Feeding Performance Horses with Myopathies

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1. Introduction

In combination with exercise, nutrition is an essential component of managing horses with myopathies. The optimal feeding program for an individual is tailored to the diagnosis of a specific underlying myopathy.

2. Classification of Exertional Myopathies

Exertional myopathies are defined by muscle pain and impaired performance during or after exercise. Exertional rhabdomyolysis (ER) represents a subset of exertional myopathies characterized by elevations in serum creatine kinase (CK) and aspartate transaminase (AST) activities. Forms of ER include type 1 polysaccharide storage myopathy (PSSM1), type 2 polysaccharide storage myopathy (PSSM2) in Quarter Horses, malignant hyperthermia, recurrent exertional rhabdomyolysis (RER), and myofibrillar myopathy (MFM) in Arabians.[1-5]

There are other exertional myopathies, such as PSSM2 and MFM in Warmbloods, that are not typically characterized by elevations in serum CK and AST activities.[6] Horses with these exertional myopathies have exercise intolerance and show reluctance to go forward, collect, and engage the hindquarters. Because these clinical signs are not specific to muscle disease, causes of decreased performance such as behavior, rider, tack, and orthopedic lameness need to be ruled out prior to investigating a primary exertional myopathy.

Exertional Rhabdomyolysis

Overt ER can arise as a sporadic event due to extrinsic factors such as exercise in excess of training, nutritional imbalances, or exercise during viral illness. ER can also occur as a chronic disease due to intrinsic abnormalities in muscle function. Acute clinical signs of ER are similar across the spectrum of etiologies and include muscle stiffness, shortened hindlimb stride, reluctance to move, and firm, painful hindquarter muscles. Anxiety, pain, sweating, and increased respiratory rate are common signs.

Sporadic ER

Sporadic forms of ER develop from exercise in excess of training, dietary imbalances (including high nonstructural carbohydrate (NSC) content and low forage content), and deficiencies in electrolytes.[7,8] ER may be exacerbated by inadequate dietary selenium and vitamin E.

NOTES

Chronic ER

Chronic forms of ER appear to develop in horses due to intrinsic abnormalities in muscle function. In some cases, muscle dysfunction is attributed to a single gene defect.[9,10] In other cases, there may be multiple genes impacting muscle dysfunction or post-translational modifications of gene products that arise under certain environmental stimuli.

Recurrent Exertional Rhabdomyolysis

Recurrent exertional rhabdomyolysis describes a subset of ER that is believed to be due to an abnormality in the regulation of muscle contraction and relaxation.[11–13] Research into RER has primarily been performed in Thoroughbreds and to a lesser extent in Standardbreds.[11,14–16] There are reports of ER in racing Quarter Horses, Arabians, and Warmbloods that may have the same underlying cause based on the overlapping histories, clinical signs, muscle biopsy findings, and response to management.[17] Mares more commonly have RER than males, although no general correlation has been observed between episodes of ER and stages of the estrous cycle.[18] Nervous horses, particularly nervous fillies, have a higher incidence of ER than calm horses.[14,19,20] Diet also has an impact with Thoroughbreds fed more than 2.5 kg of grain being more likely to show signs.[21] Research suggests that horses with RER may have an inherent abnormality in intramuscular calcium regulation that is intermittently manifested during exercise in stressful environments.[13,22]

Polysaccharide Storage Myopathy

Several acronyms have been used for polysaccharide storage myopathy (PSSM), including EPSM and EPSSM.[23–25] Muscle biopsies from PSSM-afflicted horses are characterized by the presence of abnormal polysaccharide inclusions, which are typically amylase resistant in PSSM1 and amylase sensitive in PSSM2.

