The Role of Nutrition in Managing Muscle Disorders

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KEYWORDS

• Skeletal • Muscle • Tying up • Rhabdomyolysis • Myopathy • Atrophy
• N acetylcysteine

KEY POINTS

- Horses with exertional rhabdomyolysis caused by types 1 and 2 polysaccharide storage myopathy (PSSM1 and PSSM2-ER) benefit from low nonstructural carbohydrate (NSC) diets and supplementary fat as a metabolizable form of energy.
- Horses with recurrent exertional rhabdomyolysis can be managed by lowering NSC, but not to the extremes of PSSM, and then replacing needed calories with fat.
- Arabian horses with myofibrillar myopathy (MFM-ER) can be managed with moderate NSC, supplementary fat, amino acids, and antioxidants N-acetylcysteine and coenzyme Q10.
- Warmblood horses with myofibrillar myopathy benefit from a similar diet to MFM-ER horses but with less additional fat.

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.

Classification of Exertional Myopathies

Exertional myopathies cause muscle pain and impaired performance during or after exercise. Exertional rhabdomyolysis (ER) represents a subset of exertional myopathies characterized by muscle fiber degeneration and elevations in serum creatine kinase (CK) and aspartate transaminase (AST) activities. Polysaccharide storage myopathy (PSSM) was one of the first specific causes of ER identified and is

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Vet Clin Equine ■ (2024) ■–■
https://doi.org/10.1016/j.cveq.2024.11.007 **vetequine.theclinics.com**

Abbreviations	
AST	aspartate transaminase
CK	creatine kinase
DE	digestible energy
EMND	equine motor neuron disease
ER	exertional rhabdomyolysis
FFA	free fatty acid
MFM	myofibrillar myopathy
MH	malignant hyperthermia
NRC	National Research Council
NSC	nonstructural carbohydrate
PSSM	polysaccharide storage myopathy
RER	recurrent exertional rhabdomyolysis
US	United States
VEM	vitamin E-responsive myopathy
WSC	water-soluble carbohydrate

characterized by the presence of abnormal muscle glycogen staining in muscle biopsies.[1] Genetic discoveries led to the subdivision of PSSM into type 1 PSSM (PSSM1), horses with a mutation in glycogen synthase 1, and PSSM2, horses with abnormal glycogen stains lacking the PSSM1 mutation.[2,3] With a lack of information on the cause of PSSM2, the low-nonstructural carbohydrate (NSC), high-fat PSSM1 diet was universally recommended for all horses diagnosed with PSSM regardless of whether PSSM1 or PSSM2.[4] Recommendations for feeding PSSM2, however, have now evolved according to breed-based research into muscle glycogen concentrations, histologic markers, and molecular approaches that better subclassify PSSM2.[5–10]

Forms of ER now include PSSM1, PSSM2 with ER (PSSM2-ER), malignant hyperthermia (MH), recurrent exertional rhabdomyolysis (RER), and myofibrillar myopathy (MFM-ER) in Arabians.

Other exertional myopathies do not cause ER and elevations in serum CK and AST activities with exercise.[8] Although previously grouped under the heading PSSM2, research into Warmblood horses with exercise intolerance, reluctance to go forward, collect, and engage the hindquarters led to the identification of another form of myofibrillar myopathy in this breed, MFM-WB.[7,11]

Deficiencies in vitamin E can impact performance by causing muscle atrophy and loss of strength. Temporal deficiencies affect skeletal muscle, causing vitamin E-responsive myopathy (VEM), and long-term deficiencies are neuropathic, causing equine motor neuron disease (EMND).[12,13] These disorders and nutritional requirements are covered in Carrie Finno and Erica McKenzie's article, "Vitamin E and Selenium Related Manifestations of Muscle Disease," in this issue.[14]

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 PSSM1 and RER.[4,15,16] Less evidenced-based information is available with regard to management of PSSM2-ER and MFM. Recommendations for these disorders are based largely on retrospective studies or clinical impressions.[5,17,18]

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 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 (NRC) last published its recommendations for horses in 2007.[19] NRC requirements are often considered minimums for many nutrients. Recommendations that are more commonly used in practice are also available in commercially available software such as MicroSteed[a] or Feed XL[b].
2. Select type and intake of forage. Forage should be the foundation of every equine feeding program, so it is important to establish 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 United States 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, as these nutrients rarely appear as guarantees on feed tags or bags. The American Association of Feed Control Officials (AAFCO) does not have an agreed-upon method for measuring WSC 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, New York,[c] to determine WSC and starch values in feeds, and horse owners and veterinarians can also send feeds and forages to this laboratory 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 depends on 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 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

[a] MicroSteed Ration Evaluation Software, Kentucky Equine Research, Versailles, KY 40383.

