Equine Metabolic Syndrome and Insulin Resistance in the Horse – Keeping up with Evolution
Is it as simple as spinning sugar into fat?

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Horses are relatively free of many of the health concerns that afflict people. They don't tend to develop high cholesterol, heart disease, or various other ailments common in our human population. However, one by-product of modern the equine lifestyle for many of our horsey friends is their tendency to put on extra weight. Nowadays, fat horses are an all-too-common sight in fields and stalls around the country. The odd thing is that many of us don't see that as a problem. Of course, a horse's ideal weight depends on many factors, ranging from body type and disciple to activity level. In some breeds and disciplines the fuller look provided by fat deposits along the hindquarters, neck, and withers is considered desirable. Subconsciously many people equate equine plumpness with good health. Unfortunately, sometimes what we like isn't necessarily what's best for our horses. Excessive weight can take a toll on soundness, performance, and overall good health, even shortening a horse's life.

In horses as with people, obesity increases the risk of several serious health problems. Obviously a fat horse is likely to have exercise intolerance - breathing heavy, sweating intensely, and tiring rapidly in response to exertion. More subtle is the toll extra weight can take on tendons, ligaments, and joints. Soft tissue strains are always a risk. Joints that may already be compromised by arthritis are vulnerable to the ill-effects of a heavy load. Obesity also has systemic effects. One of the most serious is insulin resistance (IR), a condition similar to adult onset (type-2) diabetes in people.

Insulin resistance is a condition that develops when the body's cells do not correctly respond to insulin. Insulin is secreted from the pancreas when triggered by a rise in blood glucose after eating a meal, especially a meal containing sugars or starches. Normally, insulin stimulates cells to take up this circulating glucose for growth, repair, and other functions. Once in the cell, glucose is used for energy or converted to glycogen (stored muscle energy) or fat for storage. With insulin resistance, the cells (for various reasons) do not respond to normal levels of insulin, depriving the cells of the glucose they need. At first the body simply produces more insulin and these higher levels succeed in regulating blood glucose temporarily. Gradually though, the body fails to respond to the higher and higher levels of insulin, and eventually exhaustion and failure of the insulin-producing cells of the pancreas occur. At this point in people, adult onset non-insulin mediated (type-2) diabetes mellitus is said to exist.

A literature review of the last 50 years shows a small number of cases of documented equine diabetes that were caused by either pancreatitis or by tumors of the pituitary gland. Recently, however, researchers have speculated that horses may be much more inclined to develop a resistance to insulin that can lead, as in humans, to type-2 diabetes mellitus. These horses may mimic Cushingoid horses and they may exhibit a variety of clinical signs and disease patterns that have been confusing veterinarians for some time.

Just as insulin resistance is on everyone's mind in human medicine, it is rapidly becoming a hot topic in equine medicine. As in people, insulin resistance appears to be related to obesity and altered fat metabolism in the horse. While the ultimate clinical outcome of insulin resistance in horses is seldom true diabetes, insulin resistance is more commonly being viewed as an important underlying contributing factor to the pathogenesis of inflammatory and vascular diseases of horses. Insulin resistance has been suggested as a contributory factor in hyperlipidemia, pituitary adenoma (PPID), osteochondrosis, reproductive failure, acute laminitis, and mild peripheral laminitis [1, 2, 3]. Some forms of exertional rhabdomyolysis may involve increased insulin sensitivity as well.

Obviously, what is now known about insulin resistance began with human studies and mouse models. Scientists were searching for a relationship between high intake of sugar and saturated fat, the function of insulin in different tissues, and the consequences of excessive circulating fats on inflammatory processes and vascular function. In 1988, a Stanford endocrinologist suggested that a high-carbohydrate diet in people aggravated or exacerbated a predisposition to insulin resistance, which he identified as a primary factor in the etiology of obesity, non-insulin mediated (type-2) diabetes mellitus, hypertension, and coronary heart disease. This was surprising because the high-carbohydrate diet was, in effect, the same as the low-fat diet championed for 40 years by the American Heart Association to lower blood cholesterol and ward off coronary heart disease! This earlier focus on cholesterol preceded knowledge of the adverse effects of hyperglycemia on vascular endothelium and blood clotting, which contributes to coronary heart disease in people. This endocrinologist, Dr. Gerald Reaven, received the Banting Medal and presented the Banting Lecture to the American Diabetes Association [4]. In that lecture, he proposed the existence of "a series of related variables – *Syndrome X* – that tends to occur in the same individual and may be of enormous importance in the pathogenesis of coronary artery disease [in people]".