PSSM1

PSSM1 is caused by an autosomal-dominant gain-of-function mutation in *GYS1* that results in elevated glycogen synthase activity and >1.5-fold higher muscle glycogen concentrations in skeletal muscle.[10] The enzyme mutation enhances synthesis of glycogen and appears to disrupt metabolism of this energy substrate. The severity of clinical signs of PSSM1 can vary widely from asymptomatic to severe incapacitation. The most common trigger for ER is less than 20 minutes of light exercise, particularly if the horse has been rested for several days prior to exercise or is unfit. Diets high in NSC also increase the risk of muscle pain and stiffness in PSSM1 horses.[26] The gold standard for diagnosis of PSSM1 is genetic testing for the *GYS1* mutation performed on whole blood or hair root samples.

PSSM2

PSSM2 is a histopathologic designation that indicates the presence of abnormal-appearing amylase-sensitive or amylase-resistant polysaccharide in muscle biopsies of horses lacking the *GYS1* mutation. Importantly, the term PSSM2 does not indicate a specific etiology since no common genetic mutations or biochemical aberrations have been defined in these horses to date. Commercial genetic tests for PSSM2 have not been scientifically validated through peer-reviewed publication. Approximately 28% of cases of PSSM diagnosed by muscle biopsy in Quarter Horse-related breeds would be classified as PSSM2.[27] Quarter Horses with PSSM2 present with ER and have biochemical elevations in muscle glycogen concentrations. PSSM2 seems to be common in both high-performance Quarter Horse types such as barrel racing, reining, and cutting horses as well as pleasure and halter horses. About 80% of Warmblood horses diagnosed with PSSM by muscle biopsy are classified as PSSM2.[27] The clinical presentation of PSSM2 in Warmbloods is often that of exercise intolerance rather than overt ER, and biochemical elevations in muscle glycogen are uncommon.

Myofibrillar Myopathy

Myofibrillar myopathy (MFM) is a recently identified disorder presenting with exercise intolerance or intermittent ER that is defined by specific histopathology.[5,6] The hallmark histopathologic feature of MFM is cytoplasmic aggregates of the cytoskeletal protein desmin in scattered muscle fibers. Desmin functions to align sarcomeres at the Z-disc and tether them to the cell membrane. MFM may represent a more extreme subset of PSSM2 in Warmbloods and Arabians, but further research is required. Mean muscle glycogen concentrations in Warmblood and Arabian horses with MFM are similar to controls.[28,29]

MFM in Warmbloods

Warmblood horses diagnosed with MFM by muscle biopsy have an insidious onset of exercise intolerance notable by 6–8 years of age characterized by a lack of stamina, unwillingness to go forward, inability to collect, abnormal canter transitions, and inability to sustain a normal canter.[6] Unresolved hindlimb lameness, stiffness, muscle pain and, rarely, an episode of ER are reported.[29] Serum CK and AST activities are usually within normal limits unless samples are taken in conjunction with ER. A recent study found no association between commercial genetic tests for MFM and a clinical and histopathologic diagnosis of MFM in Warmblood horses.[30] The basis for MFM in Warmblood horses appears to be related to the individual effects of diet, exercise, and training on gene and protein responses to exercise with downstream effects on muscle mass, the alignment of contractile proteins, mitochondrial function, and oxidative stress (unpublished observations).

MFM in Arabians

Arabian endurance horses diagnosed with MFM usually have a history of intermittent elevations in serum CK activity after endurance rides (>10,000 U/L) or during exercise that follows a week or more of rest.[5] Horses do not always show the same degree of pain, sweating, and reluctance to move, as is frequently seen in other forms of acute ER. Myoglobinuria can be observed in horses with only mild muscle stiffness. Between episodes, the heart rate, lactate, CK, and AST responses to exercise are normal. The basis for MFM in Arabian horses appears to be related to a need for enhanced cysteine synthesis, decreased cysteine-based antioxidants, and oxidative stress.[31]

3. Management of Chronic Exertional Muscle Disorders

Altering diet and exercise regimes to compensate for underlying defects is often the best available strategy to assist horses with exertional myopathies. Identifying and eliminating any known factors that trigger ER are also important in preventing further episodes. Controlled treatment trials have been performed to validate management strategies for RER and PSSM1.[26,32,33] Less evidenced-based information is available with regard to management of PSSM2 and MFM, and recommendations are based largely on retrospective studies or clinical impressions.[17,34]

4. Feeding Programs for Horses with Myopathies

A nutritionally balanced diet with appropriate caloric intake and adequate protein, vitamins, and minerals is a core element in treating all forms of exertional myopathies. As with all classes of horses, the development of a ration for these horses includes a series of steps.