[b] Feed XL Nutrition Software, available at feedxl.com.

[c] Equi-Analytical, Ithaca, New York 14850.

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.

EXERTIONAL RHABDOMYOLYSIS

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

Sporadic Exertional Rhabdomyolysis

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.

Because low forage intake may contribute to sporadic bouts of ER, horses should be provided adequate quantities of high-quality forage. Performance horses will typically consume 1.5% to 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.

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 on caloric needs.

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,

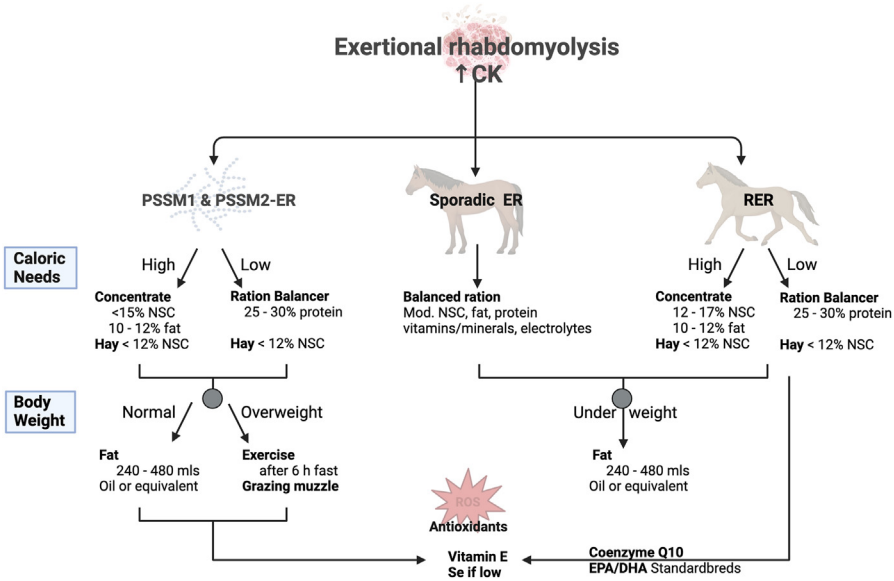

Fig. 1. An approach to managing horses with clinical signs of ER characterized by muscle stiffness, sweating, reluctance to move, and increased serum CK activity. Decisions should be based on the underlying myopathy, the horse's caloric needs, and current body weight. NSC, nonstructural carbohydrate; Omega 3s EPA, eicosapentaenoic acid, DHA eicosapentaenoic acid. (*Created in* BioRender. Valberg, S. (2025) https://BioRender.com/w15q798.)

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).

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 per day with light sweating and up to 120 to 150 g per 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.[20]

Selenium and vitamin E status should be evaluated. Low serum levels of either nutrient warrant supplementation. Natural-source vitamin E is more bioavailable than synthetic sources, and either micellized[d] or nanodispersed[e] sources rapidly restore serum status.[21,22]

Recurrent Exertional Rhabdomyolysis

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 to 35 Mcal DE per day.

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.[23] 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.

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/d).[15] The benefit of a high-fat diet for RER does not appear to be a change in muscle metabolism. Pre- and postexercise muscle glycogen and lactate concentrations are the same in RER horses fed a low-starch, high-fat diet compared with a high-starch diet.[15,24] 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.[15,25] Neurohormonal changes may develop in response to high serum glucose, insulin, and cortisol concentrations, resulting in an anxious demeanor.

Racehorses in full training typically consume 6 to 7 kg per day of concentrate. Racehorse concentrates for RER horses should contain 12% to 18% NSC and 10% to 13%

[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.

fat by weight. 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 effect of low-NSC, high-fat rations appears 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. Water-soluble carbohydrates (WSCs) produce higher glycemic responses than starch. Molasses is extremely glycemic in horses,[26] but added fat greatly reduces glycemic response, even in high-NSC feeds.[27,28] Glycemic response is also affected by rate of intake and rate of gastric emptying.[28,29] Although 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 3 days before a race to potentially boost liver glycogen and increase a horse's energy availability during the race.

