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Middle-aged obesity (body condition score 7-9 on the Henneke 1 to 9 scale) accompanied by insidious-onset laminitis is a syndrome that has been recognized by equine practitioners for decades. Equine metabolic syndrome (EMS) is a recently coined name that has gained acceptance to describe this condition. Clinical signs of laminitis commonly develop while horses are grazing spring pasture but can also occur at other times of the year and in horses without pasture access. Affected horses tend to be aged between 10-to-20 years but there does not appear to be a sex predilection. Occasionally, the syndrome can occur in younger animals that have been overfed. Pony breeds, domesticated Spanish mustangs, Peruvian Pasos, Paso Finos, Andalusians, European Warmbloods, American Saddlebreds, and Morgan horses are more commonly affected than Thoroughbreds, Standardbreds, and Quarter Horses. This breed disparity is supportive of a genetic predisposition. In the past, this syndrome was commonly attributed to hypothyroidism or pituitary pars intermedia dysfunction (PPID or classic equine Cushing’s disease); however, most affected horses do not manifest additional clinical signs or endocrinologic test results to support these conditions. It is now recognized that insulin resistance is the primary endocrinopathy induced by obesity in EMS-affected horses. However, a number of additional metabolic and endocrinologic alterations can occur in affected equids making the pathophysiology of EMS an increasingly complex subject. Finally, a subgroup of EMS-affected equids may only have abnormal fatty deposits (e.g., a cresty neck or fat deposits behind the shoulders, over the tail head, and in the sheath of male horses) without generalized obesity and these patients are often more challenging to manage than those with generalized obesity.

Prevalence of laminitis and obesity in horses:
Laminitis is a devastating clinical problem for horses and their owners. In fact, data collected in the 2000 USDA-NAHMS study revealed that laminitis was reported on 13% of horse operations. Further, the leading cause of laminitis was reported to be grazing lush pasture (Figure 1). Similarly, in the United Kingdom more than 8,000 cases of laminitis are estimated to occur annually, representing 7% of the equine population, and more than 60% of the most recent laminitis cases by perceived cause.

![Figure 1. Causes of laminitis reported in the 2000 USDA NAHMS study; note that grazing lush pasture was the most common reported cause with a peak incidence in May.](image-url)
cases were classified as pasture-associated disease. In both reports, pasture-associated laminitis had a peak incidence in May, followed by October and November. Also, as equids have transitioned from working animals to recreational companions, the physical condition of many equids has paralleled that of their human counterparts: they have been overfed and become more sedentary. As a consequence, obesity is becoming a significant problem in the equine species. In a recently studied cohort of 300 horses aged 4-20 years in Virginia, 19% were classified as obese, defined as a body condition score (BCS) of 7.5 or greater on a scale of 1 through 9.