In humans, the path to type-2 diabetes is generally paved by excessive consumption of starch and sugar that leads to synthesis of fat, which circulates in the body and deposits into a refillable fuel tank otherwise known as "the beer belly." These levels of sugar and fat lead to changes in lipid metabolism that favor fat synthesis and reduce fat burning [5]. Obesity and lack of exercise are primary risk factors for insulin resistance in humans, and the risk of developing type-2 diabetes mellitus increases with the severity of the obesity [6]. Blood fatty acid concentrations increase when adipose tissues reach their maximum capacity for fat storage and the inhibitory effects of insulin on hormone-responsive lipase is reduced [7]. As a result, the influx of fatty acids into tissues (including those of skeletal muscle) increases. This phenomenon is sometimes referred to as lipid toxicity because intracellular lipids disrupt insulin receptor signaling in myocytes (muscle cells) or *B*-cell in the pancreas [7,8]. What wasn't realized until recently was that the fat cells themselves produce endocrinologically active substances such as resistin, cytokines (TNF-alpha, IL-6), and leptin, which

make other cells in the body less sensitive to insulin. In other words, the fat deposits become their own endocrine organ that continues to drive the insulin resistance!

This recognition that adipose (fat) tissue releases a plethora of multifunctional substances has led to substantial rethinking of the mechanisms responsible for the risk of cardiovascular and endocrinological diseases associated with obesity [in people] [9]. Collectively, the constellation of products secreted by adipose tissue have been referred to as adipokines (or adipocytokines) [10]. In excess of 100 different adipokines have been described that impart physiological relevance to lipid and glucose homeostasis, inflammation, hemostasis, osteogenesis, hematopoiesis, complement activities, reproduction, angiogenesis, blood pressure, and feeding behavior [11]. In fact, adipokines have recently been claimed to represent the missing link between insulin resistance and cardiovascular disease [in humans] [12]. It is the secretion of inappropriate quantities of adipokines over time that represents the basic pathophysiological foundation of many (if not most) of the health consequences of obesity [13]. A complete discussion of the presently known adipokines is beyond the scope of this manuscript; however, there has been substantial interest in some specific adipokines. Many adipokines affect and modulate the immune system with a net effect leading to a state of chronic systemic inflammation. Although several diverse functions have been attributed to leptin, an important function is its ability to signal within the central nervous system to inhibit appetite in the face of developing obesity. That appetite is not necessarily suppressed suggests that interference with leptin signaling may play a role in the development of obesity in some individuals with elevated plasma leptin concentrations. Conceptually, adipose tissue has been transformed from a passive energy-storage organ to an endocrinologically significant gland [14].

In 2001, David Kronfeld stole the term Syndrome X from human literature and applied it to a group of metabolic disorders associated with long-term consumption of grain and molasses in pelleted or textured feeds and probably involving insulin resistance [15], calling this condition **Equine Syndrome X**. He deduced that meals of sweet feed set up a feeding-fasting cycle of high plasma glucose, high circulating insulin, and rebounding counter-regulatory hormones. It had been observed for years that obesity, hyperinsulinemia, and laminitis sometimes develop concurrently in non-Cushingoid horses. This group of disorders, once named Equine Syndrome X, was later redefined as equine metabolic syndrome (EMS) [3, 16, 17]. The term *metabolic syndrome* is also taken from human medicine and is really short for "the metabolic syndrome of risk factors for type-2 diabetes or hypercholesterolemia and coronary heart disease" [3, 18]. It is defined by the presence of insulin resistance or type-2 diabetes mellitus with at least 2 of the 4 additional risk factors of obesity/visceral adiposity, dyslipidemia, microalbuminemia, or arterial hypertension [in people] [19]. The use of the term equine metabolic syndrome has been controversial, because some specific aspects of the human syndrome are lacking for horses. Microalbuminemia and hypertension have *not* been reported in obese horses, but high blood lipid concentration has been measured in ponies and donkeys with insulin resistance and many of them were obese [20, 21]. Also, plasma triglyceride and total cholesterol concentrations *do not* differ between clinically normal and hyperinsulinemic ponies [20], but a positive correlation had been documented between plasma insulin and triglyceride concentrations in donkeys with naturally occurring hyperlipidemia [21]. So horses don't really suffer from dyslipidemia, microalbuminemia, or hypertension as people do, but we certainly recognize high circulating blood glucose, high insulin levels, and (sometimes) high lipids in blood samples of obese horses.