1. **Determine daily nutrient requirements.** A horse's nutrient requirements depend on age, breed, body size, growth rate, level of exercise, and other considerations. The National Research Council last published its recommendations for horses in 2007.[35] National Research Council requirements are often considered minimums for many nutrients. Recommendations that are more commonly used in practice are also available in commercially available software[a,b].
2. **Select type and intake of forage.** Forage should be the foundation of every equine feeding program, so it is important to establish both the type and expected intake of forage before choosing concentrates or supplements.
3. **Select energy sources in concentrate.** One of the keys to managing exertional myopathies is controlling the source of energy in a ration. Energy requirements in the US are expressed in terms of megacalories (Mcal) of digestible energy (DE). DE can be supplied from nonstructural carbohydrates (NSCs), fat, structural carbohydrates (fiber), and protein. NSC is the sum of water-soluble carbohydrates (WSCs) (sugars) and starch. Most concentrates fed to ER horses are low in NSC and high in fat. Unfortunately, determining the NSC content of commercial concentrates is not easy since these nutrients rarely appear as guarantees on feed tags or bags. Although the American Association of Feed Control Officials currently suggests that commercial feed products that bear labeling claims related to carbohydrate content should include max sugar (ESC) and max starch in the guaranteed analysis, there is not an agreed-upon method for measuring ESC and starch. Therefore, many state regulatory agencies do not allow these nutrients to appear with other nutrient analyses such as protein, fat, or crude fiber. Feed manufacturers often supply this information in supporting literature or on the internet, but these figures are not regulated by any governmental agency. Most feed manufacturers use Equi-Analytical in Ithaca, NY[c] to determine WSC and starch values in feeds, and horse owners and veterinarians can also send feeds and forages to this lab for analysis.
4. **Calculate intake of concentrate to meet energy requirement.** The quantity of concentrate required by a horse equals the DE requirement of the horse minus the DE supplied by forage. The DE requirement is dependent upon the activity level and the current energy status of the horse. DE requirements will vary depending on whether the horse needs to lose, gain, or maintain its body weight.
5. **Calculate intake of other nutrients (protein, minerals, vitamins) provided from forage and concentrate.** Most commercial concentrates are formulated to meet nearly all of the protein, mineral, and vitamin requirements of the horse if fed at a typical level of intake as recommended on the feed bag. Often, a horse will be fed below this expected range of intake and additional fortification will be required. This is particularly true when horses are fed high-quality forage or if they need to lose weight.
6. **Supplement-required nutrients not provided by forage and concentrate.** Supplements are often necessary to provide nutrients not found in the forage and concentrate, either because of low concentrate intakes or to supply levels of nutrients that are greater than typically added to commercial feeds. Electrolytes, amino acids, vitamin E, and other antioxidants fall into this category for horses suffering from myopathies.