Pathophysiology: The cause of obesity is fairly straightforward: caloric intake exceeding daily caloric requirement. Clearly, there is also a genetic predisposition towards development of obesity that has been referred to as having “thrifty genes”. Unfortunately, obesity has a number of metabolic consequences including insulin resistance (IR), hyperglycemia, altered tissue-level cortisol activity, increased leptin concentrations, altered lipid metabolism with hypertriglyceridemia, increased expression of inflammatory cytokines, and hypertension. In horses, the most obvious clinical sign that results from these metabolic alterations is laminitis. EMS appears to have some parallels to the human metabolic syndrome - a syndrome of IR and visceral adiposity (deposition of omental fat) that is recognized to affect an increasing number of middle-aged people. In affected humans, cardiovascular disease, hypertension, dyslipidemias, and type II diabetes (insulin resistant diabetes) are the common sequela. In horses, EMS is usually not recognized (or perhaps acknowledged) until insidious-onset laminitis develops in overweight horses that have no obvious risk factors for laminitis (e.g., grain overload, colic or diarrhea with endotoxemia, pleuropneumonia, or retained placenta). Hindsight often reveals that affected horses have had a decrease in exercise program (e.g., the primary rider goes away to college) while nutritional restriction was not implemented. Many horses are initially tested for hypothyroidism and serum thyroxine (T₄) and tri-iodothyronine (T₃) concentrations may be near or below the lower end of the reference range in affected equids. However, when the hypothalamic-pituitary-thyroid axis is tested dynamically by administration of thyrotropin-releasing hormone, thyroid gland function has consistently been normal. Similarly, horses older than 15 years of age are commonly suspected of having PPID but overnight dexamethasone suppression test results are normal and hirsutism is not a typical clinical sign. Elevated fasting insulin concentrations (>42 uU/ml or >300 pmol/L) may be found in some, but not all, EMS-affected equids. As an example, in the same cohort of Virginia horses described above, 10% were found to have fasting hyperinsulinemia. Although obese horses were more likely to have hyperinsulinemia than those with a BCS < 6, not all obese horses had an elevated insulin concentration. However, when dynamic testing of tissue sensitivity to insulin is pursued (several methods are available), IR is found to be a consistent feature of this syndrome. What is insulin resistance? Simply stated, IR is the metabolic state in which a greater amount of insulin is required to exert the expected physiological effect of glucose uptake by peripheral tissues. IR is most easily documented by detection of hyperinsulinemia after an overnight fast (preferably after 12 hours with no feed or, at the least, 12 hours after the last grain meal). Unfortunately, because insulin release is a dynamic physiologic response to feeding, fasting serum insulin concentration is not always elevated in horses with EMS. However, increases in serum insulin concentrations in response to administration of a bolus of glucose are typically exaggerated in horses with insulin resistance (Figure 2). A relatively simple test for IR is an intravenous glucose tolerance test (IVGTT). In the IVGTT, blood glucose concentration is measured before and for 3-6 hours after administration of an intravenous bolus of glucose (0.1 g/kg). In the normal state, blood glucose and insulin concentrations should return to baseline within 1 to 2 hours. Glucose intolerance (another name for IR) is present when insulin concentrations re-
main elevated for more than 3 hours after glucose administration. Recently, an even more simple oral sugar challenge test was validated in normal horses and horses with documented IR. The test consisted of administering an oral dose of Karo syrup (15 ml/100 kg body weight = 50 mg/kg sugar) and measuring serum insulin concentration every 30 minutes for 2 hours. A greater insulin response was found in horses with IR and it was suggested that the test could be further simplified by collecting a single blood sample 60 or 90 minutes after administration of Karo syrup.

It is also recognized that both endogenous and exogenous corticosteroids can alter tissue insulin sensitivity and uptake or release of glucose and fatty acids by hepatic, adipose, and muscle tissue. Syndromes of cortisol excess, such as PPID, can also lead to IR in some but not all affected equids and, as with EMS, IR can sometimes be manifested by hyperinsulinemia. With EMS, serum cortisol concentration is not elevated (and is often nearer the lower end of the reference range) and results of overnight dexamethasone suppression tests are normal. However, cortisol activity at the tissue level is

Figure 2. Andalusian mare with mild laminitis attributable to EMS at initial evaluation (2004, left picture) with a BCS of 8/9 and weight of 1250 lbs and at reevaluation 2 years later after a 160 pound weight loss (2006, right picture) to a BCS of 6/9; a glucose tolerance test (0.3 g/kg IV) was performed at both times and revealed greater increases in both glucose (left graph) and insulin (right graph) in 2004 (lines with squares) as compared to 2006 (lines with circles); however, it is important to note that fasting insulin concentrations were not remarkably different at these two evaluation periods emphasizing the limitation of using fasting insulin concentration alone for documentation of insulin resistance.
largely regulated by the enzyme 1-1-beta-hydroxysteroid dehydrogenase type 1 (11-β-HSD1). This enzyme has both oxo-reductase and dehydrogenase activities. Dehydrogenase activity of this enzyme converts active cortisol to inactive cortisone. In contrast, oxo-reductase activity of this enzyme results in greater conversion of inactive cortisone to active cortisol in the tissues, thereby magnifying the effects of circulating glucocorticoids on target tissues. As has been recently found in people, some horses are likely genetically predisposed to developing EMS due to tissue-specific dysregulation of 11-β-HSD1. Because the alteration in cortisol metabolism occurs at the tissue level, rather than through the hypothalamic-pituitary-adrenal axis, the terms “Cushing’s disease of the omentum” and “peripheral Cushing’s syndrome” were early descriptors used for the metabolic syndrome.

