Characteristics and dietary management of Exertional Rhabdomyolysis in horses

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Sammanfattning

Fenomenet korsförlamning blev först känt som “Monday-morning disease” (Zentek, 1991). Tillståndet förknippas främst med arbetshästar som efter en hård arbetsvecka fick en dags vila. När hästarna åter skulle användas i arbete, följande måndag, var de stela, visade tecken på smärta i bakdelen och var ovilliga att röra på sig (Jones, 2003). Korsförlamning hos hästar har huvudsakligen två bakomliggande orsaker. Malign Hypertermi (MH) och ”polysaccharide storage myopathy” (PSSM). MH är ett ärftligt tillstånd där en defekt i den intracellulära kalciumregleringen orsakar korsförlamning, smärta i musklerna och stel gång (Lentz *et al*., 1999). Det är framförallt fullblod som lider av MH och symptomen anses utlösas av olika stressfaktorer (Valberg *et al.,* 1999). PSSM orsakar korsförlamning hos t ex Quarterhästar och är en neuromuskulär sjukdom. Hästar som har PSSM tycks ha ett överaktivt glukosupptag från blodet (De La Corte *et al.,* 2002), och ackumulerar på grund av detta onormala mängder av glycogenliknande polysackarider i sina musklerfibrer, har förhöjda muskelglykogenkoncentrationer och utvecklar korsförlamning (Valberg *et al.,* 1999; Valentine *et al.,* 2000). Den onormala inlagringen av glykogen kan åskådliggöras genom färgning av muskelbiopsier. Ackumuleringen av polysackarider i musklerna tycks öka med hästens ålder (De La Corte *et al.,* 2002). Hästar som inte sätts i träning och som inte tvingas till rörelse kan ha både MH och PSSM utan att utveckla symptom på korsförlamning (Valentine *et al.,* 2000; De La Corte *et al.,* 2002). Uttrycket av både MH och PSSM, i form av korsförlaming, kan regleras med hjälp av regelbunden motion och en balanserad diet som innehåller så lite socker och stärkelse som möjligt. För att tillgodose det höga behovet av energi hos t ex galopphästar kan man komplettera foderstaten med fett istället för socker och stärkelse (Geor, 2005).

Abstract

Tying-up or exertional rhabdomyolysis (ER) was previously known as Monday-morning disease (Zentek, 1991). Monday morning disease was associated with workhorses that was given a day of rest after a week of hard work. When the horses were supposed to return to work on the following Monday, they developed stiffness and pain in the hindquarter musclulature, and reluctance to move (Jones, 2003). Tying–up or ER in horses is mainly caused by two defects, Malignant Hyperthermia (MH) and Polysaccharide Storage Myopathy (PSSM). MH is an inherited condition where a disorder in cellular calcium regulation causes ER manifested as muscle pain and stiffness of gait (Lentz *et al*., 1999). Especially Thoroughbreds are suffering from MH, and the symptoms are thought to be induced by different stress factors (Valberg *et al.,* 1999). PSSM is causing ER in *e.g.* Quarter Horses (QH) and is a neuro-muscular disorder. Horses with PSSM are suggested to have an enhanced glucose clearance from the bloodstream (De La Corte *et al.,* 2002). Affected horses then accumulate abnormal glycogen related polysaccharide inclusions within their muscle fiber, have muscle glycogen concentrations above normal and develop ER symptoms (Valberg *et al.,* 1999; Valentine *et al.,* 2000). The abnormal glycogen inclusions within the muscles can be visualized by staining muscle biopsies. The accumulation of abnormal polysaccharides is suggested to increase as the individual becomes older (De La Corte *et al*., 2002). Horses that are not forced to move, *e.g.* not put into training, are not always developing ER symptoms even if they have PSSM or MH (Valentine *et al.,* 2000; De La Corte *et al.,* 2002). The expression of both MH and PSSM as tying-up can be managed with regular exercise and a balanced diet, composed of as little starch and sugars as possible. To meet the high energy need

in *e.g.* thoroughbred racehorses, fat can be added to the diet instead of sugar and starch (Geor, 2005).

