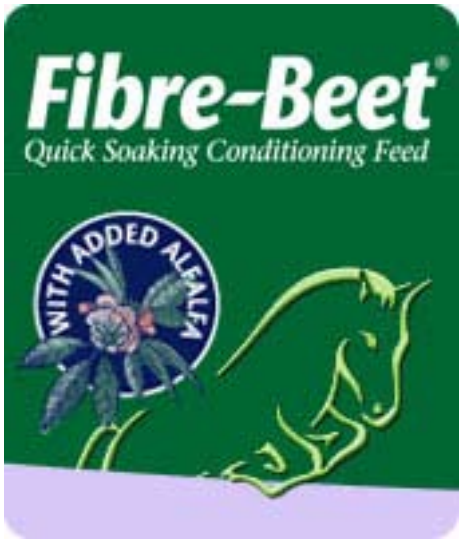
Untying a Tied Up Horse



Tying Up, Monday Morning Sickness, gait abnormalities, back pains, colic symptoms, stiffness are all familiar terms to the worried horse owner when their horse is in distress either during or after exercise.

What may be surprising is that these symptoms may well be different aspects of the same condition: exertional rhabdomyolysis. However this condition can be caused or exacerbated by a number of biochemical or physiological phenomena generally called myopathies. Simply this means damage to muscle, and we seem to be going around in circles.

There are three distinct types of pain associated with exercising muscles, pain during or immediately after exercise, pain delayed for 1 or two days and cramp. Of the three the delayed pain probably describes the majority of conditions in the opening statement and is possibly the most worrying as it can be accompanied by serious muscle damage.

However there is a common factor and this is the biochemistry of muscular activity and recovery.

Briefly muscle strands consist of two types of peptides, actin and myosin, that combine to form actinomyosin, a reaction mediated by calcium. Through a series of complex mechanisms the reaction slides the actin over the myosin, contracting the muscle bundle.

This mechanism requires energy, in the form of ATP, which is the principle energy release from glucose metabolism. During the contraction ATP is reduced to ADP. This ADP can be re-phosphorylated to ATP by the action of creatine kinase.

Another important enzyme is involved in the energy transfer during muscle activity; Lactic dehydrogenase. When a horse is undergoing hard exercise there comes a point when insufficient oxygen reaches the muscles. Glucose breakdown, through glycolysis and the Krebs cycle, requires oxygen for complete metabolism. When oxygen is limiting then the process stops before the Krebs cycle and the glucose metabolite pyruvic acid is converted to lactic acid. Although the lactic acid is removed to the liver and reconverted to pyruvic acid, this recovery period takes time. The conversion of pyruvate to lactate in the muscle, and the reverse in the liver, is mediated by the enzyme lactic dehydrogenase.

Although interesting in itself, to a biochemist, the importance of lactic acid, lactic dehydrogenase and creatine kinase are to act as indicators of what is happening during muscular dysfunction.

When glucose is metabolised optimally it generates 38 units of ATP, which then are used, amongst other things for muscle contraction. When glucose is converted to lactic acid during oxygen shortage then only 8 units are produced. This manifests itself by causing muscular activity to slow and, at the same time, the build up of lactic acid causes the muscle to ache.

Continuing activity requires an additional source of ATP, and this is taken from the creatine phosphate pool, converting ADP to ATP with creatine kinase. Levels of creatine kinase are therefore elevated to maintain the production of ATP.

Meanwhile the liver is taking lactic acid and regenerating pyruvic acid and as the supply of oxygen to the muscle resumes – either by improved breathing, or a reduction in exercise and a slowing of the metabolism of glucose, the supply of ATP stabilizes and creatine kinase levels drop.

The cause of exertional rhabdomyolysis is not fully known. However it is accompanied by elevated levels of creatine kinase and lactic dehydrogenase. Lactic acid levels do not necessarily rise and therefore the implication is that the body is trying to produce greater amounts of ATP. This would drive muscle contraction, which has been described as a ratchet mechanism. High levels of ATP would tend to inhibit the ATP in actinomyosin from breaking down, releasing the link and relaxing the muscle fibre. Therefore high levels of creatine kinase would indicate contraction without relaxation – Tying Up?

Add to this a condition called polysaccharide storage myopathies – PSSM – where there is an abnormal accumulation of glycogen. There is reduced glucose for activity and reduced ATP. Creatine kinase rises and tying up can follow.

Even with normal conditions, if over exercising, there will be a build up of lactic acid and an accompanying rise

of creatine kinase, and lactic dehydrogenase.

All these conditions are different but they have a common factor and this is the breakdown of glucose to generate energy for muscular activity. Where the breakdown is incomplete or disabled through myopathic condition, energy is sourced elsewhere. In the short term this appears to be from creatine phosphate and would also appear, in some circumstances to over compensate, resulting in muscular damage.

The horse is not a natural glucose user. It can generate sufficient glucose for essential metabolism from the propionate produced by fibre fermentation. In the wild it is not required to exercise heavily and so tying up would not be a problem. But what can be done for the modern horse that ties up?

Independent research has shown that substituting starch with fibre and fat can reduce the incidence of tying up. The end products of fibre fermentation (VFA) can enter the Krebs Cycle, bypassing lactic acid production and generate about 75% of the ATP that can be derived from glucose. Long chain fatty acids undergo oxidation, releasing pairs of carbon atoms which are converted to acetyl coA and this chemical can also enter Krebs Cycle.

By doing this the requirement for glucose to generate ATP is greatly reduced. By generating in the region of 15 ATP's per cycle, Krebs Cycle – generated by fibre and fat – can replace glucose metabolism for muscle contraction.

Glucose does have an important role to play in the biochemistry and physiology of any animal. It fuels active transport across the gut wall and is essential for brain function. Glucose should not be eradicated from a horse's diet. However by relying more on fibre/fat will reduce incidences of tying up in sensitive horses.

This can cause inconvenience. Providing a good quality compound feed, balanced against forage can provide the energy needed for all activities. If the starch content of the compound will reduce the energy level, and there is only so much fat that can be added to replace it. So the energy level of the whole ration can drop.

However, incorporating a superfibre will help. Speedi-Beet (and Fibre-Beet, containing Speedi-Beet) has undergone a physical process that markedly increases its energy value. Micronization of beet allows more of the fibre faction to be fermented by the hindgut bacteria and therefore provides more VFA per unit than ordinary forage. More VFA enter the Krebs Cycle and more ATP is generated. In addition scientific work has shown that beet fibre has a unique ability to improve the fermentability of other fibre sources, presumably by providing a substrate that modifies the hindgut microbial population.

Incorporating Speedi-Beet or Fibre-Beet into the ration of a horse liable to tying up allows the reduction in levels of high starch feeds without reducing the energy level of the daily ration. For those horses with some degree of exertional rhabdomyolysis, PSSM or simply those that often undergo anaerobic glucose metabolism (hard exercise over short periods), then there is an alternative to high starch diets.

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