As with sporadic ER, concentrate intakes less than 3 kg per 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 postexercise serum CK activity occurs within a week of commencing a low-starch, high-fat diet.[15] Days of no training and standing in a stall are discouraged, because postexercise CK activity is higher after 2 days of rest compared with values taken when performing consecutive days of the same amount of submaximal exercise.[15]

The gluteal muscle of RER-susceptible Standardbreds is characterized by perturbation of pathways for calcium regulation, cellular/oxidative stress, and inflammation weeks after an episode of rhabdomyolysis.[30] Supplementation with vitamin E, vitamin C, and Coenyzme Q10 may help reduce oxidative stress[15,24,31,32] Additionally, supplementation with n−3 long-chain polyunsaturated fatty acids (EPA and DHA) may be useful in mitigating long-term inflammation.[33,34]

Type 1 Polysaccharide Storage Myopathy and Type 2 Polysaccharide Storage Myopathy with Exertional Rhabdomyolysis

PSSM1 and recently PSSM2 in Quarter Horses with ER (PSSM2-ER) have been found to be glycogen storage diseases.[1,5] Dietary recommendations for PSSM1 are based on controlled trials, whereas recommendations for PSSM2-ER are based on owner surveys.[4,5]

Meeting the horse's caloric requirements for an ideal body weight is the most important consideration in designing a ration for PSSM1 and PSSM2-ER, 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% to 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 (FFAs) before exercise and alleviate any restrictions in energy metabolism in muscle.

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.[16] Because insulin stimulates 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 per day. A mixed-grass hay (12% NSC, 55% NDF, 2.0 Mcal DE/kg) fed at 9 kg per day meets the horse's daily caloric requirement. In contrast, 8 kg of 4% NSC hay (1.7 Mcal DE/kg) would provide 13.6 Mcal DE per day, which would allow a reasonable addition of 4.4 Mcal DE from fat per day (530 mL of vegetable oil).

A high-NSC diet increases the propensity to develop muscle pain with aerobic exercise in PSSM1 and PSSM2-ER horses.[4,35] A high-NSC diet results in enhanced glycogen synthase activity, which may impair oxidative metabolism of substrates such as pyruvate and fatty acids.[36] PSSM1 horses on high-NSC diets have low plasma non-esterified FFA concentrations, possibly because of suppression of lipolysis by high insulin.[4] Low dietary starch and fat supplementation facilitate muscle fat metabolism in PSSM1 horses.

Concentrates for PSSM1 and PSSM2-ER horses should be low in NSC (<15% by weight) and low glycemic. High-fat (10%–12%) concentrates can be included in the diet 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.[26] 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.

Myofibrillar Myopathy in Arabian Endurance Horses

Whereas some excitable endurance horses present with RER early in races, other seasoned endurance horses develop signs of muscle stiffness, cramping, myoglobinuria, and high serum CK at the end of long endurance races. Muscle biopsies from the latter horses recently found glycogen concentration to be normal. Rather, myofibrillar disarray and characteristic aggregates of the cytoskeletal protein desmin were identified, leading to the term myofibrillar myopathy with ER (MFM-ER) being applied to endurance horses with desmin aggregates and high serum CK activity.[6,37] This subset of horses was previously classified as PSSM2 because of glycogen pooling in regions of broken myofilaments.[11,37] Transcriptomic and proteomic studies implicate a pro-oxidant environment and low cysteine-based antioxidants as potential causes of MFM-ER and led to a new approach to managing MFM-ER.[9]

Endurance riders feed low-protein rations because they are concerned that high-protein diets may increase body heat, urine production, and water needs. Although 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 in MFM-ER. Leucine stimulates protein synthesis in the muscle after exercise,[38] which would be beneficial to MFM-ER horses. Arabian endurance horses are typically fed higher-fat diets, as Arabians depend more on fat oxidation than Thoroughbreds during exercise.[39] However, because 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). Because oxidative stress is likely involved in the degenerative process, antioxidants or precursors of antioxidants are additionally important to support the mitochondrial respiratory chain, the major source of reactive oxygen species in exercising muscle.

MFM-ER horses will typically consume 1.5% to 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.