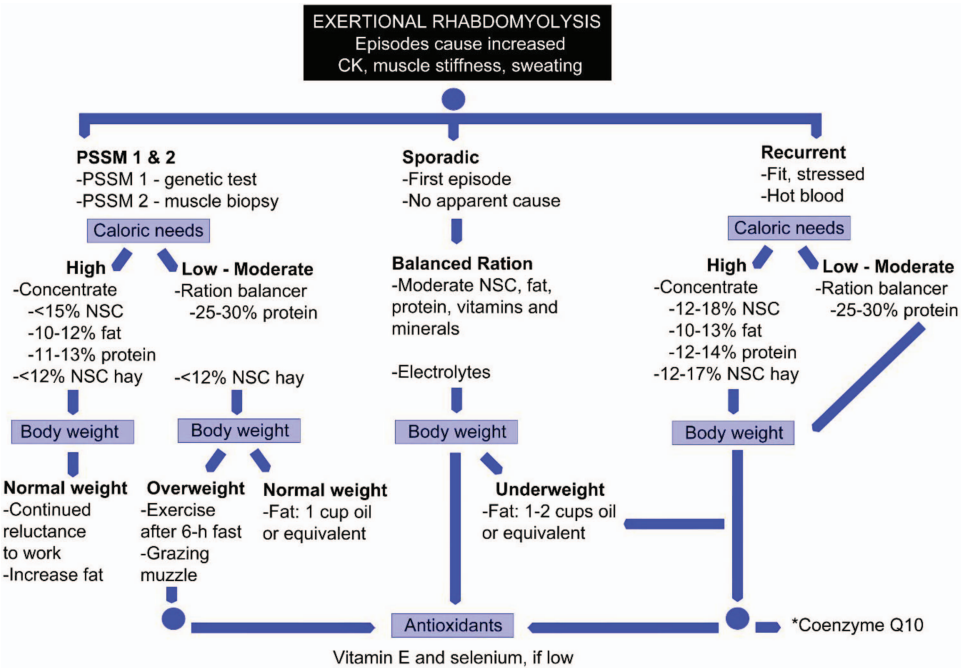

Fig. 1. An approach to managing horses with clinical signs of ER characterized by muscle stiffness, sweating, reluctance to move, and increased serum CK activity. Decisions should be based on the underlying myopathy, the horse's caloric needs, and current body weight.
*These are suggested recommendations based on current research in normal horses and anecdotal reports from the field and have not yet been tested on horses with this myopathy.

5. Exertional Rhabdomyolysis

A general approach to designing a ration for horses with primary clinical signs of ER is outlined in Fig. 1.

6. Sporadic ER

Nutrient Requirements

Total nutrient requirements will vary depending on the horse's size, breed, discipline, and level of activity. DE requirements will vary from near maintenance to twice maintenance.

Forage

Since low forage intake may contribute to sporadic bouts of ER, provide adequate quantities of high-quality forage. Performance horses will typically consume 1.5%–2% of body weight per day of hay. Good-quality grass or grass-legume mixed hays (55%–65% neutral detergent fiber [NDG], 10%–12% crude protein [CP], 10%–17% NSC) are preferable.

Energy Sources

A concentrate with moderate levels of soluble carbohydrate (20%–30% NSC), fat (4%–8%), and fiber (20%–30% NDF) is appropriate. Horses with sporadic ER do not necessarily benefit from increased dietary fat, so addition of fat should depend upon caloric needs.

Concentrate Intake

Concentrate intake will depend on the horse's DE requirement, and the quality and quantity of forage. If low concentrate (<3 kg/d) is required, supplemental protein, minerals, and vitamins may be required. This is best accomplished with appropriately fortified ration balancer pellets. Underweight horses may benefit from additional vegetable oil (120–240 mL) or stabilized rice bran (0.5–1 kg).

Supplements

Electrolyte imbalances and deficiencies are a common cause of sporadic ER. Horses should have free-choice access to a salt block and be supplemented with salt or a commercial electrolyte at levels to meet requirements. This can vary from 30 to 60 g/day with light sweating and up to 120–150 g/day with heavy sweating. Furosemide administration (5 cc) results in around 20 g of sodium and 35 g chloride loss in urine in the first 4 hours after administration.[36]

Selenium and vitamin E status should be evaluated. Low serum levels of either nutrient warrant supplementation. For horses with a neuromuscular disease, serum vitamin E levels should be checked periodically to ensure levels are > 3 μg/mL and supplemental vitamin E dosages adjusted accordingly. Large individual variation has been found in serum alpha tocopherol concentrations

when horses are supplemented with 2000–5000 IU/day of vitamin E.[37] Natural-source vitamin E is more bioavailable than synthetic sources, and either micellized[d] or nanodispersed[e] sources rapidly restore serum status.[37,38]

7. RER

Nutrient Requirements

As with sporadic ER, RER nutrient requirements will vary depending on the horse's size, breed, discipline, and level of activity. DE requirements will vary from near maintenance to twice maintenance. RER occurs most frequently in Thoroughbred and Standardbred racehorses that have DE requirements of 30–35 Mcal DE/day.