Introduction

Tying-up or exertional rhabdomyolysis (ER) was previously known as Monday-morning disease (Zentek, 1991). Monday morning disease was associated with workhorses that was given a day of rest after a week of hard work. When the horses were supposed to return to work on the following Monday, they developed stiffness and pain in the hindquarter musculature, and reluctance to move (Jones, 2003). Today, ER is not an uncommon condition even if the horses are used only for pleasure riding, and horses are euthanized because of a high frequency of ER (Firshman *et al*., 2003).

Exertional Rhabdomyolysis in horses is a disease which is caused mainly by two muscle defects. The condition occurs in many different breeds *e.g.* Quarter horse (QH), draft horse related breeds, Arabians, Thoroughbreds and Morgans (Valentine *et al.,* 2000; McKenzie *et al.,* 2003). Previously, ER in horses was assumed to be one condition caused by the same defect, because the affected horses showed similar symptoms such as ER, muscle pain, stiffness and even colic-like signs (Ribeiro *et al.,* 2004). More recent studies have pointed out that the underlying aetiology and cause of ER may differ between different horse breeds (Valberg *et al.,* 1999).

Malignant Hyperthermia (MH) is one of the defects causing ER. Malignant Hyperthermia is an inherited condition where a disorder in cellular calcium regulation causes ER symptoms, *i.e.*, muscle pain and stiffness of gait (Lentz *et al*., 1999). Especially Thoroughbreds are suffering from MH, and the symptoms are thought to be induced by different stress factors (Valberg *et al.*, 1999). It is not a metabolic disorder and is mentioned here only to give an overview of the complexity of ER, and for comparison with another defect that causes ER, Polysaccharide Storage Myopathy (PSSM), which is a metabolic disorder. The purpose of this review was to investigate the underlying causes of ER and to find out how the disease, primarily when caused by PSSM, can be managed by means of adapting the diet.

Characteristics of exertional rhabdomyolysis

Symptoms

Development of muscle stiffness, pain, shifting lameness, gait changes, colic-like signs, muscle atrophy and ER are clinical features typical for PSSM (Ribeiro *et al.,* 2004; Geor, 2005). Usually the horse develops signs of ER when exercising. The type of exercise at the time of ER is variable from riding and lungeing to hand walking. A horse not exercising can still develop ER, *e.g.* when standing in a trailer or at pasture. Signs of ER at exercise can be observed at all gaits (Firshman *et al.*, 2003).

Veterinary diagnosis and treatment

Blood samples and analysis

When diagnosing a horse with ER the veterinarian draws a blood sample that is being analyzed (C. Petersson, pers. comm.). An increase in serum Creatine Kinase (CK) and Aspartate Transaminase (AST) are signs that are thought to be indicative of ER (Valentine *et al.,* 2001).

Creatine Kinase catalyses chemical reactions within the muscle, and when elevated levels of serum CK are detected in the blood stream, muscle damage is suspected (Sjaastad *et al*., 2004). Elevated AST levels are also an indicator of muscle damage associated with recumbency (Skenderi *et al*., 2006; Valentine & Löhr, 2007). Elevated levels of AST are also thought to be indicative of liver damage (Nyblom *et al.,* 2004). In the cell, AST is involved in formation of adenosinetriphosphate (ATP) (Pesch *et al*., 2006).

Veterinary treatment

Different methods are used by veterinarians to treat horses with ER. Examples of treatments are phenylbutazone, muscle relaxants and sedatives, and vitamin E and selenium supplements. These treatments primarily help to ease the symptoms of ER, however, the underlying causes are not cured. Most horses need repeated treatments, and there are also other types of treatments recommended, such as hand-walking (Firshman *et al.,* 2003).

The muscle

Features of PSSM

Polysaccharide Storage Myopathy is a neuromuscular disorder where glycogen-related polysaccharide inclusions are accumulated within skeletal muscle fibres (Geor, 2005). A horse with PSSM usually develops pain, stiffness and ER when exercising together with an increase in serum CK activity (McKenzie *et al.,* 2003). The most common occurring sign of PSSM is ER. In some cases, episodes of ER can be combined with mild colic, a shiver-like gait, recumbency, lameness and swollen muscles. It is the hindquarter musculature of the horse that is most often affected. The back, the forelimb, the abdominal, the neck and pectoral musculature are other muscle areas that also can develop stiffness and pain (Valberg *et al*., 1999).