Nonetheless, in the past few years the term **equine metabolic syndrome (EMS)** has been adopted to tie two important physiologic disturbances, obesity and insulin resistance, to laminitis [3, 22]. EMS is defined by the presence of obesity and/or regional adiposity, insulin resistance, and prior or current **laminitis**. Regional adiposity takes the form of expanded adipose tissue deposits within the neck (cresty neck), prepuce, and tailhead. Laminitis varies in severity and owners often report that the first episode occurred when the horse was grazing on lush pasture. Alternatively, laminitis can develop insidiously without an identifiable cause. Some horses have clinical laminitis, whereas others only show evidence of prior laminitis in the form of divergent growth rings on the hooves (founder lines) or radiographic findings consistent with third phalanx rotation.

All of the pieces of the puzzle must be assembled before we can fully understand the association between insulin resistance and laminitis in horses and ponies. It is reasonable to infer that, by virtue of relatively **increased weight** bearing, obesity could contribute to an increased likelihood of laminitis simply through the effect of the additional force of distraction (weight) at the level of the hoof-lamellar interface. More scientifically, however, compelling experimental data [23] suggests that glucose is essential for the health and strength of the equine hoof-lamellar interface. Insulin resistance decreases the amount of glucose getting into hoof tissue cells, which starves them. **Glucose starvation** weakens the link, which leads to separation and dramatically increases the risk for laminitis. Insulin resistance can also **alter blood flow** to the foot. The hoof may be uniquely susceptible to compromise from the endocrinological circumstances that potentiate vascular smooth-muscle contractions (vasoconstriction) in the horse [24]. Vasoconstriction at the hoof-lamellar microvasculature then decreases tissue perfusion and oxygen delivery to the hoof structures. These changes closely mimic those seen in peripheral neuropathies in human diabetics.

What triggers the laminitis episode itself? Certain starches and sugars (non-structural carbohydrates) in the diet play an important role in this process. Excessive sugar consumption exacerbates insulin resistance in the horse like it does in diabetic humans. Another response to excessive carbohydrates in the diet, distinctly equine in nature, is alterations to the bacteria found within the large intestine. These changes in bacterial flora are thought to increase the production of some yet unidentified triggering factors for laminitis that may include exotoxins, endotoxins, or vasoactive amines [25].

The underlying **metabolic status** of the horse is of primary importance when discussing EMS, because affected horses tend to become obese and appear to require fewer calories to maintain body weight. Horses of any breed can develop insulin resistance if

they become obese, and it only takes a bout of laminitis to turn a diagnosis of insulin resistance into a diagnosis of EMS. It has been suggested that some horses are genetically predisposed to obesity because of adaptations to survival on poorer quality forages [3, 26]. Certain breeds (e.g., ponies, Morgans, Paso Finos, Arabians, and Quarter Horses) do appear to be over-represented. According to this theory, consumption of concentrated feeds or grazing on rich pastures might therefore promote obesity in susceptible horses. Genetic and environmental factors are likely to be important in the development of obesity in horses, and it is interesting that all of the insulin resistant horses in most studies are 10 years of age or greater, which suggests that time is required for environmental factors to alter glucose metabolism [27]. Not all obese horses become insulin resistant, but obesity should be addressed to lower the risk of insulin resistance, which raises the risk of laminitis [28].

A **pre-laminitic metabolic syndrome (PLMS)** has been described in healthy ponies [23]. It is analogous to the pre-diabetic metabolic syndrome in apparently healthy people. In this study, apparently healthy Welsh and Dartmoore ponies were split into two groups: those previously diagnosed with pasture laminitis and those without. Pedigree analysis revealed a dominant mode of inheritance for PLMS, with partial suppression in males and partial suppression by environmental factors. The latter could include thresholds for grass fructan and clover starch and hence be amenable to nutritional intervention [29]. A diet of fat and fiber improved metabolic regulation or, looking at it another way, unimpaired metabolic regulation, in these ponies compared with the sweet feed sugar and starch diets. Adding interval training had a beneficial effect on insulin resistance as well, with improved response in those eating the fat and fiber diet compared to the sugar and starch diet. Management of insulin resistance will lower the risk of laminitis, and one of the cornerstones of management is diet. In short, insulin resistant horses need to exercise more and take in less sugar.