In a survey of US endurance riders, concentrate intake averaged 2.27 kg per day, with an average protein content of 10% CP (range 6.2%–15.7% by weight).[32] Concentrates for MFM-ER 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

Whey-based proteins are recommended because they are rich in cysteine. Cysteine is a key component of many antioxidants, and MFM-ER Arabian horses appear to have an increased cysteine requirement following exercise.[9] The most absorbable form of cysteine is N-acetylcysteine, and supplementation with N-acetylcysteine has been shown to increase gluteal muscle glutathione (a ubiquitous cysteine-based antioxidant) in healthy fit thoroughbreds.[40]

Coenzyme Q10 (CoQ10) is a key component of the first step - Complex I - in the mitochondrial electron transport chain, which generates reactive oxygen species. MFM-ER Arabians have altered expression of subunits of complex I.[9] When fed to healthy horses, CoQ10[9] supplementation enhanced electron transfer capacity via complex II, which could decrease reactive oxygen species formation and enhance fat metabolism. CoQ10 is used in human muscle disorders and is now being trialed as a supplement for MFM horses.

EXERTIONAL MYOPATHIES

Myofibrillar Myopathy in Warmblood Horses

Research into a subset of Warmbloods with PSSM2 found normal muscle glycogen concentrations, myofibrillar disarray, and a desmin aggregation leading to a reclassification as MFM-WB.[7] Based on this new finding and transcriptomic and proteomic analyses of muscle, a new dietary approach was developed for MFM-WB horses.[10] This new diet outlined in **Fig. 2** is informed by indicators that myofibrillar instability, Z disc signaling, aberrations in cysteine-based antioxidants, oxidative stress, and the mitochondrial respiratory chain are key drivers of MFM-WB.[10] A diagnosis of MFM-WB is based on muscle biopsy and not commercial genetic tests for PSSM2 or MFM that have not been scientifically validated through peer-reviewed publication.[41–43] Many horses with signs of exercise intolerance are initially managed with a low-NSC, high-fat diet with limited relief of exercise intolerance. It seems sensible to assume that if horses have not responded satisfactorily to this diet, a trial period of 6 to 8 weeks on the MFM-WB diet would be warranted provided other causes of exercise intolerance have been investigated and ruled out (see **Fig. 2**).

Because Warmblood horses diagnosed with MFM have normal glycogen concentrations, the rationale for a low-NSC diet in these breeds appears lacking.[11,37] The presence of muscle sarcomere breakdown and atrophy indicates that rations should focus on providing quality protein and specific amino acids as described for MFM-ER to aid in sarcomere regenesis. Additionally, because oxidative stress is likely involved in the degenerative process, antioxidants or precursors of antioxidants are important to reduce oxidative stress and support the mitochondrial respiratory chain, the major source of reactive oxygen species in exercising muscle.

[9] Nano-Q10, Kentucky Equine Research, Versailles, KY 40383.