Polysaccharide storage myopathy was first reported in Quarter Horse-related breeds with recurrent ER (McKenzie *et al.,* 2003). The condition can also occur in other breeds such as draft-horse related breeds, Warmblood horses, Morgans and Arabians (Valentine *et al.,* 2000). Different breeds have different incidence of PSSM. According to Valentine & Cooper (2005), in a group of Thoroughbreds about 27 % of the horses had had ER, while in a group of draft-related horses, in the same study, about 86 % of the horses had had ER (Valentine & Cooper, 2005). The frequency and severity of ER is different also between individual horses. In some horses ER occurs just once (Valberg *et al.,* 1999). Repeated episodes of ER due to light exercise, *e.g* the work that is performed by horses used for pleasure riding, is also an occurring scenario. In those cases the disease may be the end of the career of the horse, and in severe cases with a high frequency of ER the horse is euthanized (Firshman *et al.,* 2003).

Horses affected by PSSM do not necessarily have to show clinical signs of ER (Firshman *et al.,* 2003). The disorder is thought to be inherited but may not show clinically for several years (Valentine *et al.,* 2000). De la Corte *et al.* (2002) suggests that clinical signs of ER do not have to be evident until the affected horse is forced to move. Therefore it is possible that horses affected by PSSM but not put into training never develop symptoms of ER (Valentine *et al.,* 2000; De La Corte *et al.*, 2002). If horses affected by PSSM are put into training, Firshman *et al.* (2003) suggests that these horses have the same ability as unaffected horses to build muscles.

Diagnosis of PSSM

The diagnosis of PSSM is done by finding periodic acid-schiff (PAS)-positive amylase-resistant inclusions of polysaccharide complex within biopsies of the muscle fibres (Valentine *et al.*, 2000*;* Riberio *et al.*, 2004). The severity and changes in skeletal muscles of horses with PSSM is variable. Even if the horse is not showing clinical signs of PSSM, *e.g.* ER, the muscle biopsies of clinically normal horses can contain aggregates of material replacing part of or the entire affected fibre segment. This was an unexpected finding in the study by Valentine *et al.* (2000), and may not be indicative of underlying myopathy. Similar signs can be found in draft horses that have clinical signs of neuromuscular dysfunction due to PSSM, and the accumulations are then considered part of the spectrum of changes possible in the PSSM disorder. Ponies participating in the study by Valentine *et al.* (2000) did not have clinical signs of PSSM. This could be explained by the fact that they were not put into training (Valentine *et al.*, 2000). Other studies suggest that when the horse itself is able to control its exercise level, they do not show the typical signs of pain and stiffness (De La Corte *et al.*, 2002). The accumulation of polysaccharides within the muscles is also suggested to increase in the muscle fibres as the horse grows older (De La Corte *et al.*, 2002*; Geor.*, 2005). Looking at muscle biopsies from horses with PSSM, centrally located nuclei can be seen as small, round and darker stained dots within the muscle fibre (Figure 1). These nuclei are thought to be an indicative feature of PSSM (De La Corte *et al.*, 2002). Another characteristic feature of PSSM is subsarcolemmal vacuoles. These are small vacuoles, located just below the membrane of the endoplasmic reticulum (De La Corte *et al.*, 2002).