Forage

Thoroughbred horses do not appear to show the same significant increase in serum insulin concentrations in response to consuming hay with an NSC of 17% as seen in Quarter Horses.[39] This fact combined with the high caloric requirements of racehorses suggests that it is not as important to select hay with very low NSC content in RER Thoroughbreds as it is in PSSM horses. Anecdotally, some trainers find horses with RER have more frequent episodes of ER on alfalfa hay, in which case it should be avoided on an individual basis. The nervous disposition of some RER horses may predispose them to gastric ulcers, and thus frequent provision of hay with a moderate NSC and mixed alfalfa content may be indicated.

Energy Sources

Substitution of fat for NSC in an energy-dense ration significantly reduces muscle damage in exercising RER horses. A controlled trial using a specialized feed[f] developed for RER showed that NSC should provide no greater than 20% and fat should provide between 20% and 25% of daily DE intake for optimal management of RER horses requiring high DE intakes (>30 Mcal DE/day).[32] The benefit of a high-fat diet for RER does not appear to be a change in muscle metabolism. Pre- and post-exercise muscle glycogen and lactate concentrations are the same in RER horses fed a low-starch, high-fat diet compared with a high-starch diet.[32,40] Rather, low-NSC, high-fat diets in RER horses may decrease muscle damage by assuaging anxiety and excitability, which are tightly linked to developing rhabdomyolysis in susceptible horses. High-fat, low-NSC diets fed to fit RER horses produce lower glucose, insulin, and cortisol responses and led to a calmer demeanor and lower pre-exercise heart rates.[41] Neurohormonal changes may develop in response to high serum glucose, insulin, and cortisol concentrations, resulting in an anxious demeanor.

Concentrates

Racehorses in full training typically consume 6–7 kg/day of concentrate. Racehorse concentrates for RER horses should contain 12%–18% NSC and 10%–13% fat. To maintain high energy densities (3.2–3.4 Mcal DE/kg), they should also contain sources of highly digestible fiber such as beet pulp or soy hulls. The beneficial effects of low-NSC, high-fat rations appear to be more directly related to the glycemic and insulinemic nature of the feeds rather than their absolute NSC and fat content. Therefore, the ingredients used in a concentrate also affect its suitably as an RER feed. WSCs produce higher glycemic responses than starch. Molasses is extremely glycemic in horses,[42] but added fat greatly reduces glycemic response, even in high-NSC feeds.[43,44] Glycemic response is also affected by rate of intake and rate of gastric emptying.[44,45]

While a calm demeanor is desired during training, some racehorse trainers feeding low-NSC, high-fat feeds prefer to supplement with a titrated amount of grain three days prior to a race to potentially boost liver glycogen and increase a horse's energy during the race.

As with sporadic ER, concentrate intakes <3 kg/day may not provide adequate amounts of protein, minerals, or vitamins and a balancer pellet may be required.

Studies in RER horses show that significant reductions or normalization of post-exercise serum CK activity occurs within a week of commencing a low-starch, high-fat diet.[32] Days of no training and standing in a stall are discouraged because post-exercise CK activity is higher after 2 days of rest compared with values taken when performing consecutive days of the same amount of submaximal exercise.[32]

8. PSSM1

Nutrient Requirements

Meeting the horse's caloric requirements for an ideal body weight is the most important consideration in designing a ration for PSSM, as many horses with PSSM are easy keepers and may be overweight at the time of diagnosis. Adding excessive calories in the form of fat to the diet of an obese horse may produce metabolic syndrome and is contraindicated. If necessary, caloric intake should be reduced by using a grazing muzzle during turnout, feeding hay with a much lower NSC content at 1%–1.5% of body weight, providing a low-calorie ration balancer, and gradually introducing daily exercise. Rather than provide dietary fat to an overweight horse, fasting for 6 hours before exercise can be used to elevate plasma free fatty acids prior to exercise and alleviate any restrictions in energy metabolism in muscle.