A number of further metabolic alterations can accompany both human and equine metabolic syndrome. However, obesity is the primary problem that sets off the cascade of metabolic abnormalities. In humans, excess fat can either be deposited centrally (android or apple-shaped individuals) or around the hips (gynoid or pear-shaped individuals). The former are typically insulin resistant and may have hyperinsulinemia while the latter typically are not insulin resistant. One mechanism that appears to contribute to IR in android individuals is excess fat storage in liver and skeletal muscle, perhaps because adipocytes in other areas of the body are less effective in taking up and storing fat. Excess fat in liver and muscle tissue has been demonstrated to inhibit glucose uptake by these tissues. Decreased glucose uptake subsequently leads to increased insulin release and hyperinsulinemia. With either advancing age or inheritance of “thrifty genes”, mitochondrial density and function in skeletal muscle is also reduced. Lower mitochondrial oxidation of fat can also lead to excess fat in skeletal muscle. Interestingly, it is becoming clear that adipocytes are not simply repositories of stored energy. Adipocytes also produce hormones (adipokines) that may exert actions throughout the body. One of these hormones is leptin and recent research has found an association between obesity and elevated serum leptin concentrations in horses. Another of these hormones is resistin – it is named for the fact that it plays a substantial role in IR. It remains to be seen what roles these hormones play in development of EMS. Of interest, obese horses can either have low (1.5 ng/ml) or high (10-50 ng/ml) resting plasma leptin concentrations. Horses with high leptin concentrations also had a greater insulin response to glucose infusion (during an IVGTT). Despite the fact that obesity is an important risk factor for human metabolic syndrome, it has also been recognized that not all obese people develop the clinical problems clustered under the metabolic syndrome. This again reflects the importance of genetic predisposition. Perhaps a high vs. a low resting leptin concentration in horses is another risk factor, in combination with obesity, for development of laminitis in EMS. Adipocytes also produce a family of adiponectins, peptide hormones that actually enhance insulin action. It is currently speculated that higher circulating concentrations of these peptides may be a protective factor against development of the complications of obesity. Clearly, the pathophysiology of IR with obesity is complex and incompletely understood at this time.

Finally, insulin has a broader range of actions than simply enhancing tissue glucose uptake. Specifically, insulin can act as a pro-inflammatory agent stimulating inflammatory mediator production (e.g., tumor necrosis factor-α, interleukin-6, and others) and alterations in hemodynamics (vasoconstriction and hypertension). Recent studies in both ponies and young Standardbred horses demonstrated that laminitis can be experimentally induced within 48 hours of starting an intravenous infusion of insulin (that produced supraphysiologic blood insulin concentrations). Further, endothelial cells are particularly susceptible to the effects of excess insulin and glucose. Specifically, there is a reduction in endothelial-derived nitric oxide (NO) activity and increased expression of endothelin-1 (ET-1). The combination of reduced NO and enhanced ET-1 production leads to an increased
state of vasospasticity because NO and ET -1 represent the two most potent endothelium-derived vasorelaxing and vasocontracting factors, respectively. The role of these hemodynamic changes on the development of laminitis in obese horses is currently unknown but it is becoming increasingly evident that the traditional “vascular hypothesis” and more recently described “metabolic hypothesis” for development of laminitis are not mutually exclusive.

**Diagnosis:** So how does knowledge of this complex and incompletely understood pathophysiology help an equine practitioner evaluating the overweight, foundered horse? Unfortunately, we are perhaps not a lot further ahead than we were a few years ago because no drugs have been developed to effectively treat EMS. Further, we are not a whole lot better at treating or predicting the outcome of horses with acute laminitis. However, knowledge is useful as it allows principles to be followed in the evaluation of horses with EMS and, more importantly, it fosters increased emphasis on prevention of laminitis in at risk horses. At present, diagnosis of EMS is based on physical characteristics, specifically obesity and/or regional fat deposits with or without laminitis. Further support for EMS can be demonstrated by measurement of hyperinsulinemia and mildly elevated plasma triglyceride concentrations. Of interest, some clinicians have recently suggested that the upper limit of the reference range for insulin in healthy horses should be reduced to ≈30 uU/ml or 200 pmol/L. Detection of fasting hyperinsulinemia in an overweight horse or a horse with abnormal fat deposits is supportive of EMS. It is important to remember that medications can sometimes alter blood glucose and insulin concentrations (i.e., α-2-agonist such as xylazine or detomidine). Thus, blood samples should be collected before sedation that may be needed for farrier work or other diagnostic procedures. Unfortunately, EMS-affected horses do not consistently manifest hyperinsulinemia. In those cases in which further evidence to support EMS is needed (e.g., for clients that refuse to believe that their overweight horse may be at risk for laminitis), an IV or oral GTT can be pursued.