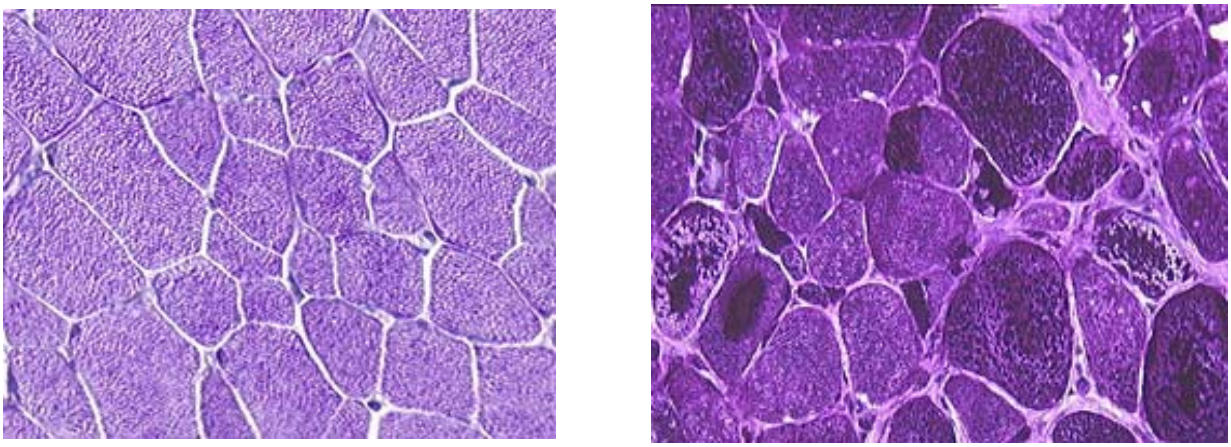

Figure 1. Pictures demonstrating PAS-staining. The picture to the left is demonstrating a normal muscle fibre without polysaccharide inclusions. The picture to the right shows darker areas which are demonstrating fibres with abnormal polysaccharide and glycogen inclusions. (Pictures taken from University of Minnesota http://www.cvm.umn.edu/umec/lab/PSSM.html 2008-04-28)

De La Corte *et al.* (2002) studied horses bred to inherit PSSM. The horses were followed from birth and different tests were performed to determine the development of different features typical for PSSM. All horses in the study had a slightly elevated PAS-staining intensity compared to controls (Table 1). However, the age of the foals when abnormal PAS-staining was discovered were variable (Table 1). Foal no. 4 was three years old when the abnormal polysaccharide inclusions were first detected, suggesting that the polysaccharides within the muscles accumulated with time (Table 1) (De La Corte *et al.*, 2002).

Table 1. Age of horses when discovering different features of PSSM, PAS-staining intensity and glycogen concentrations compared to controls based on a study by De La Corte *et al.* (2002)

Foal no.	**1 (filly)**	**2 (filly)**	**3 (filly)**	**4 (colt)**
Age of mucle necrosis (*Gluteus medius*)	6 month	6 month	-	-
Age when centrally located nuclei were first discovered	2 year	6 month	1 year	-
Age when centrally located nuclei secondly were discovered	-	2 year	2 year	-
Subsarcolemmal vacuoles	6 month	6 month	-	15 month
Age when abnormal PAS-staining was discovered	15 month	18 month	7 month	3 year
Percentage of fibre affected by PAS-staining	20%	1-2%	Very few fibres affected	Very few fibres affected
PAS intensity	+	+	+	+
Glycogen concentration (mmol/kg)	648 ± 33 (++)	514 ± 20 (++)	397 ± 35 (0)	614 ± 44 (++)

+ = slightly elevated compared to controls
++ = markedly elevated compared to controls
0 = results do not significantly differ from the results of controls
- = no abnormalities discovered

None of the control foals had any abnormal polysaccharide inclusions identified. In one biopsy sample of one of the foal in the control group, a few subsarcolemmal vacuoles were found. The mean glycogen concentration of the controls was 434 ± 23 mmol/kg (De La Corte *et al.*, 2002).