Recently there has been substantial interest in the implications of oxidative stress on equine health, especially from the perspective of laminitis [30] and **pars pituitary intermedia disease** (PPID or **Equine Cushing's Syndrome**) [31]. It is possible that the development of insulin resistance in obese horses leads to the development of a pro-inflammatory state throughout the vasculature, which is the case in the human metabolic syndrome. By doing so, insulin resistance may, in turn, promote the risk of laminitis (discussed above). It has been previously suggested that obesity and insulin resistance represent risk factors for the development of PPID in horses and ponies [32]. It has been shown that hyperglycemia generates oxidative stresses – the production of oxygen free radicals that can damage many kinds of tissues. Oxidative damage in portions of the brain have been linked to the development of Cushing's disease (pituitary gland disease) in humans. It is intriguing to ponder the possibility that chronic hyperleptinemia and chronic hyperglycemia might contribute to the pathophysiology of PPID by virtue of its actions in the paraventricular nucleus of the brain. In other words, PPID can be the geriatric condition of the chronic-refractory insulin resistant horses in our population. More research is being done along these lines to prove it.

It is now accepted that lower circulating thyroid hormone (T3 and T4) concentrations are a consequence rather than a cause of the horse's metabolic state, and these concentrations can be attributed to **secondary hypothyroidism**. The response to thyroid medication can be explained. The supplement exerts a pharmacologic effect to boost metabolism - obese horses lose weight (increased activity), frequency or severity of laminitis episodes (insulin sensitivity increases) are reduced, reproductive performance improves, and physical appearance improves. Weight loss may occur too slowly if the horse has a very efficient metabolism or if it cannot be exercised because of laminitis. In this situation, levothyroxine sodium can be administered for 3-6 months to accelerate weight loss and improve insulin sensitivity. This treatment is not a substitute for dietary management, and treated horses with free access to pasture may remain obese because feed intake increases when on this drug.

Prevention is the preferred approach. That means feeding a sensible, balanced diet and providing the horse with sufficient exercise to keep it from becoming obese. Once the horse is afflicted with EMS, the first step in a treatment approach should be a weight-loss program. This means decreased food intake and increased exercise. If the horse already has developed laminitis, increased exercise might not be an option in the early going. The most important aspect of feeding a horse with metabolic syndrome is limitation of nonstructural (soluble) carbohydrates in the diet.

Nonstructural carbohydrates (NSCs) are the sugars and starches inside plant cells. **Structural carbohydrates** make up the cell walls of plants, primarily cellulose and hemicelluloses, both referred to as **fiber**. NSCs are primarily digested in the foregut - the stomach and small intestine. Enzymatic digestion in this region quickly breaks down starches into glucose, the body's main energy source. This, in turn, causes a rise in the horse's glycemic response. The increase in blood sugar stimulates the release of insulin. The glycemic response triggered by traditional feedstuff varies, but corn, oats, barley, and molasses create some of the biggest swings. Structural carbohydrates (fiber) are digested in the hindgut – the cecum and large intestine. This is where horses are designed to have the bulk of their digestion occur. The digestive action of the structural carbohydrates is performed primarily by microbes that ferment the fiber, and digestion of structural carbohydrates is slower than that of NSC digestion. Fiber does not appreciably elevate the glycemic response. Most horses are well-equipped to digest a diet comprised of both types of carbohydrates in varying proportions. However, if a horse consumes more NSCs than the foregut can fully digest, the extras spill over into the hindgut, where NSC sugars are fermented by lactic acid-producing microbes. The resulting increase in acidity of the hindgut leads to digestive disorders.