Development of a “metabolic profile” that would include physical measurements (e.g., body weight, body condition score, neck circumference to assess “crestiness”, and others) and laboratory values (e.g., insulin, glucose, triglycerides, leptin, adiponectin, and others) that would allow risk assessment for development of laminitis in overweight horses is a goal of several research groups. Unfortunately, such a profile may take years to develop due to our limited understanding of EMS pathophysiology. Ideally, our goal should be to identify at risk horses prior to onset of laminitis in order to implement diet changes and an exercise program. Thus, this author strongly advocates assessment of obesity by performing a body condition score during annual or semiannual preventive health care visits. When BCS is 6 or greater, diet should be evaluated (concentrate feeds discontinued and pasture access limited or a grazing muzzle used) and an exercise program should be initiated with owners. In addition, hoof conformation should be regularly assessed with an emphasis on looking for early changes indicative of chronic laminitis. If changes are noted, lateral foot radiographs should also be pursued. Lastly, a good veterinarian-client relationship is critical for diet and exercise recommendations to be practically implemented by owners.

**Management:** Management of EMS can be challenging as it primarily involves client education and acceptance to comply with dietary recommendations to effect substantial weight loss. In addition, an understanding of the differences in nonstructural carbohydrate content of various forages is important for appropriate dietary recommendations to be made. Next, implementation of an exercise program for both at risk and affected horses (those with laminitis) is strongly recommended for overweight ponies and horses but may be difficult to implement in equids suffering from laminitis. Although medications (thyroid hormone and metformin) and dietary supplements (magnesium, chromium, vanadium, cinnamon, etc.)
have been advocated to both assist with laminitis recovery and enhance weight loss, data supporting use of these agents is limited.

**Endocrinopathic laminitis:** Unfortunately for horses, syndromes of IR and cortisol excess (with both EMS [at the tissue level] and PPID [systemic cortisol excess]) appear to be accompanied by alterations in the integrity of the basement membrane between the epidermis and dermis of the laminar bed. Over time, weakening and degradation of the basement membrane can lead to separation of the epidermal-dermal junction and development of laminitis. The most recently advanced term for this type of laminitis is endocrinopathic laminitis. The mechanisms behind development of laminitis appear to be complex and remain incompletely understood. Nevertheless, research over the past decade has provided new insights into some of these mechanisms and may lay the groundwork for novel approaches to treatment of this devastating problem in horses.

**Anatomy and physiology of the equine foot:** The equine hoof is a complex epidermal-dermal structure that has evolved to support the large body mass of the horse. Although not typically thought of as skin, the hoof is actually comprised of the same basic epidermal-dermal layers as skin. However, the area of epidermal-dermal attachment has changed from a nearly straight junction to an undulating or interdigitating junction of primary and secondary lamellae. This “laminar bed” markedly increases the surface area for attachment of the epidermis (hoof capsule) to the underlying dermis, thereby increasing the strength of attachment and capacity to support weight. The primary lamellae (600-800 within each hoof) are long finger-like projections and interdigitation of the epidermal lamellae and the dermal lamellae holds the hoof capsule onto the underlying dermis. However, the real strength of attachment is provided by the secondary lamellae that consist of numerous short projections of each primary lamella. Secondary lamellae can be thought of as velcro-like projections that provide incredible strength of attachment to the primary lamellae. At the junction of the epidermis and dermis lies the basement membrane. The basement membrane consists of a lamina lucida, a lamina densa, and extracellular matrix. Within these layers are several proteins including laminin, type IV collagen, type VII collagen, integrins, anchoring filaments, and others. In addition to forming the supporting extracellular matrix of the basement membrane, these proteins, along with others, also anchor or attach the secondary epidermal lamellae to the basal cells of the secondary dermal lamellae.