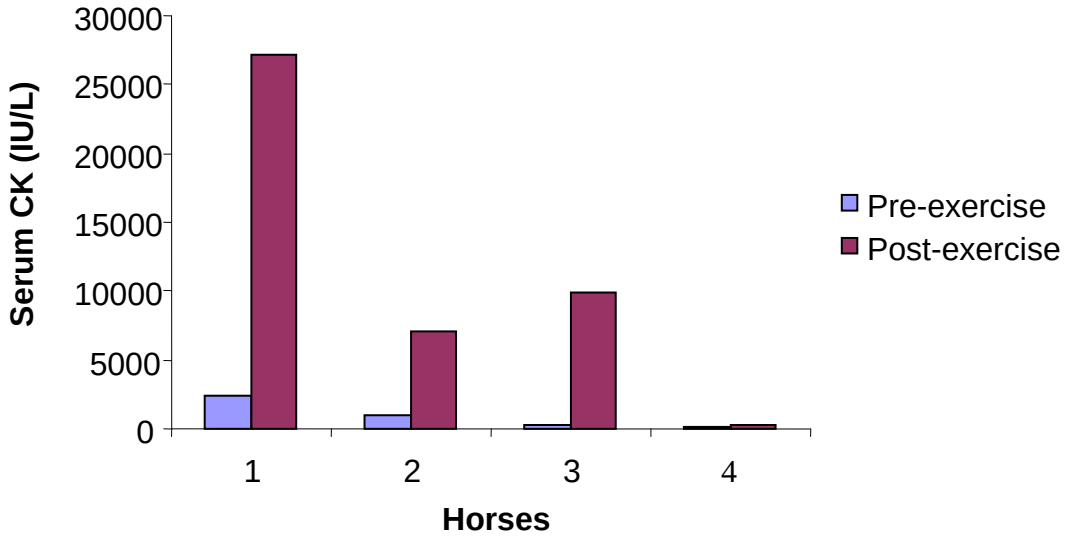

Figure 2. Concentrations of serum Creatine Kinase pre-& postexercise in four horses suffering from PSSM. Values based on a study by De La Corte *et al.* (2002).

In the study by De La Corte *et al.* (2002), horses 1, 2 and 3 all developed some kind of stiffness and pain during exercise, horse 4 however did not. Looking at the serum CK levels pre-exercise

and post-exercise (Figure 2) it is evident that horse 4 is not affected as much as the others. Horses 1, 2 and 3 all had a marked increase in serum CK compared to controls. In horse 4, no significant elevation was observed. The author suggested that the gender of this foal (no 4), which was the only male with PSSM participating in the study, may have something to do with the less severe signs (De La Corte *et al.*, 2002).

The mechanism behind PSSM

Quarter Horses affected by PSSM have a high concentration of glycogen in their muscles (Valberg *et al.* 1999). In human beings and other animal species where glycogen storage disorders have been identified, the disorder is caused by a single gene mutation that causes deficiencies in the enzyme activities of the glycogenolytic or glycolytic pathways (De La Corte *et al.*, 2002). Decreased capacity for glycogen utilization is however not the defect causing horses to accumulate excessive glycogen within their muscles (Geor, 2005). Studies done by Valberg *et al*. (1999) suggests that net glycogen breakdown and accumulation of lactate in skeletal muscle were similar in both QH affected by PSSM as in QH not affected by PSSM. Also, the activities of glycolytic enzymes were the same in affected and unaffected QH (Geor, 2005). Intravenous and oral glucose tolerance tests in adult PSSM horses suggests that the accumulation of polysaccharide within the skeletal muscle of the horse is due to an overactive glucose clearance from the bloodstream, which means that at the same insulin concentrations, more glucose is taken up from the blood by the muscles of affected horses compared to clinically healthy horses (De La Corte *et al.*, 2002; Firshman *et al.*, 2003).

Malignant hyperthermia

In Thoroughbred horses the primary cause of ER is not due to PSSM. The abnormal contracture of the muscles in ER-affected Thoroughbreds indicates that the condition can be caused by MH. Thoroughbreds suffering from ER are hypersensitive to agents that induce release of calcium from the endoplasmic reticulum, which in muscle fibres is called sarcoplasmic reticulum (Sjaastad *et al.*, 2004). Excitement and stress can trigger excessive muscle contraction in horses with ER caused by MH. Nervous temperament is a key factor in horses affected by MH. There are a number of different features varying between biopsies from horses suffering from MH and horses suffering from PSSM. Biopsies from horses with MH, unlike horses with PSSM, have a normal PAS-staining, normal glycogen concentrations, normal metabolic response to exercise, but affected horses have numerous muscle fibres with centrally located nuclei (Valberg *et al.*, 1999). The finding of centrally located nuclei is a nonspecific sign typical for muscular regeneration (Lentz *et al*., 1999). It is suggested that mares tend to experience ER caused by MH more frequent than males (Firshman *et al.*, 2003; Cole *et al.*, 2004). However in an Australian study based on questionnaires to horse owners, sex predilection for MH was not found (Cole *et al*., 2004).

How to manage horses with exertional rhabdomyolysis

The diet

It seems like PSSM horses have an abnormal carbohydrate metabolism (Valentine *et al.,* 2001). To manage the condition it is therefore important to enhance glucose and glycogen utilization (Valberg *et al.,* 1999). This can be done by feeding a diet consisting of as little starch and sugar as possible (Valentine *et al.,* 2001). High calorie requiring horses such as race-horses and lactating mares may need extra energy to complement a diet containing primarily forage. By adding fat to the diet this energy need can be covered (Valentine *et al.,* 2001; McKenzie *et al.,* 2003). It is primarily the Non Structural Carbohydrates (NSC) content of the feed, *e.g.* sugar and starch, that should be taken into consideration when composing diets to PSSM-affected horses. The fermentation of hemicellulose, Neutral Detergent Fiber (NDF), and Acid Detergent Fiber (ADF) in the large intestine produces Volatile fatty acids (VFA). Volatile fatty acids can be used as energy by the horse and the fermentation of fiber helps to keep a healthy environment in the large intestine (Valentine *et al.,* 2001).

