

EQUINE METABOLIC SYNDROME:

More Unknowns Than Knowns



Horsemen have known for decades that obesity is an unhealthy condition in horses. In olden times, overweight horses were not common because horses were required to toil all day in front of a plow or behind a herd of cattle. Their reward for a day's work was suitable feed, but not enough to become too fleshy. Many wore the badges of hard work, their hides scarred from ill-fitting harnesses or overzealous spurring. In certain regions of the country, obesity was frowned upon. Peddled by unscrupulous traders of the day, hog-fat, slick horses were often eyed with suspicion at weekly trading fairs. A fat horse was suspected of being temperamental, lame, or otherwise inapt, as sound horses of honest disposition were nearly always employed.

In this age, obese horses are more the rule rather than the exception. Despite admonitions by veterinarians and nutritionists to keep horses in moderate body weight, well-meaning horse owners ply their charges with high-calorie concentrates and hay. More times than not, the result is a plump middle-aged horse that is anything but healthy or athletic. He gasps for breath when subjected to mild exer-

cise, and his limbs bear the brunt of unnecessary pounds.

Now, scientists have uncovered yet another reason to keep mature horses slim and conditioned: equine metabolic syndrome. Because it is a relatively novel discovery, scientists are just beginning to learn the intricacies of this disorder. At first, the veterinary sect could not agree on a suitable name. In the past, it has been commonly referred to as peripheral Cushing's syndrome, pseudo-Cushing's syndrome, hypothyroidism, and insulin resistance syndrome. Less common names included omental Cushing's syndrome or central obesity. A mysterious-sounding moniker evolved as well, syndrome X. Eventually, researchers agreed on the terminology proposed by the World Health Organization to designate this condition: equine metabolic syndrome.

As the accurate diagnosis of equine metabolic syndrome becomes more widespread, researchers are learning more about the causes, signs, and treatments of the disorder.

Insulin Resistance: The Root

Equine metabolic syndrome is characterized foremost by insulin resistance, defined as a peculiar physiological response to the ingestion of foods that are eventually broken down to glucose or other sugar molecules. Abundant in certain feedstuffs commonly fed to horses, glucose causes a normal state of hyperglycemia or elevated sugar in the blood. Hyperglycemia prompts the release of insulin from the pancreas, which encourages the removal of glucose from the bloodstream by fat or skeletal muscle cells. Once in the cells, glucose can be put to work immediately to fuel exercise or growth or stored as glycogen or fat for later use. Insulin resistance implies that either the central tissue



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(liver) or the peripheral tissues (the skeletal muscle or the fat cells) are relatively insensitive to the action of insulin or that the quantity of insulin released by the pancreas in response to hyperglycemia is diminished. This leaves glucose circulating in the blood. Because glucose levels do not drop, the pancreas continues to discharge insulin, leading to elevated concentrations of insulin in the bloodstream, a condition known as hyperinsulinemia.

What predisposes a horse to insulin resistance? Little is known on this front but responsibility might rest on genetic, gestational, and environmental factors. In humans, causative factors are well documented: aging, pregnancy, smoking, reduced physical activity, and obesity. In genetically susceptible humans, glucose intolerance can lead to noninsulin-dependent diabetes mellitus.

In the equine model, obesity appears to be related to the onset of metabolic syndrome. One suggested cause is that certain fat cells produce cortisol, among other hormones, which interferes with the ability of insulin to move the glucose into cells. Because obese horses have more fat cells, more cortisol is produced and there is greater interference with insulin. This explains why weight reduction is effective in increasing insulin sensitivity.

Not all fat horses are insulin resistant. Current beliefs hold that horses whose fat cells produce high levels of leptin as well as cortisol are the ones prone to insulin resistance. Leptin is not believed to cause insulin resistance but is found to be higher in horses that are insulin resistant.

