, where fat deposition for later use as energy is advantageous. Wild horses—and even many domestic pastured horses—still endure the feast-and-famine feeding regime each year. But the captive performance horse, in contrast, is fed energy- and nutrient-dense feeds all year, with little opportunity for browsing and grazing low-quality feeds.

Performance horses do not get the benefit of gaining weight and storing nutrients, and then depleting these stores during periods of low intake, a cycle that appears to be necessary to maintain normal metabolic function.

High sugar in the diet leads to insulin resistance

Recently, a higher percentage of performance horses have been diagnosed with a wide array of metabolic, growth, and performance-related disorders that appear to be directly related to sedentary living, stress, and, especially, high-starch diets. Many of these horses have become resistant to the effects of insulin, the hormone released from the pancreas in response to an increase in blood glucose, which results when dietary starch is metabolized to glucose and absorbed through the gut wall into the bloodstream following ingestion of a grain meal. Insulin is required to help remove glucose from the bloodstream and assist cells in the body with storing it for later use.

But when a horse is insulin-resistant, excessive amounts of insulin have to be released to get glucose into cells. Eventually, when a horse becomes resistant to the effects of insulin, glucose and insulin levels remain elevated for four hours after feeding, and this scenario causes alterations in other hormone levels and physiological processes.

For example, even foals as young as three months of age have been reported to be insulin-resistant. This is probably an evolutionary safety net that allows the newly weaned foal to store energy and nutrients efficiently from forages during the growing season in order to survive the oncoming winter. Unfortunately, domesticated horses in this age group are just beginning to be fed

large amounts of grain and high-nutrient density forage to replace mare's milk.

There is now clear evidence that the increase in starch consumption is highly correlated with the increase of blood glucose and insulin, and it may well be a factor in the development of osteochondritis dissecans (OCD). It is believed that elevated insulin levels suppress thyroid and growth hormones that have a direct affect on the conversion of cartilage into bone. Disruption of the normal process of converting cartilage into bone after a grain meal is fed may be a factor in the development of lesions at the growth plates of long bones.

Insulin resistance also can occur in broodmares during late pregnancy and may lead to complications with delivery, as well as affect fetal growth. Today, it is more common to see very heavy, overly fat, cresty-necked broodmares than lean, fitter mares. Many of these mares are deliberately kept in this condition as it was proved years ago that there was no need to "flush" mares (keep mares thin over the winter and then in a weight-gaining state by spring) to improve fertility and conception rates and prevent dystocia (difficult delivery).

Though this is true, we may have traded healthy, lean noninsulin-resistant mares that have been allowed to fatten naturally and then become appropriately thin through winter for mares that are insulin-resistant, and prone to laminitis and other metabolic disorders.

Insulin resistance plays role in numerous disorders

Whatever the exact cause, with insulin resistance, the horse cannot clear glucose efficiently. Researchers continue to investigate why so many categories of horses have trouble regulating glucose and insulin, but the culprit behind many of these disorders, at least in part, appears to be dietary starch and the inability of the horse to efficiently metabolize large quantities.

Since performance horses are generally fed grain meals two to three times a day, the large doses of sugar derived from these meals may be involved in the onset of several metabolic disorders directly related to insulin resistance.

In addition to the clinical disorders mentioned above, insulin resistance and aberrant carbohydrate metabolism appear to play a role in such disorders as metabolic syndrome (Cushing's disease, peripheral Cushing's), laminitis, and recurrent exer-

tional rhabdomyolysis (tying up).

Dietary sugar affects cellular proteins

In mammals, high-starch diets causes a decrease in insulin receptor numbers over time, possibly as a result of the sustained, increased cellular contact with insulin. A lower number of receptor sites for insulin to bind to on the cell surface prevents insulin from entering cells and exerting its affect. This scenario certainly could be a contributing factor to insulin resistance.

Additionally, in experiments with fat cells, scientists at Johns Hopkins University have discovered direct evidence that a buildup of sugar on cellular proteins triggers insulin resistance, a key feature of most cases of human diabetes.