The appropriate balance between fat and NSC in the diet, for horses with PSSM, is not yet established. This balance is likely to differ between different horses (Valentine *et al.,* 2001). Since horses with PSSM is likely to have an enhanced insulin sensitivity, adding fat to the diet provides energy without stimulating insulin secretion and glucose uptake into muscle cells, and thereby muscle glycogen concentrations is reduced (Firshman *et al*., 2003). To control the dysfunction of the muscles, diets composed for horses suffering from PSSM should contain at least 20% of the daily calorie intake from fat and not more than 15% of the daily calorie intake from starch and sugar (Valentine *et al.,* 2001). A study by Ribeiro *et al.* (2004) indicated that blood glucose response was higher, when feeding horses with PSSM a diet where a majority of the digestible energy (DE) came from starch. Also, horses fed a high-starch diet had a significantly higher insulin response compared to horses where fat was the major component of the DE (Ribeiro *et al*., 2004). When feeding horses a high-fat diet, the concentrations of Free Fatty Acids (FFA) in the gastrointestinal tract is higher compared to when feeding less fat. Since horses with PSSM have an enhanced sensitivity to insulin, it is favourable with a high amount of FFA which is negatively correlated with insulin concentration. Lower insulin concentration slows down the transport of glucose from the blood stream into the muscles in PSSM-horses (Ribeiro *et al*., 2004). A high-fat diet or a high-starch diet did not however affect the number of muscle fibres containing abnormal polysaccharide inclusions (Ribeiro *et al.,* 2004). Diets containing more than 25% of the total daily calories from fat have been fed to horses with PSSM. The diet was formulated by replacing grain with alfalfa pellets and vegatble oil. This type of diet did not cause ER symptoms in horses with PSSM (Valentine *et al.,* 2001).

A high level of serum CK is assumed to be indicative of ER (De La Corte *et al.,* 2002; Ribeiro *et al.,* 2004). Feeding PSSM-horses a high-fat diet results in serum CK concentrations within a normal range, while PSSM-horses fed a higher concentration of starch had increased serum CK concentrations (Ribeiro *et al.,* 2004). Two horses out of four had a marked increase in serum CK when fed diets containing more starch than 8% of the daily DE intake. These horses also showed clinical signs of ER, such as stiffness, when exercised on a treadmill. The same horses did not show signs of stiffness when fed a high-fat diet (Ribeiro *et al.,* 2004).

It is necessary to analyse the forage given to horses. Forage can possibly contain high amounts of NSC in the form of sugars, and when controlling the diet of horses affected by PSSM, high amounts of NSC should be avoided (Valentine *et al.,* 2001). Since grass possibly can contain high amounts of sugar, when on pasture, horses that are not fed any concentrates still can develop ER (Firshman *et al.,* 2003).

Exercise

Improving the feeding of affected horses is often not enough to achieve a decrease in severity and frequency of ER. Regular daily exercise is recommended for horses with PSSM (Valberg *et al.,* 1999). Studies show that owners that followed a specific training routine got significantly better results than the owners that only change the diet of the horse (Geor, 2005). Horses for which both dietary recommendations and exercise recommendations were followed were more likely to improve in severity and frequency of ER, than horses for which only dietary recommendations were followed. However, no significant difference between horses that followed both recommendations and horses that were only exercised correctly was found (Firshman *et al.,* 2003). Daily exercise is suggested to be important because it enhances the capacity for glucose oxidation and fatty acid oxidation by skeletal muscle. Muscle glycogen concentration may with time be decreased in horses suffering from PSSM if they are exercised (De La Corte *et al.,* 2002). De La Corte *et al*. (2002) also suggests that a low-starch diet may reduce glycogen concentrations, but do not totally prevent the accumulation of abnormal polysaccharides within skeletal muscle fibres. Accumulation of polysaccharides in skeletal muscle occurs later in life and seems to be a secondary characteristic of PSSM (De La Corte *et al*., 2002).