Age and diet may be directly related to the development of equine metabolic syndrome. Age is thought to decrease the horse's sensitivity to insulin. Meals high in starch and sugar cause significant spikes in blood glucose and insulin, and years of consuming such meals might lead to insulin resistance.

Obesity-Associated Laminitis

An overwhelming clinical sign of equine metabolic syndrome is laminitis, but not the disabling, painful disease related to gastrointestinal failure and endotoxemic insults. The laminitis exhibited by these obese, middle-aged horses tends to be mild. On occasion, so minimal are the laminitic episodes that knowledgeable, conscientious horse owners cannot vouch definitively for any clinical signs of lameness. The hard evidence speaks a different tale, however. Abnormal hoof growth occurs. Dropped soles, unusual growth lines, and separation of the hoof at the white line are frequently observed. More damning, however, is the shifting of the coffin bone within the hoof capsule, which is obvious upon radiography.

But what causes the laminitis? Over the years, the root of laminitis in obese mature horses has been attributed to (1) endocrine disorders, namely hypothyroidism; (2) aggra-

vation of a preexisting laminitis caused by endotoxemia (overconsumption of grain, for example); and (3) mechanical inadequacy due to the stress of excessive weight on soft tissues of the leg.

Regardless of the cause, laminitis is the result of changed circulation to the laminae, the interconnected layers of tissue that insure the integrity of the hoof. In obese horses, researchers believe that insulin insensitivity and vascular spasms may incite changes in the endothelial tissue of the laminae. On a physiological level, this concurs with the circulation problems observed in human patients with noninsulin-resistant diabetes mellitus. Despite well-founded theories, a definitive cause for obesity-associated laminitis remains elusive.

Diagnosis

At this time, diagnosis of equine metabolic syndrome is based on description and physical characteristics, results of glucose-tolerance testing, and elimination of similar conditions.

Description and physical characteristics. Affected horses are usually between the ages of eight and 18, though numerous patients have fallen outside this range. Horses and ponies of nearly all breeds have been diagnosed, though Morgans, Peruvian Pasos, Paso Finos, domesticated Spanish Mustangs, and warmbloods appear to be especially predisposed to the syndrome. As a group, ponies tend to become overweight more readily than horses and are often inclined to suffer from laminitis.

What's more telling than either age or breed of the patient is distribution of exterior body fat. Areas of unusual fat accumulation include the top of the neck (commonly called the crest), over the shoulders, and the rump (including deposits over the croup and just above the tailhead). Significant fat sometimes settles in the sheaths of geldings, so much so that they may appear swollen.

Affected broodmares show unusual estrous cycling, which makes them incredibly difficult to get pregnant.

Anecdotal evidence by owners is also instrumental in diagnosing equine metabolic disease. Owners frequently describe their horses as easy keepers, finding it virtually impossible to reduce the weight of these horses by calorie restriction alone. Many report that high-calorie feeds such as grain are not being fed.

Results of glucose-intolerance testing. Veterinarians often perform an oral or intravenous glucose tolerance test on horses they suspect to be insulin resistant. Following the administration of glucose, insulin and glucose responses are measured and compared against the responses of normal horses. This test should be performed on a fasted animal so glucose from a recent meal does not shade the results of the assessment.

According to some equine veterinarians, the only truly effective method of diagnosing insulin resistance is the “euglycemic hyperinsulinemic clamp.” The procedure is complicated, time-consuming, and can be expensive. Because of these limitations, veterinarians typically diagnose on clinical signs alone.

Elimination of similar conditions. In the past, veterinarians often misdiagnosed equine metabolic syndrome, suggesting hypothyroidism or Cushing’s syndrome instead.

In humans, hypothyroidism occurs when the thyroid gland fails to produce sufficient thyroid hormone, leading to clinical manifestations of thyroid insufficiency such as low metabolic rate and tendency to gain weight. In horses, neither obesity nor laminitis develops in mature horses from which the thyroid gland has been removed. Thyroid stimulation tests, designed to gauge thyroid function, fail to identify hypothyroidism. Additionally, the thyroid glands from horses affected with equine metabolic syndrome appear normal. Hence, it is clear that the combination of obesity and laminitis are not always ramifications of inadequate thyroid hormone production.