Scientists reported in the April 16, 2002, issue of the *Proceedings of the National Academy of Sciences* that at least two cellular proteins involved in potentiating the action of insulin were unlikely to work properly when exposed to high levels of sugar. Type 2 diabetes, the most common form in human adults and similar to insulin resistance seen in equines, occurs when muscle, fat, and other tissues stop responding to insulin's signals to clear glucose from the blood. Scientists who are studying the problem hope that understanding sugar's precise influence on insulin's activity may help improve treatment and prevention.

"Cells don't respond to insulin itself," explained Gerald Hart, Ph.D., professor and director of biological chemistry in the school's Institute for Basic Biomedical Sciences. "Instead, a whole cascade of events, set in motion by insulin, eventually causes cells to take up sugar. We now have an explanation of how sugar can affect these signals, and even a hypothesis for how high blood sugar could cause tissue damage in diabetes—by improperly modifying proteins."

Hart's laboratory discovered 18 years ago that sugar is used routinely inside cells to modify proteins, turning them on and off. If proteins have too many sugars on them, they cannot be controlled properly by the cell and are unlikely to work correctly, suggested Hart.

"We think we've come across a major mechanistic reason for insulin resistance," Hart said. "These cells developed insulin resistance simply because their proteins had more than the normal number of sugar tags attached to them."

Metabolic syndrome

Equine metabolic syndrome, replacing the term Cushing's disease, is a physiological aberration in the normal feedback loop that exists between the pituitary gland in the brain and the adrenal glands, one located atop each kidney. The exact etiology of the disease is still debated, with the original hypothesis suggesting a small tumor or adenoma affected the pituitary gland. Other researchers believe only an enlargement of

the intermediate lobe of the pituitary gland is required to produce a problem with normal feedback.

The pituitary gland secretes a hormone called adrenal corticotropin hormone (ACTH), which signals the adrenal glands to secrete a hormone called cortisol. Cortisol is the most potent glucocorticoid produced by the adrenal gland.

Cortisol acts through specific intracellular receptors and affects numerous physiologic systems, including stress response, immune function, carbohydrate regulation, vascular tone, and bone metabolism.

In a normal horse, once enough cortisol has been released, the elevated concentration signals the pituitary gland to stop ACTH production. This pituitary-adrenal feedback loop does not work properly in horses with metabolic syndrome. With these horses, ACTH is continuously released, despite high levels of circulating cortisol. Cortisol negatively affects insulin's ability to assist cellular uptake of glucose. The adrenal glands continue to produce more cortisol, and the result is elevated insulin levels, which leads to insulin resistance.

Additionally, in the case of obese horses, large amounts of fat cells called adipocytes, which in the past were thought to be nothing more than repositories for stored energy, have been shown to release a variety of hormones, or adipokines, that play a major role in regulating body mass and composition.

Fat cells in the abdominal area possess an enzyme that converts circulating inactive cortisone into active cortisol, which directly inhibits the action of insulin and leads to or creates insulin resistance. This disorder has been termed peripheral Cushing's disease. Horses affected by peripheral Cushing's have symptoms similar to regular Cushing's disease, such as increased urination and water intake, muscle wasting, and difficult to control laminitis, but they may not have the characteristic long hair (hirsutism) that does not shed out easily in spring. They also may show filling above the eyes.

This filling is fat deposition in the supra-orbital fossae. Normal horses have depressions above the eyes and you can see these depressions moving when a horse chews. Younger horses affected by the disorder may exhibit obesity with lumpy fat pads, poor immune response, chronic infections, and laminitis.

Laminitis and insulin resistance

The relationship between insulin resistance and laminitis is becoming evident as research into both disorders continues. It now is recognized that fat cells in the abdominal area of humans and horses are very endocrinologically active and essentially function as an endocrine system. As mentioned earlier, one of the products of omental adipocytes is 11-Beta-hydroxysteroid dehydrogenase 1 (11b-HSD1). This enzyme resynthesizes cortisol from its inactive metabolite, cortisone.

It also has been suggested that 11b-HSD1 may increase within the laminae of horses and ponies affected by metabolic syndrome, and this may be relevant in the propensity of these horses to develop laminitis. Among its many metabolic effects, increased circulating levels of cortisol cause high blood glucose levels and inhibit the uptake of glucose into cells, such as those in muscle, due to antagonism of insulin. Continuous high cortisol levels produce glucotoxicity, a term that has been coined to describe the deleterious systemic effects of high blood glucose levels, a significant contributor to laminitis.