Discussion

A blood sample taken from the horse only tells if concentrations of serum CK and AST are above normal levels, it does not say which type of deficiency the horse suffers from, PSSM or MH (C. Petersson, pers. comm.). Both conditions are managed in the same way with a low starch diet and regular exercise. However, MH is thought to be induced by stress and it is my belief that the life of a Thoroughbred race horse is stressful. If that is not taken into account, it does not matter if the ER-symptoms are managed with diet and exercise, ER will still develop in MH-horses living in a stressful environment even if the severity and frequency ER might be lowered.

The diagnosis criteria of horses with PSSM are controversial, since the blood sample only tells us if the concentrations of serum CK and AST are elevated. It is questionable if it is the abnormal polysaccharide inclusions that are a diagnostic feature of PSSM. The ponies and draft horses mentioned by Valentine *et al.* (2000) had such inclusions without ER-symptoms, and it is not known when the inclusions are thought to be normal or abnormal. The subsarcolemmal vacuoles mentioned by De La Corte *et al.* (2002) can also be indicative of PSSM. Veterinarians are diagnosing ER based on the stiffness and pain of the horse, combined with elevated values of serum CK and AST. When diagnosing PSSM it seems logical that more than one feature should be taken into account. However, it is not possible to do a biopsy of all horses developing stiffness and pain during exercise, and maybe that is not needed since the long-term regulation of MH and PSSM are the same; regular exercise and low starch diet.

As horses traditionally are fed a diet where a majority of the DE comes from starch, it is not surprising that horses with PSSM develop ER. Studies have indicated that horses with PSSM can have a decrease in severity and frequency of ER when fed a diet where most of the DE comes from fat (Firshman *et al.,* 2003). With the right diet, PSSM-horses have the same athletic abilities as normal horses. There was no significant difference in the use of affected and unaffected horses, both were used for pleasure riding, and both unaffected and affected horses seem to have the same ability to build muscles (Firshman *et al.,* 2003). The prices for grain today are high, and most horses used for pleasure riding can be fed diets containing more forage without loosing weight. Horses affected with ER can thus be given the ability to function almost as clinically normal horses, and the horse-owners will save money on both feeding regimes and veterinary costs.

When doing studies on ER and the underlying aetiologies there are always a limited number of horses participating. The horses in the studies have often shown clinical signs of PSSM such as ER. It would be interesting to do a study and focus on clinically healthy horses to see which proportion of clinically healthy and hard working horses that have abnormal polysaccharide inclusions within their muscles, muscle fibers with centrally located nuclei and subsarcolemmal vacuoles without developing ER.

The dietary energy source modifies clinical expression of PSSM. A horse that is fed large amount of concentrates and receives little exercise have more frequent and severe clinical signs of ER than horses fed smaller amounts of concentrates and receives regular exercise (Firshman *et al.,* 2003). Feeding affected horses a diet with restricted hydrolysable carbohydrate content added up with fat results in decreased frequency and severity of episodes of muscle pain and necrosis. Whether or not it is the high fat content of the diet or the low carbohydrate content of the feed that is the reason for the improved clinical result is not yet established. I can see the importance of knowing if it is the high amount of fat or the low content of starch that is the reason for clinical improvement of the horses for research purposes. It is not easy to compose a suitable feeding plan for horses affected by PSSM. Naturally diets with a low content of starch should be recommended, but the optimal balance between fat and starch may vary between horses (Valentine *et al.,* 2001). The horse- owners have a responsibility to manage the feed and exercise of their horse, so that the horse stay healthy and do not put on unnecessary weight (Valentine *et al.,* 2001).

Conclusion

Horses are grazers and are not meant to eat large amounts of starch. The tradition to give horses large amounts of grain, which contain starch, to meet the need for energy in the hard working horse makes little sense from a health point-of-view. Writing this review it has become clear that many problems associated with ER can be managed with common sense. Feeding horses forage of good nutritive value, it would be possible to lower the concentration of starch in the diet and still keep the horses healthy and in good shape without development of ER, even if they have defects such as PSSM and MH.