**Mechanisms involved in basement membrane damage in laminitis:** Epidermal tissues have somewhat different metabolic requirements and machinery than many other organs. Specifically, the epidermis has an absolute requirement for glucose as an energy substrate. Pollitt and coworkers have nicely demonstrated this glucose requirement using an *in vitro* hoof explant model system. When cubes of hoof material were incubated in various media, integrity of the basement membrane was lost after 48 hours of incubation in media without glucose. In contrast, integrity of the basement membrane was maintained when glucose was present in the media. Another piece of evidence, albeit indirect, is the efflux of lactate from epidermal and hoof tissue. This has been demonstrated by the finding of higher lactate concentrations in digital venous plasma than jugular venous plasma. With an acute insult to the laminar tissue, as in spontaneous diseases or with the carbohydrate overload model for induction of laminitis, another mechanism for damage to the laminar bed is induction of matrix metalloprotease (MMP) activity. Specifically, increased amounts of the active forms of the basement membrane degrading enzymes, Eq-MMP-2 and Eq-MMP-9, have been found in laminar tissues affected by laminitis 48 hours after carbohydrate overload. Of interest, the damage to the basement membrane caused by glucose deprivation and activation of MMPs differs. With glucose deprivation, the anchoring filaments detach from the basal cells of the secondary dermal lamellae. In contrast, with
activation of MMPs, the anchoring filaments and other proteins of the extracellular matrix are destroyed.

**Role of cortisol in development of laminitis:** Although development of laminitis with use of exogenous glucocorticoids is clinically recognized in horses, the mechanism(s) for this adverse effect of glucocorticoids has not been well established. One possible explanation is alteration in glucose uptake by tissues due to decreased tissue sensitivity to insulin under the influence of glucocorticoids. If glucocorticoid action leads to decreased glucose uptake and utilization, and glucose is absolutely required for maintenance of the integrity of the lamellar basement membrane, then glucocorticoids could lead to slow, insidious degradation of the basement membrane and eventual separation of the lamina.

Regulation of cortisol activity at the tissue level is largely mediated by the enzyme 11-beta-hydroxysteroid dehydrogenase type 1 (11-β-HSD1). In the horse’s foot elevated 11-β-HSD1 o xo-reductase activity may enhance the action of cortisol on the metabolism of extracellular matrix of lamellar connective tissue and the regular turnover of anchoring filaments connecting basal cells to the lamellar basement membrane. In short, local cortisol activity may downregulate the natural turnover of the anchoring filaments in the basement membrane. In contrast to more acute insults such as grain overload in which activation of MMPs leads to rapid degradation of the basement membrane, the process with the cortisol excess is more insidious in onset. As a consequence, endocrinopathic laminitis is often subclinical and chronic before overt lameness becomes apparent.

Recently, investigators at the University of Missouri demonstrated increased 11-β-HSD1 o xo-reductase activity in both skin and laminar tissue collected from horses with both acute (carbohydrate overload model) and naturally occurring chronic laminitis. This novel finding is an attractive explanation for the long recognized syndromes of obesity-associated laminitis in horses as well as laminitis associated with PPID. However, it is also important to recognize that the pathogenesis of laminitis in both of these syndromes of endogenous glucocorticoid excess remains incompletely understood.

**Carbohydrate content of forage:** It is well recognized that “sweet feeds” and other concentrates high in soluble carbohydrate (with a high glycemic index) are best avoided in obese, IR, laminitic horses. Despite proliferation of “low starch” pelleted feeds, it is also important to recognize and to recommend to clients that overweight horses do not need any concentrate feeds at all. In fact, all essential nutrients and energy can often be found in good quality forage, although many practitioners and nutritionists continue to recommend addition of a vitamin and mineral supplement to a forage only diet. Such supplementation may not always be necessary but would certainly be prudent in areas where specific nutrients (i.e., selenium) may be low.