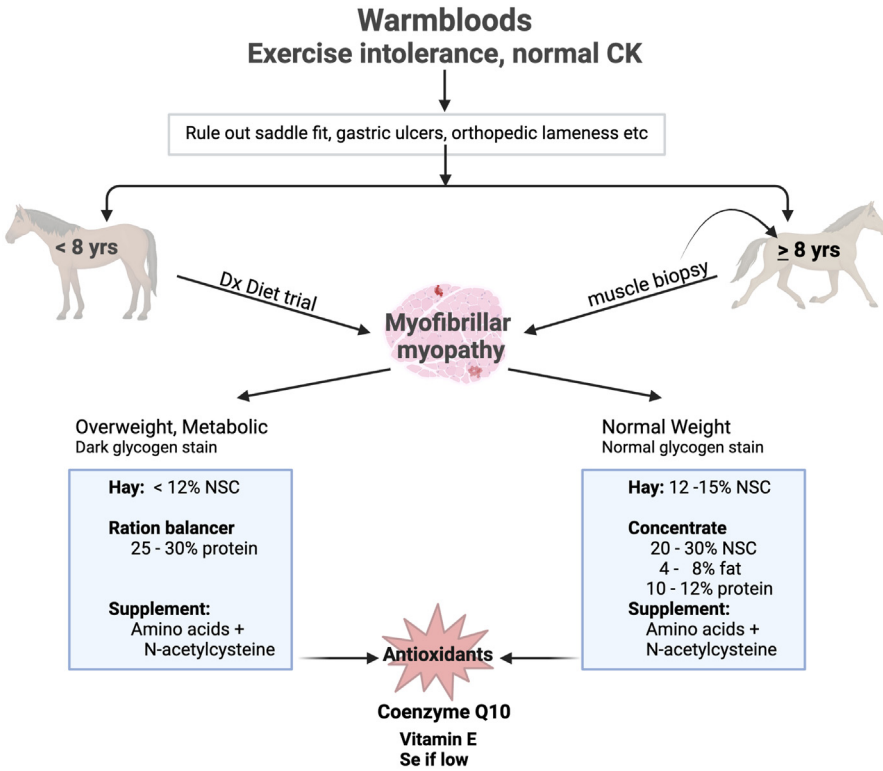

Fig. 2. An approach to managing MFM-WB horses with clinical signs of exertional myopathy characterized by exercise intolerance and normal to mildly increased serum CK activity. To conclude a myopathy is responsible for exercise intolerance, other common causes should first be ruled out. Because the diagnostic feature desmin aggregation may lag behind clinical signs, muscle biopsies from young horses can give false-negative results; therefore a treatment trial for 4 weeks looking for a clinical response may be the best diagnostic option for young horses. Horses that are overweight or have evidence of equine metabolic syndrome are fed a diet lower in NSCs than normal-weight horses. (*Created in* BioRender. Valberg, S. (2025) https://BioRender.com/l64f863.)

MFM horses will typically consume 1.5% to 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.

In the United States, 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%).[44] There is no evidence that extremely low-NSC, high-fat diets are needed by Warmbloods with MFM-WB unless horses have metabolic disease, excess adipose cells, or dark muscle glycogen stains in muscle biopsies. 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.

Concentrates for MFM-WB horses should include higher levels of protein (12%–14% CP by weight) containing high-quality amino acids and moderate levels of NSC (20%–30%) and fat (4%–8%). This level of protein intake will provide 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 after exercise,[38] which would be beneficial to MFM-WB horses.

Supplements

For horses with symmetric topline muscle atrophy and horses with MFM, amino acid supplements are currently recommended.[8,45] Whey-based proteins are recommended, because they are rich in cysteine. The top upregulated gene in MFM-WB horses is *CHAC1,* and the CHAC1 enzyme degrades the cysteine-based antioxidant glutathione. Because of this, supplementation with N-acetylcysteine is recommended. N-acetylcysteine can increase postexercise gluteal muscle glutathione.[40]

Horses with MFM have altered expression of genes and proteins involved in mitochondrial complex 1 and glutathione degradation in their muscle.[10] Coenzyme Q10 (CoQ10) is a key component of the first step in the mitochondrial electron transport chain and a potent antioxidant, and has been recommended for MFM-WB.

A survey of 50 owners of MFM-WB horses feeding the recommended diet (17 responses) found that 92% of horses improved after 4 weeks, with hindlimb engagement and reluctance to work being the clinical signs to show the most improvement (Valberg unpublished observation, 2023).

SUMMARY

In combination with exercise, nutrition is an essential component of managing horses with myopathies. The type of diet needed depends on the specific myopathy in question. ER represents a subset of exertional myopathies characterized by elevations in serum CK and AST activities. Several forms of ER including PSSM1, PSSM2-ER, and RER require low-NSC diets. If extra exergy is also required, then supplemental fat may also be beneficial. MFM horses do not require low-NSC, high-fat diets. Instead, they respond to protein and amino acid supplementation to rebuild muscle and antioxidants such as N-acetylcysteine, CoQ10, and vitamin E.

CLINICS CARE POINTS

- In order to best manage horses with myopathies it is important to distinguish between those with exertional rhabdomyolysis by measuring serum CK and those with normal serum CK and exercise intolerance due to myofibrillar myopathy.
- An exercise challenge test with CK measured 4 h after exercise can be of benefit to make this distinction.
- While horses with exertional rhabdomyolysis are fed diets with lower nonstructural carbohydrates and fat supplementation, horses with myofibrillar myopathy are fed moderate amounts of nonstructural carbohydrates, less fat, increased amino acids with N-acetylcysteine and antioxidants.

DISCLOSURES

J.D. Pagan is the founder and owner of Kentucky Equine Research, which owns Microsteed Ration Evaluation Software, Nano-E, Re-Leve, MFM Pellet, and Nano-Q10. S.J. Valberg directs the Valberg Neuromuscular Disease Laboratory (ValbergNMDL.com) and receives remuneration for analyzing muscle biopsies. Her website is sponsored by Kentucky Equine Research, and she receives royalties from the PSSM1 genetic test and the feeds Re-Leve and MFM Pellet developed in association with Kentucky Equine Research.

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