Forage

Quarter Horses develop a significant increase in serum insulin concentrations in response to consuming hay with an NSC of 17%, whereas insulin concentrations are fairly stable when fed hay with 12% or 4% NSC content.[33] Because insulin stimu-

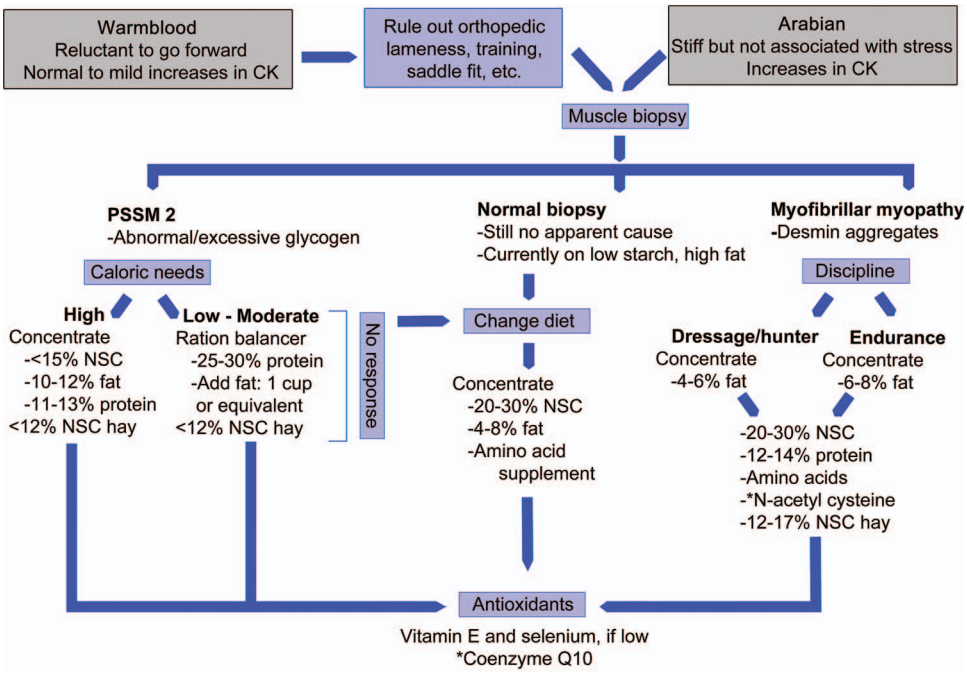

Fig. 2. An approach to managing horses with clinical signs of exertional myopathy characterized by exercise intolerance and normal to mildly increased serum CK activity. To conclude that a myopathy is responsible for exercise intolerance, other common causes should first be ruled out.
*These are suggested recommendations based on current research in normal horses and anecdotal reports from the field and have not yet been fully tested on horses with this myopathy.

lates the already overactive enzyme glycogen synthase in the muscle of PSSM1 horses, selecting hay with 12% or less NSC is advisable. The degree to which the NSC content of hay should be restricted below 12% NSC depends on the caloric requirements of the horse. Feeding a low-NSC (<5%), high-fiber (>65% NDF) hay provides room to add an adequate amount of fat to the diet of easy keepers without exceeding the daily caloric requirement and inducing excessive weight gain. For example, a 500-kg lightly exercised horse generally requires 18 Mcal DE/day. A mixed-grass hay (12% NSC, 55% NDF, 2.0 Mcal DE/kg) fed at 9 kg/day meets the horse's daily caloric requirement. In contrast, 8 kg of a 4% NSC hay (1.7 Mcal DE/kg) would provide 13.6 Mcal DE/day, which would allow a reasonable addition of 4.4 Mcal DE from fat per day (530 mL of vegetable oil).

Energy Sources

A high-NSC diet increases the propensity to develop muscle pain with aerobic exercise in PSSM1 horses.[26] A high-NSC diet results in enhanced glycogen synthase activity, which may impair oxidative metabolism of substrates such as pyruvate and fatty acids. PSSM horses on high-NSC diets have low plasma non-esterified free fatty acid concentrations, possibly due to suppression of lipolysis by high insulin.[26] Low dietary starch and fat supplementation facilitate muscle fat metabolism in PSSM1 horses.