Most insulin-resistant horses are maintained on a **hay diet**. Nutritionists often advise owners to choose the most nutritious forage they can afford, but fat horses don't necessarily need the best of the best. The cornerstone of a good insulin resistant diet is a low-sugar grass hay with a low glycemic index. It is important to collect hay samples and analyze the carbohydrate content (Dairy One Forage Laboratory 800-496-3344, and Equi-analytical Laboratories 877-819-4110). Particular attention is paid to the

nonstructural carbohydrate content (also called soluble carbohydrates) of the hay. Owners of obese horses should try to purchase hay containing less than 12% NSC, and insulin-resistant horses should ideally be fed hay containing less than 10% NSC. Grass or alfalfa hay can be fed as long as the NSC content has been measured.

It is impossible to estimate the NSC or overall nutritional value of hay by its type or appearance, so it must be analyzed. There are, however, some general guidelines. Hays made from grain crops tend to be very high in NSC (wheat, rye, barley, and oat hay are high in sugar, especially when cut just before the heads form). Avoid cuttings from plants that have been subjected to drought stress or weather extremes, which can drive up sugar content. The more mature the hay is when it's harvested, the lower the sugar and calorie content and the higher the fiber. Any kind of grass can have high sugars under certain conditions, but perennial rye, fescue, and brome generally show the highest numbers in experiments done to purposely increase NSC. The lowest-sugar hays tend to be Bermuda and other warm-season grasses grown in cloudy, humid climates. Hay that was rained on between cutting and baling (and isn't moldy!) will generally have a low-sugar content. If you can't find low-sugar hay, it's possible to reduce the sugar content by soaking it. Soaking hay in room-temperature water for an hour removes an average 30% of the sugar, with a similar reduction in hay soaked in hot water for 30 minutes. Another stable of an insulin resistant diet is **beet pulp**, preferably a brand with no molasses added. If your beet pulp contains molasses, rinsing, soaking, and straining it will remove the substance. Beet pulp is low in soluble carbohydrates, giving it a low glycemic index. Studies have shown the glycemic index of beet pulp to be essentially zero, making it an excellent choice to replace as much as 40% of the horse's daily hay intake, at a rate of approximately one pound (dry weight) of beet pulp per two pounds of hay. Clients should also weigh their feeds to ensure that correct amounts are fed.

Whether your horse eats succulent **pasture grass** or the dried form as hay, the forages they consume are better quality than they were many years ago. The forage species have been improved, resulting in more nutrients per pound. These genetic hybrids and improvements in plant physiology were driven by the food-producing animal industry (beef, dairy, etc.). That's not necessarily bad, but we need to be aware of it and manage it. Your pasture grasses may provide more calories than your horse needs. Horses have proven that they will eat beyond their nutritional needs when given the opportunity. Studies have demonstrated some individuals will consume 3.3% of their body weight while grazing pasture. The goal is to grow healthy, vigorous grass with lime, fertilizer, and rotational grazing, then limit access so horses don't overeat.

Persistently hyper-insulinemic horses should be held off pasture altogether, with some affected horses allowed to graze again once the serum insulin concentration has returned to normal after dietary intervention and exercise. However, horses should be reintroduced to pasture gradually, and the insulin response should be assessed again after this change in diet. Gradual reintroduction to pasture can be accomplished by using a grazing muzzle, restricting grazing time (<2 hours), or confining the horse to a small section of the pasture. Horses with a history of insulin resistance and laminitis are at greater risk for developing **pasture-associated laminitis**, so they must always be kept off pasture when the grass is in a dynamic phase. Dynamic phases occur when the grasses grow rapidly in the spring or summer after heavy rains or when the grass stores energy in preparation for drought or winter. Some horses with EMS can never be returned to pasture because they are too sensitive to seasonal changes in grass nutrient content.

Horses suffering from 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 the diet. Reduce sugars and starches by eliminating all grain products, including oats, corn, barley, and wheat bran in any significant amount. Check the ingredients label of pellet feeds and supplements to determine whether they are made of grain or grain products. Feedstuffs that contain sugar or molasses should be eliminated too unless the sweet coating can be rinsed off. It's also necessary to stop feeding apples or carrots. Even in small amounts, these seemingly healthy treats contain enough sugar to trigger problems in susceptible horses.

