GLYCOGEN DEPLETION AND REPLETION IN THE HORSE – POSSIBLE LIMITING FACTOR IN PERFORMANCE (REVIEW)

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Introduction

The importance of glycogen as a substrate and its role in both performance and recovery has been extensively studied in human athletes. Glycogen’s key role is as a substrate for muscle. Contraction of muscle involves the conversion of chemical energy into mechanical work, with adenosine triphosphate (ATP), adenosine diphosphate (ADP), inorganic phosphate (P_i), magnesium (Mg^{2+}) and hydrogen (H^+) being directly involved in this process. As ATP supply within the muscle is limited, the energy for the rephosphorylation of ATP comes from creatine phosphate (PCr) and substrate breakdown (carbohydrate and fat). However, the rate of ATP production from fat oxidation is slow and limits its usage to low intensity activities (Sahlin, 1986). As the intensity increases above 30% to 50% VO_{2max}, the importance of a contribution of carbohydrates increases with the contribution of ATP from glycolysis and PCr increasing at intensities above 70% to 80% VO_{2max}. Carbohydrates for muscle energy supply can be provided both endogenously (within the muscle) or exogenously (liver and blood). The importance of the initial muscle glycogen concentration in endurance performance in humans has been reported by Bergström et al. (1967), Hargreaves et al. (1984) and Coyle (1991) and for the horse by studies of endurance rides (Snow et al., 1981; Hodgson et al., 1983), road and track components of three-day event competitions (Hodgson et al., 1984) and during a 4-hour slow trot (Lindholm, 1979).

In contrast to the findings supporting the importance of the muscle’s initial glycogen concentration on endurance performance, the same support has not been coming forth for muscle glycogen and sprint performance in human studies (Costill et al., 1981; Symons and Jacobs, 1989) or high intensity exercise in horses (Topliff et al., 1983; Topliff et al., 1985; Davie, 1996). The muscle’s initial glycogen concentration seems to be more important as the time period of the exercise increases with the short term high intensity exercise not so dependent on the muscle glycogen concentrations.
Glycogen Use During Exercise

The pattern of glycogen usage and selective fiber depletion in humans with varying intensities of exercise (Essén and Henriksson, 1974) has also been demonstrated for the horse (Essén et al., 1984). In low intensity activities (endurance rides of 100 km), the ST fibers display the highest degree of depletion with FT fibers showing moderate levels of depletion. This selective glycogen depletion was also reported by Davie (1996) in which horses exercising at intensities of 60% \(\text{VO}_{2\text{max}}\) for 30 min resulted in some glycogen depletion in all fiber types, with most depletion evident in the ST and FTH fibers. Essén and Henriksson (1974) reported that during a 2-hour exercise period, the glycogen concentration in FT fibers had decreased by 29% whereas the glycogen concentration of ST fibers had decreased by 86% of their resting value. Rates of glycogen utilization during low intensity exercise have been reported at 4.1 mmol/kg (dwt) (Hodgson, 1984). For high intensity exercise, Lindholm (1974) reported that when trotting speed was increased from approximately 5 m/s to 12.5 m/s the rate of glycogen usage increased from 0.3 to 14 mmol/kg/min. This rate of glycogen usage is similar to that reported by Hodgson (1984) of 15.4 mmol/kg/min during a graded exercise test and that of Snow and Harris (1991) in which they found similar glycogen depletion rates following a 1000 m and 1600 m gallop, and Snow et al. (1985) following four 620 m gallops. However, these rates are much lower than those reported by Harris et al. (1987) of 160 and 64 mmol/kg/min (dwt) during an 800 m and 1000 m gallop.

Fatigue

Exercise requires an integration of many systems, each containing varied elements, and any factor that upsets this integration could cause fatigue (Brooks and Fahey, 1985). The cause of this inability of muscle to maintain a specific level of contraction could lie in either the central nervous system, the final motor neuron, the neuromuscular junction or the muscle (Åstrand and Rodahl, 1970; Saltin and Karlsson, 1971; Brooks and Fahey, 1985; Sahlin, 1986; Enoka and Stuart, 1992).

One problem in trying to identify the cause of fatigue is that it is a multidimensional phenomenon and can vary in accordance with the activity itself, the training and physiological status of the individual and the environmental conditions (Brooks and Fahey, 1985). Fatigue may take place at a single site, in the case of a maximal lift, or it may involve many sites when there is depletion of energy supplies, accumulation of waste products, changes in pH, depletion of nervous system transmitters and dehydration (Kraemer, 1983).

The onset of fatigue is most often associated with either the accumulation of metabolic by-products such as hydrogen and inorganic phosphate or a decline in muscle glycogen concentration. In an endeavor to improve endurance performance, athletes have attempted to increase muscle glycogen storage capacity 2 to 3 times greater than normal. The issue of glycogen loading, as it applies to
humans, would seem to have little practical application for the racing horse based on the degree of depletion that occurs during such events (Harris et al., 1987; Snow and Harris, 1991). Further, Topliff et al. (1983 and 1985) reported no improvement in performance with increases in muscle glycogen concentration of up to 36% above resting concentrations.