Concentrates

Concentrates for PSSM1 horses should be low in NSC (<15%) and low glycemic. High fat (10%–12%) can be included in the concentrate but, if daily intake is low (<2 kg/d), then additional fat supplementation may be required from added vegetable oil (120–240 mL) or stabilized rice bran. Hydrated, rinsed beet pulp produces a very low glycemic response and can be used as a carrier for added vegetable oil.[42] One kilogram of beet pulp (prehydrated weight) and 1 cup (240 mL) of vegetable oil and 500 g of a balancer pellet (to meet protein, mineral, and vitamin requirements) would provide around 6.0 Mcal DE, which is equivalent to the DE supplied by 2 kg of a typical commercial concentrate.

Exertional Myopathies

A current approach to managing horses with exertional myopathies that are characterized by exercise intolerance and normal serum CK activity is outlined in Fig. 2. This approach is based on muscle biopsy diagnosis of PSSM2 or MFM and not based on a diagnosis using commercial genetic tests for PSSM2 or MFM that have not been scientifically validated through peer-reviewed publication.[30]

9. PSSM2 and Myofibrillar Myopathy

As mentioned above, PSSM2 appears to represent a histologic description of glycogen staining in muscle biopsy rather than one specific disease.[29] With a lack of information on the cause of PSSM2, the low-NSC, high-fat PSSM1 diet has been universally recommended for all horses diagnosed with PSSM regardless of whether PSSM1 or PSSM2.[46] Recommendations for feeding PSSM2, however, have now evolved according to breed based on recent research into muscle glycogen concentrations, histologic markers, and molecular approaches that better subclassify PSSM2.

Biochemical analysis of muscle biopsies indicates that Quarter Horses with PSSM2 have muscle glycogen concentrations that are as high as PSSM1 and that lack abnormal desmin staining characteristic of MFM (unpublished observation). Thus, the PSSM1 diet remains the appropriate recommended diet for Quarter Horses with PSSM2. An unpublished survey of horse owners indicates that episodes of ER significantly decrease with this dietary approach (personal observation).

Biochemical analysis of muscle biopsies in Arabians and Warmblood horses with PSSM2 has found that glycogen concentrations are similar to those of healthy, breed-matched controls.[28,29] Thus, the rationale for a low-NSC diet in these breeds appears lacking. Additionally, a subset of PSSM2 horses has been found to have a histologic marker (desmin aggregation) indicative of MFM, a muscle disorder characterized by weakness, atrophy, and myofibrillar disarray.[6] Based on this new finding and transcriptomic and proteomic analyses of muscle from horses with MFM, a new dietary approach has been developed for MFM horses.[31]

This new diet is informed by indictors that aberrations in cysteine-based antioxidants, oxidative stress, and the mitochondrial respiratory chain are key drivers of MFM.[31] An unanswered question is whether desmin aggregation is a late stage of PSSM2 in Arabians and Warmbloods and, if so, would Arabians and Warmbloods with PSSM2 that lack desmin aggregation benefit from the MFM diet outlined below. It seems sensible to assume that if PSSM2 horses have not responded satisfactorily to a low-NSC, high-fat diet, a trial period of 6–8 weeks on the MFM diet would be warranted (Fig. 2). Note that these recommendations are developed based on a muscle biopsy diagnosis of MFM, which does not appear to correspond with MFM diagnosis by commercial genetic tests.[30]

Because the caloric/nutrition needs and symptomology differ between MFM endurance Arabians capable of performing hours of aerobic exercise and MFM Warmbloods incapable of satisfactorily performing for 45 minutes, dietary approaches differ.

Nutrient Requirements

MFM involves muscle sarcomere breakdown and atrophy, so rations should focus on providing quality protein and specific amino acids to aid in sarcomere regenesis. Additionally, since oxidative stress is likely involved in the degenerative process, antioxidants or precursors of antioxidants are important to support the mitochondrial respiratory chain, the major source of reactive oxygen species in exercising muscle.