Lush grass pasture and some hays can also be a rich source of soluble carbohydrate. In fact, grazing lush spring pasture can be similar in dietary intake of soluble carbohydrate to feeding a high concentrate ration. Soluble carbohydrates are those that can be absorbed in the small intestine and lead to increases in glucose and insulin concentrations. In contrast, insoluble carbohydrates are contained in plant cell walls and require bacterial fermentation in the cecum and large colon for digestion. The end products of fermentation are volatile fatty acids that are absorbed in the large intestine and provide more than 50% of an equids daily energy needs.

Studies in which soluble carbohydrate content of pasture grass has been measured at various times of the year have shown a spring rise, peaking in April through June in the northern hemisphere, depending on latitude. In addition, there is a diurnal pattern to pasture soluble carbohydrate content. Specifically, during daylight hours fructan content (fructan is one of the soluble carbohydrates in forage) increases. During the night, plant cells utilize fructans as an energy source in the absence of photosynthesis much as liver glycogen is utilized in mammals in the hours between meal ingestion.
Not surprisingly, serum insulin concentrations in equids grazing pasture at these various times of the year have shown substantially greater increases when pasture soluble carbohydrate content is high (spring and to a lesser extent in fall). Further, in ponies with historical pasture-associated laminitis, a more dramatic increase in insulin concentration is observed after grazing, in comparison to ponies on the same that have never had laminitis (supporting a genetic predisposition even within similar phenotypes or breeds).

Whether or not it is “safe” to allow overweight equids to graze pasture as a forage source remains controversial. Pasture, especially lush spring and early summer pasture, should be considered similar to feeding concentrates high in soluble carbohydrates and should be avoided. Further, if pasture is to be utilized it may be preferable to turn equids out in the early morning hours while plant fructan content is relatively low. On the flip side, being turned out to pasture has the benefit of some exercise, as compared to being in a stall or small dirt lot. Grazing muzzles can be a good compromise for turning overweight horses out on pasture.

Hay is usually cut once plants have neared maturity; thus, hays typically have less soluble carbohydrate content than pasture and grass hays have less than legume hays (alfalfa and clover). However, various batches of hay cut from nearby fields may appear similar but can have variation in soluble carbohydrate content depending on maturity, time of day that hay was cut from nearby fields may appear similar but can have variation in soluble carbohydrate content depending on maturity, time of day that hay was cut (often in the late afternoon to allow the dew to dry off – when fructan content may be highest), or whether or not there may have been frost damage (fructan is not utilized as rapidly overnight when temperatures are cool). Unfortunately, the only true way to assess forage soluble carbohydrate content is to have the forage analyzed. This is not really important for horses that are in good weight and in a regular exercise program but it is warranted when trying to identify a hay source for an overweight, IR, and possibly laminitic horse. In the absence of forage analysis, hay can also be soaked for 30-60 minutes prior to feeding in an effort to leech out some of the soluble carbohydrate.

**Dietary management of overweight horses:**

It is clearly difficult to have owners comply with dietary recommendations that eliminate all concentrate feeds and pasture turn out for their overweight horses. Thus, I recommend that a horse owner approach the problem of an obese horse in much the same way that they may have to approach a family member with a substance abuse problem. An intervention is needed and for the horse the drug is too much feed. If an owner accepts this approach and follows strict dietary recommendations for 60-90 days, improvement (specifically weight loss) can usually be seen providing positive reinforcement for further owner compliance. It is commonly assumed that an adult horse requires 2% of its body mass in daily forage intake for maintenance. However, horses with EMS are “easy keepers”, likely due to presence of thrifty genes and they may need only 1.5-1.7% of their body mass in daily forage.

In the 6th revision of the Nutrient Requirements of Horses (2007) prepared by the Committee on Nutrient Requirements for Horses of the National Research Council Board on Agriculture and Natural Resources, the average recommended daily energy intake for a sedentary adult horse (1000 pounds) was 16.7 Mcal/day with a minimum value of 15.2 Mcal/day. The latter amount digestible energy can easily be provided by 15 pounds of good quality hay daily. To put energy intake into perspective, if an owner feels they must add a pound of concentrate feed twice daily to the ration, this would increase daily energy intake by 3 Mcal/day (to about 18 Mcal/day). After a year, an additional 1000 Mcal would have been fed and would have produced a 100 pound increase in body mass!