The Effects of Muscle Glycogen Concentration on Low Intensity Exercise Performance

The onset of fatigue in human endurance events has been related to muscle glycogen depletion (Bergström et al., 1967; Hargreaves et al., 1984; Coyle, 1991). Bergström et al. (1967) found a strong correlation between the initial muscle glycogen concentration and work time during endurance exercise. At exercise intensities of between 70% to 80% \( \dot{V}O_2 \text{max} \), exhaustion coincided with the muscle’s glycogen stores being depleted (Saltin and Karlsson, 1971).

Topliff et al. (1983) reported that in horses the onset of fatigue during a run at 3.0 m/s on a treadmill was earlier when the muscle glycogen concentration was reduced prior to the run. In endurance rides of 160 km, muscle glycogen depletion of more than 70% of ST fibers and substantial depletion of FT have been reported in horses (Hodgson et al., 1983). In the road and track components of three day event competitions, mean decreases in muscle glycogen concentration of 306 mmol/kg (dwt) with a mean rate of utilization of 4.1 mmol/kg (dwt) have been reported (Hodgson et al., 1984).

The effects of a reduced skeletal muscle glycogen concentration on physiological responses to low intensity treadmill exercise were examined by Davie (1996). Reductions in skeletal muscle glycogen concentrations of up to 29% had no significant effect on \( \dot{V}O_2 \), heart rate and temperature during a 30-min treadmill run at 60% \( \dot{V}O_2 \text{max} \). A decreased glycogen concentration did not affect the rate of glycolysis, confirming the findings of Topliff et al. (1983). In 7 of the 18 endurance runs, horses were unable to complete the 30 min of exercise, even though substantial quantities of muscle glycogen were still available, suggesting that muscle glycogen concentration was not the key contributing factor to fatigue. The horses’ inability to complete the treadmill run also was not associated with lactate accumulation. Other factors that may have contributed to the fatigue are accumulation of ammonia or increased temperature (Greenhaff et al., 1991; McConaghy et al., 1995). It is interesting to note that in some human studies, similar results have been reported. Costill et al. (1971), Gollnick et al. (1973) and Symons and Jacobs (1989) reported that for both prolonged and short exhaustive runs, glycogen depletion was the unlikely cause of fatigue. However, Gollnick et al. (1973) stated that even though the total muscle glycogen concentration had decreased by 63%, the loss of glycogen from the FT fibers may have been sufficient to result in the inability of these fibers to function adequately. In contrast, Bergström et al. (1967) found a good correlation between the initial muscle glycogen concentration and exercise time.
The Effects of Muscle Glycogen Concentration on High Intensity Exercise Performance

In both humans and horses, the muscle’s initial glycogen concentration appears to be unimportant for performance of high intensity exercise (Costill et al., 1981; Symons and Jacobs, 1989; Davie, 1996). Further, the provision of a carbohydrate supplement prior to high intensity exercise in humans also has been shown not to be beneficial. Glycogen supercompensation, which is used for performance enhancement in prolonged exercise, does not enhance high intensity exercise performance in humans (Housh et al., 1990; Madsen et al., 1990).

In contrast to human studies, Topliff et al. (1985) reported that a decrease in muscle glycogen concentration in the horse does affect its capacity for anaerobic work. This is in contrast to the work by Davie (1996) who failed to support Topliff’s findings. In a study in which horses ran on a treadmill to exhaustion at 115% VO$_{2\text{max}}$, 5 hours after a glycogen depletion run (high intensity exercise), there was no significant difference between horses performing with either a decreased muscle glycogen concentration or normal resting concentrations in reference to oxygen uptake or plasma lactate concentrations. Muscle glycogen concentration in the horse was reduced by 22% without having a significant effect on physical performance.

The lack of an effect of a reduced muscle glycogen concentration on lactate concentration during exercise is in contrast to that reported for humans. Studies in humans have shown that lactate production is reduced with a decreased muscle glycogen concentration. The basis of a reduced lactate formation with a low initial muscle glycogen concentration is the relationship between energy demand and by-product removal. If the glycogen concentrations are low to begin with, then the available substrate for glycolysis is reduced, leading to a reduced rate of glycolysis and reduced lactate production. Reductions in muscle glycogen concentration of greater than 22% may be required in horses before an effect on lactate accumulation are observed.

The rate of change of the VO$_2$ at the commencement of high intensity exercise in this study was also not affected by the initial glycogen concentrations. There was no significant difference between horses with reduced versus normal muscle glycogen concentrations for time to peak oxygen uptake, with all horses reaching peak oxygen uptake levels between 45 and 60 seconds. This time to peak VO$_{2\text{max}}$ supports the findings of Bellenger et al. (1994) who reported that horses reached 90% to 95% of mean steady state VO$_2$ by 45 seconds. The VO$_2$ responses are in agreement with responses reported for humans. Bergström et al. (1967) reported similar oxygen uptake responses during exercise regardless of the differing initial glycogen concentrations. However, Widrick et al. (1993) reported that in subjects performing time trials of 120-min duration, under conditions of varying initial muscle glycogen concentrations and carbohydrate feeding throughout the trials, in the first 71% of each trial the VO$_2$ was similar for all conditions. In the low glycogen trial VO$_2$ was lower at the end of the exercise period.