Forage

MFM horses will typically consume 1.5%–2% of body weight per day of hay. Good-quality grass or grass-legume mixed hays (55%–65% NDF, 10%–12% CP, 10%–17% NSC) are preferable.

Energy Sources

In the US the trend for feeding Warmbloods has been toward low-NSC, high-fat diets. This is not the case in Europe. Elite European sport horses consume feeds that are higher in NSC (25%–35%) and more moderate in fat (4%–6%).[47] There is no evidence that extremely low-NSC, high-fat diets are needed by Warmbloods with MFM. In addition, there does not appear to be a scientific reason why additional fat, a potential source of oxidative stress, would be of benefit to Warmbloods with MFM. Arabian endurance horses are typically fed higher fat diets, as Arabians depend more on fat oxidation than Thoroughbreds during exercise.[48] However, since MFM in Arabian endurance horses is related to oxidative stress resulting from fat oxidation, it is questionable whether these horses need extremely high levels of fat intake (>15% total DE intake).

Concentrates

Both Warmbloods and Arabian endurance horses in the US are typically fed fairly low levels of concentrate. In a survey of US endurance riders, concentrate intake averaged 2.27 kg/day.[49] The riders preferred lower protein concentrates (10% CP) and overall protein content of the diet averaged 10.2%, ranging from 6.2% to 15.7%. Endurance riders feed low-protein rations because they are concerned that high-protein diets may increase body heat, urine production, and water needs. While this level of protein intake may meet crude protein requirements in normal horses, it may be deficient in specific amino acids such as lysine, methionine, and threonine needed for muscle repair and generation of cysteine-based antioxidants. Leucine stimulates protein synthesis in the muscle post-exercise,[50] which would be beneficial to MFM horses. Therefore, concentrates for MFM horses should include higher levels of protein (12%–14% CP) containing high-quality amino acids and moderate levels of NSC (20%–30%) and fat (4%–8%).

Supplements

Amino acids

For horses with symmetrical topline muscle atrophy and horses with MFM, amino acid supplements are

currently recommended.[6,51] Whey-based proteins are recommended because they are rich in cysteine. Cysteine is a key component of many antioxidants, and Arabian horses with MFM appear to have an increased cysteine requirement following exercise.[31]

Antioxidants

Horses with MFM have decreased expression of mitochondrial proteins and antioxidants in their muscle.[31] Coenzyme Q10 (CoQ10) is a key component of the first step in the mitochondrial electron transport chain. Arabian and Warmblood horses with MFM have decreased expression of proteins involved in this first step. When fed with amino acids, CoQ10[g] increases mitochondrial proteins when fed to healthy horses (Valberg unpublished). CoQ10 is used in human muscle disorders and is now being trialed as a supplement for MFM horses.

Acknowledgments

Declaration of Ethics

The Authors have adhered to the Principles of Veterinary Medical Ethics of the AVMA.

Conflict of Interest

Dr. Pagan is the founder and owner of Kentucky Equine Research, which owns MicroSteed® Ration Evaluation Software, Nano-E®, Re-Leve® and Nano-Q10™. Dr. Valberg is one of the patent holders for the genetic test for type 1 polysaccharide storage myopathy and receives royalties from genetic testing and receives royalties from the sale of the equine feed Re-Leve®.

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[a]MicroSteed® Ration Evaluation Software, Kentucky Equine Research, Versailles, KY 40383.
[b]Feed XL Nutrition Software, available at feedxl.com.
[c]Equi-Analytical, Ithaca, NY 14850.
[d]Elevate® WS, Kentucky Performance Products, Versailles, KY 40383.
[e]Nano-E®, Kentucky Equine Research, Versailles, KY 40383.
[f]Re-Leve®, Kentucky Equine Research, Versailles, KY 40383.
[g]Nano-Q10™, Kentucky Equine Research, Versailles, KY 40383.