Insulin-resistant horses that are thin or being worked may require additional calories, but care must be taken to provide energy without exacerbating insulin resistance. Affected horses may be leaner but still have a cresty neck and enlarged fat pads. Sweet feeds should never be fed to these patients because these feeds contain sugars and starches that induce a higher glycemic response and potentially exacerbate insulin resistance. Affected horses should be fed a commercial pellet feed with a low NSC content. The use of **high fat-high fiber rations** (instead of concentrate meals high in starch) is largely becoming the standard choice of rations fed to these horses. A new generation of feeds is now available to help meet this challenge (see Appendix 1). By supplying the horse with energy or calories in the form of fat or fiber, insulin resistance is curtailed because blood glucose levels remain low following a meal. These feeds contain sugar beet pulp, which is primarily digested within the large intestine and has a minimal effect on the glycemic response. Molasses-free sugar beet pulp can also be fed alone as a less expensive option.

Exercise is beneficial because it promotes weight loss by increasing energy expenditure and improves sensitivity to insulin. Horses with active laminitis cannot be exercised until the hoof structures have stabilized, but all other horses should be exercised daily or every other day. Walking on a lead rope, exercising on a lunge line, turnout in a dirt paddock with no grass, and riding should be encouraged. If an insulin resistant horse is sound enough to exercise, it is wise to make every effort to get him moving on a daily basis, as even moderate exercise has been shown to decrease insulin resistance. Exercise stimulates the uptake of glucose by muscle cells using mechanisms that do not require insulin.

In summary, prevention is best, but if your horse has become overweight, management of insulin resistance will reduce the risk of laminitis and PPID (Cushing's disease). The most effective way to improve insulin sensitivity in obese horses is weight loss. Here is a rough plan to follow:

1. Eliminate grain and pellets from the diet. Give a handful of beet pulp (that does not contain molasses) or low-sugar pellet feed as a treat if necessary, but that is all. Do not feed apples, carrots, or sugar cubes. Sugar aggravates insulin resistance in the same way that it does diabetes mellitus in people. If you can't kick the habit or your horse is tearing down the barn because his feed tub is empty, consider a low-calorie feed formulated for overweight horses. Such feeds – labeled specifically for weight loss or weight control – often come in a pellet or nugget form and have less fat and calories per pound. A balancer pellet is also recommended. Horse's don't understand portion sizes; they only know "did I eat or not?"

Dispense feed by the pound and not the 'scoopful' - a scoop is not a regulated standard unit of measure. Don't mix feeds unless you are a nutritionist. Equine feed companies spend millions of dollars to create feeds that offer balanced nutrition. When people mix them, they remove that balance.

Because some horses find these low-NSC feeds less palatable, it may take up to 7 days for a horse to adapt to the new feed. It is advisable to provide smaller amounts of concentrates more frequently and to feed hay first in an attempt to lower the glycemic response. It is important to reassess the horse 2 weeks after starting a new diet by measuring the serum insulin concentration. Minor changes in the serum insulin level can be attributed to day-to-day variability, but an increase of greater than 10 IU/ml signals that the feed is exacerbating insulin resistance.

2. Exercise an obese horse as often as possible, as long as lameness associated with laminitis has resolved. Increase the amount of exercise gradually with a goal of five days of exercise per week. Trotting and exercising on a hillside will increase calories burned. Keep in mind that short rides are better than none at all. To increase fitness, a horse must work until he breathes hard. If he does not do this during your workout, he's ready for more challenge. However, to minimize the risk of fatigue and injury, add distance or speed to your routine each week, but never both at once. Days off allow a horse to adapt and recover from work. An unfit horse may need every other day off; a fitter horse needs two days of rest per week.

Don't forget to add variety. Ride outside the ring as much as you can to help keep your horse's mind fresh and protect against stress injury resulting from repeatedly going in small circles.

Your horse will provide the best indicators of the program's effectiveness. If he's eager in his work, you're on track. If he's sore or sour, you may be pushing too hard and need to drop back a level. Horses with a lot of heart may not tell you when they are being pushed too hard. Of course, as you increase your horse's exercise, you'll need to monitor his diet to ensure he is burning more calories than he's ingesting. Don't undermine your efforts by giving him an "extra scoop" of grain or treats because he's working hard.

Other fun idea - take photos, use a weight tape, and keep a log.

3. Horses with laminitis must be taken off pasture completely until their feet have recovered and obesity and insulin resistance have been controlled. Keep the horse in a dirt paddock instead of a stall to encourage exercise and reduce stress.