Also mistaken for equine metabolic disease is Cushing’s syndrome. This endocrine disorder involves dysfunction of the pituitary pars intermedia. Using tests most commonly administered to verify Cushing’s syndrome (including the dexamethasone suppression test), veterinarians yielded negative results on these obese, laminitic horses. The pituitary glands of these horses also revealed no pathology, leading researchers to believe that Cushing’s syndrome was not to be blamed for the signs. Misdiagnosis of Cushing’s syndrome is understandable as a few of the clinical symptoms are shared by individuals with equine metabolic syndrome: abnormal distribution of fat, elevated circulating insulin, glucose intolerance, predisposition to laminitis, and infertility. Other clinical features of Cushing’s are not normally documented in horses suffering from metabolic syndrome, notably an excessively shaggy coat that fails to shed and increased drinking and urination.

(For more information on Cushing’s syndrome, check out Equineews, Volume 5, Issue 3 by going to www.ker.com/EQmag/v5n3.html. The complexities of this disease, including proper care of afflicted horses and ponies, are detailed.)

Prevention and Treatment

Diet. Too many horses eat too many groceries; it’s that simple. The objective of all equine feeding programs should be straightforward: provide sufficient feed to satisfy nutrient requirements for growth, maintenance, or work while maintaining optimal body condition. Optimal should not be confused with maximal or obese. Optimal body condition

can be defined as a nutritional state in which the animal’s ribs can be felt with gentle palpation but cannot be seen.

Horses become overweight because they consume too many calories in relation to the work asked of them. Those that perform mild to moderate work may need little more than good-quality grass hay or pasture and a complete vitamin and mineral supplement, particularly if they are good keepers (able to maintain weight easily). This ration, though simple, is considered low in starch, one important step in dodging equine metabolic syndrome.

Mature horses diagnosed with metabolic syndrome should not be given grain, grain mixes with molasses, or unlimited access to pasture. A balancer pellet (concentrated protein, minerals, and vitamins) can be given to provide essential nutrients without unwanted carbohydrates. If a horse requires additional energy, nonstarch alternatives such as corn oil or rice bran can be fed.

In young growing horses, feeding grain in large quantities should be discouraged. Horses that are overfed as youngsters are the very ones that are likely to be obese in midlife and become prone to laminitis. Termed “easy keepers,” these horses harbor disproportionate quantities of fat within their abdomens, which in turn makes them more susceptible to metabolic syndrome.

Exercise. In addition to changes in diet, an exercise program should be implemented to slim down overweight horses or prevent them from becoming too heavy. Exercise can be provided in numerous ways: riding, driving, ponying, round pen work, hand walking, longeing, or long-lining. Not only does exercise ward off obesity, research has shown that it improves insulin sensitivity in horses and ponies. A combination of diet changes and increased exercise is the most effective way to increase insulin sensitivity. Exercise programs must be designed with the individual in mind. A realistic assessment of the horse must be made and an appropriate exercise regime chalked out, especially with horses that are old, unfit, or of questionable soundness. If a horse has suffered a mild bout of laminitis, consultation with a veterinarian and farrier is warranted before any exercise is started.

Medication. No medication is suitable for treating metabolic syndrome. The two most commonly used medications for the management of Cushing’s syndrome—pergolide and cyproheptadine—have proven ineffective for treatment of metabolic syndrome. Both medications have a tendency to limit pancreatic insulin secretion, which only adds to the problem.

Equine metabolic syndrome has emerged on the veterinary scene as a health threat to middle-aged, obese horses. Though deep understanding of the disease has not occurred, a diet and near-daily exercise program that emphasizes moderate body condition may be just enough to elude this dangerous disease. ☺☺



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