Reference

Cole, F. L., Mellor, D. J., Hodgson, D. R. & Reid, S. W. J. 2004 Prevalence and demographic characteristics of exertional rhabdomyolysis in horses in Australia. *The Veterinary Record 155,* 625-630.

De La Corte, F. D., Valberg, S. J., MacLeay, J. M. & Mickelson, J. R. 2002 Developmental Onset of Polysaccharide Storage Myopathy in 4 Quarter Horse Foals. *Journal of Internal Medicine 16,* 581-587.

Firshman, A. M., Valberg, S. J., Bender, J. B., & Finno, C. J. 2003 Epidemiologic characteristics and management of polysaccharide storage myopathy in Quarter Horses. *American Journal of Veterinary Research 64,* 1319-1327.

Geor, R. J. 2005 Role of dietary energy source in the expression of chronic exertional myopathies in horses. *Journal of animal science 83,* E32-36.

Jones, W. E. 2003 Nutritional Support for Rhabdomyolysis. *Journal of Equine Veterinary Science 23,* 325-326.

Lentz, L. R., Valberg S. J., Balong, E. M., Mickelson, J. R. & Gallant, E. M. 1999 Abnormal regulation of muscle contraction in horses with recurrent exertional rhabdomyolysis. *American Journal of Veterinary Research 60,* 992-999.

McKenzie, E. C., Valberg, S. J., Godden, S. M., Pagan, J. D., MacLeay, J. M., Geor, R. J. & Carlson, G. P. 2003 Effect of Dietary Starch, Fat and Bicarbonate Content on Exercise Responses and Serum Creatine Kinase Activity in Equine Recurrent Exertional Rhabdomyolysis. *Journal of Veterinary Iinternal Medicine 17,* 693-701.

Nyblom. H., Bergren, U., Balldin, J. & Olsson, R. 2004 High AST/ALT ratio may indicate advanced alcoholic liver disease rather than heavy drinking. *Alcohol & Alcoholism 39,* 336–339.

Pesch, S., Bermann, M. & Bostedt, H. 2006 determination of some enzymes and macro- and microelements in stallions seminal plasma and their correlations to semen quality. *Theriogenology 66,* 307-313.

Peterson, C. April 2008. Personal communications. Leg. Vet., Svedala Veterinär Praktik.

Ribeiro, W. P., Valberg, S. J., Pagan, J. D. & Essen Gustavsson, B. 2004 The Effect of Varying Dietary Starch and Fat Content on Serum Creatine Kinase Activity and Substrate Availability in Equine Polysaccharide Storage Myopathy. *Journal of Veterinary Internal Medicine 17,* 887-894.

Sjaastad, Ø. V., Hove, K. & Sand, O. 2004. *Physiology of Domestic Animals,* 266-267. Oslo, Scandinavian Veterinary Press.

Skenderi, K., Kavouras, S., Anastasiou, C., Yiannakouris, N. & Matalas AL. 2006 Exertional Rhabdomyolysis during a 246-km continuous running race. *Medicine & Science in sports and exercise 38,* 1054-1057.

Valberg, S. J., Mickelson , J. R., Gallant, E. M., MacLeay, J. M., Lentz, L. & De La Corte, F. 1999 Tying-Up in Quarter Horses and Thoroughbreds: Separate Diseases with Common Clinical Signs. *Annual Convention of the American Association of Equine Practitioners 45,* 311-313.

Valentine, B. A., McDonough, S. P., Chang, Y.-F. & Vonderchek, A. J. 2000 Polysaccharide Storage Myopathy in Morgan, Arabian and Standardbred Related Horses and Welsh-cross Ponies. *Veterinary Pathology 37,* 193-196.

Valentine, B. A., Van Saun, R. J., Thompson, K. N. & Hintz, H. F. 2001 Role of dietary carbohydrate and fat in horses with equine polysaccharide storage myopathy. *American Veterinary Medical Association 219,* 1537-1544.

Valentine, B. A. & Cooper, B. J. 2005 Incidence of Polysaccharide Storage Myopathy: Necropsy Study of 225 Horses. *Veterinary Pathology 42,* 823-827.

Valentine, B. A. & Löhr, C.V. 2007 Myonecrosis in three horses with colic: evidence for endotoxic injury. *The veterinary Record 161,* 786-789.

Zentek J. 1991 Myopathies in a riding horse stable. *Tierarztliche Praxis 19,* 167-169.