Thus, in order to produce weight loss, even less than 15 pounds of hay needs to be fed daily. However, this feed restriction would clearly result in an unhappy horse that may start chewing on wood or other parts of its surroundings. A compromise can be achieved by finding a lower quality of hay (generally an over mature hay) that could be fed in a similar amount. If an adequate hay source cannot be identified, soaking the hay would be another option to decrease energy content. The diet is started at 1.5% of the...
horse’s body mass but it is important to realize that current body weight is greater than ideal body weight. As a consequence, forage intake is slowly decreased over 4 weeks to 1.5% of the target body weight. The only way that such a diet can be appropriately fed is to weigh the daily hay ration. This can easily be accomplished by placing the daily ration in a hay net or plastic bag and holding it while the owner steps on their bathroom scale (that they have brought to the barn). A weight loss of approximately 50 pounds is required to produce a numerical reduction in body condition score. This can be loosely equated to an energy deficit of about 350 Mcal for the average adult horse. The goal should be to accomplish this 50 pound weight loss within 30-60 days after the diet has been started. If that target is not reached, a further reduction (10-15%) in hay intake will be required or hay soaking will need to be started if not yet implemented. Once ideal body weight has been achieved, it is clearly important to continue to limit feed intake and provide minimal pasture access in order to maintain ideal weight.

**Further management of overweight horses:**

Another important management tool is implementation of an exercise program. A caloric deficit can be accomplished both by limiting feed intake as well as by increasing caloric expenditure through exercise. It is well recognized that overweight human patients accomplish and maintain weight loss more effectively if diet is combined with an exercise program and the same is likely true for horses. Unfortunately, the major complication of EMS is chronic laminitis and a painful gait may limit the ability to exercise. However, as soon as the horse is deemed comfortable enough to walk, regular hand walking for 20-30 minutes three to five times a week can be useful. In addition to burning calories, exercise also improves tissue sensitivity to insulin and may further limit ongoing lamellar damage.

Appropriate hoof care is also essential in horses with obesity-associated laminitis. Dietary restriction and exercise may lead to desired weight loss but substantial lameness may persist if the angle of the distal phalanx within the hoof capsule remains uncorrected by proper trimming and possibly shoeing. Judicious use of non-steroidal anti-inflammatory drugs is also often required to alleviate the pain of chronic laminitis.

**Medications and supplements from managing EMS:** Although there is a great desire for pharmacological intervention in both human and equine metabolic syndromes, there will likely never be a “magic pill” for either weight loss or to markedly improve tissue insulin sensitivity. Supplementation with thyroid hormone has been demonstrated to produce further weight loss, as compared to diet alone, when overweight horses were administered twice the daily recommended dose for a year. Next, a recent study by Durham and coworkers reported potential benefits of metformin (15 mg/kg, PO, q 12 hours) in 18 insulin resistant horses and ponies with laminitis. Results of insulin sensitivity testing showed improvement within 1-2 weeks of starting the medication but the improvement did not persist with long-term treatment and four patients continued to suffer recurrent bouts of laminitis. Unfortunately, recent pharmacologic studies in normal and IR equids have shown low bioavailability of metformin (<10% of an oral dose when fasted and <5% of an oral dose when fed). Thiazolidinedione drugs also increase insulin sensitivity in humans and cats, but their use in EMS is only speculative at present. It has been suggested that anti-oxidants might also be beneficial. Vitamin E can safely be administered to horses at high levels (10,000 units, PO/day) but supportive data for improvement in EMS are lacking. Currently, data that show therapeutic value for chromium, magnesium, vanadium, or cinnamon supplementation for insulin insensitive horses are also lacking.

At present, prevention of obesity, especially in those breeds at greater risk for EMS, is the best advice that is available and equine veterinarians should strongly consider assessing body weight and fat stores (by using a weight tape or other measures to estimate weight and assigning a body condition score) as part of their preventive care practices.
REFERENCES

Equine and human metabolic syndrome


Endocrinopathic laminitis