4. Feed grass hay with low non-structural carbohydrate (NSC) content to horses with insulin resistance. Have a hay sample analyzed to determine the carbohydrate content, or purchase bagged forages.

5. Obese horses require a weight-loss diet. Simply eliminating grain and pellets from the diet is sufficient to induce weight loss if the horse has been overfed. Suspend pasture access while weight loss is being induced because grazing represents an unregulated source of calories. Ideally, keep the horse in a dry lot to allow exercise, which increases calorie consumption. Reduce the amount of hay fed. Feed an obese horse 1.5% of its ideal body weight in hay, which is equivalent to 1.5 pounds hay per 100 pounds of (ideal) body weight. Use a kitchen scale to weight the hay or purchase a hanging scale at a feed store. Divide the daily amount of hay into two to four feedings.

The risk of laminitis triggered by diet (grain founder or grass founder) is high as long as the horse remains obese and insulin resistant. Eliminate grain from the diet of obese horses and hold them off pasture until their blood glucose and insulin concentrations return to normal.

6. Medical treatment of obesity may become necessary if the horse does not lose weight on the recommended diet and exercise program. Levothyroxine sodium can be prescribed to induce weight loss and improve insulin sensitivity. Unfortunately there are no drugs that deliver a quick fix for metabolic syndrome. The drugs pergolide and cyproheptidine, which act at the pituitary gland, are not appropriate for metabolic syndrome.

7. Give a balanced daily vitamin and mineral supplement to horses that are on hay-only diets. Vitamin E should be added to provide 1000 IU/day; this replaces the vitamin E found in pasture grass and may counteract oxidative damage, which is involved in the development of pituitary pars intermedia dysfunction.

Little scientific information is available to guide practitioners using supplements available for managing insulin resistance. Magnesium and chromium (studied in humans) can be beneficial. An association exists between whole-body magnesium deficits and

insulin resistance in humans. Horses should receive 4-8 grams per day magnesium or 1 tablespoon magnesium oxide added to feed daily. Chromium is a cofactor for the tyrosine-kinase insulin-receptor pathway; supplement 2.5-5 mg/day of this mineral.

Appendix 1: In the marketplace

As soon as researchers discovered the link between sugar, starch and metabolic disorders, feed companies responded with appropriate products. All aim to provide energy in safe forms, relying on alternative sources of calories, rather than the traditional grains and molasses. Most are not complete feeds and are intended to be fed along with quality hay. In addition, the levels of NSC and other nutrients listed on the product labels are based on the manufacturers recommended feeding rates – if you give your horse more or less, his actual intake of nutrients will be affected accordingly.

Safechoice, made by Nutrena; Cargill Inc, Minneapolis, MN (www.nutrenaworld.com): soybean hulls, wheat middlings, alfalfa, high fat rice bran, soybean oil

-NSC 21%

-Formulated for horses in all life stages who require energy without the risk of starch overload

Low Starch, made by Triple Crown; Triple Crown Nutrition, Inc, Wayzata, MN (www.triplecrownfeed.com) soybean hulls, shredded beet pulp, wheat middlings, rice bran

-NSC 15%

-Formulated for horses with Cushings/PPID, insulin resistance, laminitis

Safe'n Easy Pellets, made by Buckeye; Buckeye Nutrition, Dalton, OH (www.buckeyenutrition.com) soybean hulls, wheat middlings, alfalfa meal, beet pulp

-NSC 11.9%

-Formulated for special needs and mature horses

WellSolve L/S, made by Purina; Purina Mills LLS, St Louis, MS (www.wellsolveequine.com) beet pulp, soy hulls, alfalfa meal

-NSC 11%

-Formulated for horses with insulin resistance, laminitis, Cushings syndrome or other special needs

Senior Glo or Gro Strong Ultra-fiber, made by ADM Alliance Nutrition; ADM Alliance Nutrition, Quince IL (www.admani.com/AllianceEquine) soybean hulls, wheat middlings and stabilized rice; -NSC 12%

-Formulated for working horses over age 4, broodmares, geriatric horses who need energy

Wellness Perform Safe, made by Seminole (www.seminolefeed.com) soybean hulls, beet pulp, wheat middlings, alfalfa meal

-NSC 9%

-Formulated for active horses and ponies

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