Horses and ponies with metabolic syndrome tend to be "good doers" and have difficulty keeping weight off. Clinical or subclinical laminitis, where pain is not always evident, are common features in many of these cases.

Over time, horses prone to laminitis become more difficult to manage, as they seem to gain weight and suffer laminitic episodes when fed very small amounts of feed, forage, and pasture.

Starch is particularly difficult for these animals, and grain overload often leads to laminitis. Subclinical acidosis in the hindgut may actually occur in some horses every time they are fed a high-starch meal. This can lead to a kill off of certain bacteria that cannot tolerate acidic conditions. When these bacteria die, they release an exotoxin from the outer lining of their cell bodies that has an extraordinary effect on the laminae in the horse's foot.

Enzymes in the laminae called matrix metalloproteinases (MMPs) normally function to allow growth of the hoof down the foot by attaching and detaching the hoof capsule from the underlying laminae. Exotoxins from the bacteria in the gut cause a mass activation of these enzymes that appears to cause the hoof to detach from the horse. This allows rotation of the coffin bone, and the horse founders.

The key to preventing laminitis, metabolic syndrome, and insulin resistance is to feed each horse individually to maintain the correct body condition, not allow a horse to become overweight, and provide the animal access to some type of exercise on a daily basis.

More fiber, less sugar prevents obesity, insulin resistance

Certain classes of horses must be fed high-quality feeds to perform properly, such as performance horses, pregnant and lactating mares, growing foals, and underweight geriatric horses. Others, such as mature stallions, barren mares, retired racehorses, and geriatrics that tend to be easy keepers, can be fed lesser-quality feedstuffs, with protein, vitamin, and mineral supplementation as needed.

Not every horse has to eat grain every day, but horses will die if they do not eat at least 1% of their body weight daily in fiber. And using more fibrous, lower protein and energy forages may be the way to go with

these types of horses, as it allows them to forage on a more continuous, natural basis. This is healthier for the horse from a mental and physical standpoint.

It is better to supply 15 pounds of lesser-quality grass hay to an easy keeper than to feed that horse only five pounds of high-quality alfalfa hay. It also is important to remember that an idle horse with nothing to eat can develop ulcers and behavioral problems.

Now, more than ever, with so many horses developing metabolic problems, we must recognize that horses still are simply grazing herbivores. They are equipped with a digestive tract and endocrine system that can handle large quantities of fiber but are not very efficient at metabolizing, absorbing, and processing sugar. High-energy and nutrient-rich feeds are needed at certain stages of every horse's life, but when they are not required, we should give our horses a break. 🐾



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Dietary recommendations for horses with metabolic disorders

HORSES WITH any type of metabolic disorder must be managed very closely to ensure the intake of sugars and starch are minimized, if not completely eliminated, from their diet.

The use of high-fat and -fiber rations to offset the effects of blood glucose response to concentrate meals high in starch is largely becoming the standard choice of ration fed to these horses. By supplying the horse with energy or calories in the form of fat or soluble fiber, insulin resistance is curtailed because blood glucose levels remain low following a meal.

These horses also should be maintained on grass forages or forages low in soluble sugars called fructans, as these also will cause a rise in blood glucose if consumed in large quantities.

Spring and fall pasture is an excellent source of fructans. Therefore, horses with metabolic syndrome should have limited

access to pasture during these periods, either by using grazing muzzles that limit the amount of grass the horse can grab in a bite or by placing horses in dry lots.

Geriatric horses are living longer and are being kept in production and work longer than ever before. Many have higher nutritional requirements due to these factors.

However, many older horses tend to easily become overweight, which may be related to insulin resistance or metabolic syndrome (possibly as high as 70% of the equine geriatric population in the United States). Feeds high in starch are contraindicated for these horses, as the sugar in these feeds can exacerbate the problem. Older horses still being used for work that have insulin resistance should be fed concentrates high in soluble fiber and fat, with higher levels of vitamin and mineral fortification to account for losses in digestion and absorption due to deteriorating intestinal wall capacity.

Other specific nutrients that are useful in managing these disorders are magnesium and chromium. These nutrients play a role in reducing resistance to insulin. However, the addition of these nutrients to the ration should be monitored by an equine nutritionist, because excesses can cause serious nutritional imbalances.—*Amy Gill, Ph.D.*