Key factors causing fatigue in humans parallel factors causing fatigue in the horse. In both human and horse endurance events, heat stress and substrate
availability have been indicated as key factors in fatigue. In the horse substantial depletion of muscle glycogen has been reported during endurance rides, supporting the concept that substrate availability may play a key role in fatigue. Miller and Lawrence (1986) and Snow and Harris (1991) argued that the changes in muscle lactate and pH are also unlikely to be the cause of fatigue in such events. Even though some metabolic acidosis occurs, the changes are unlikely to be of sufficient magnitude to induce fatigue. Heat stress, however, has been shown to play a role in reduced performance in such events (McConaghy et al., 1995).

In sprinting events such as Thoroughbred racing, the onset of fatigue is unlikely to be muscle glycogen related. In such events the total time of performance is from 57 s for 1000 m to 3 min 20 s for 3200 m. Fatigue in such events could be either central or peripheral based with the most likely factors being changes in the chemistry of the muscle cell. Lactate concentrations as high as 31 mmol/L and muscle pH of 7.0 have been reported following a 1000 m gallop (Harris et al., 1987). The accumulations of lactate and resultant fall in cell pH have been demonstrated as factors affecting both energy production and excitation contraction in the muscle.

Muscle Glycogen Resynthesis Following Exercise

The rate of muscle glycogen resynthesis following exercise is affected by factors such as the type of fibers depleted (Terjung et al., 1974), glucose dose administered after exercise (Blom et al., 1986; Blom et al., 1987), the timing of the administration of the supplement after exercise (Ivy et al., 1988), the activity state of glycogen synthetase and the initial glycogen concentration (Danforth, 1965; Larner et al., 1967; Bergström et al., 1972; Kochan et al., 1979). The major limiting factor in glycogen synthesis appears to be the muscle’s ability to synthesize glycogen, rather than the availability of intracellular substrate (Fell et al., 1982).

In humans, rates of muscle glycogen resynthesis of 23 mmol/kg/h (dwt) during the first 2 hours of recovery, following the provision of 3.0 g/kg of glucose (Ivy et al., 1988), 30.8 mmol/kg/h (dwt) following the ingestion of 2 g/kg of a 25% carbohydrate solution (Ivy et al., 1988) and 23 mmol/kg/h (dwt) with the ingestion of 0.7 g/kg of glucose every 2 hours for 8 hours (Blom et al., 1987) have been reported.

In horses, muscle glycogen resynthesis rates of 12.5 mmol/kg/h (dwt) for the first 8 hours following a high carbohydrate diet plus infusion (Snow et al., 1987), 5.6 mmol/kg/h (dwt) and 7.8 mmol/kg/h (dwt) for the 20-24 hour period following a 160 km endurance ride and for the first 4 hours immediately following a treadmill exercise test (Hodgson, 1984) and 19.8 mmol/kg/h for the first 6 hours and 14.6 mmol/kg/h and 7.1 mmol/kg/h for the first 12 and 24 hours following exercise (Davie, 1996) have been reported.
Effects of Fiber Type on Muscle Glycogen Resynthesis

The rate of muscle glycogen resynthesis depends on the type of muscle fiber. Conlee et al. (1978) showed that fast twitch red fibers, which have the highest total glycogen synthetase I activity, displayed the most rapid rate of glycogen synthesis, while fast twitch white fibers, which had the slowest rate of glycogen synthesis, had the lowest total glycogen synthetase I activity. Slow twitch red muscle which had an intermediate total glycogen synthetase activity, had an intermediate rate of glycogen synthesis. This difference in rate of resynthesis between fiber types is based on differences in the enzymatic capacity of the different muscle fiber types to convert glucose to glycogen.

Effects of Diet on Muscle Glycogen Resynthesis

Davie (1996) illustrated that the administration of 3.0 g/kg of glucose polymer, as either a single or split dose, after exercise did not have a significant effect on the rate of muscle glycogen resynthesis in the 24 hours after exercise. However, blood glucose and insulin concentrations were higher in treated horses than in controls. This suggests that differences in the blood glucose concentrations were not sufficiently different to affect glycogen resynthesis, or that blood glucose per se is not an important factor affecting the rate of muscle glycogen resynthesis in the horse, or that the contribution of glucose, via hepatic glycogenolysis and gluconeogenesis, was adequate to maintain a sufficient level of plasma glucose for glycogen resynthesis in the control group.

In a further study investigating the effect of post-exercise administration of dextrose (d-(+)-glucose) at a rate of 6 g/kg (bwt) intravenously as a 20% solution at a mean infusion rate of 1.67±0.05 l/h for 8 hours, it was reported that there was a significant increase in resynthesis rates. The rates reported of 19.8 mmol/kg/hr are much higher than those reported previously after a routine hay/grain diet in the post-exercise period (Hodgson, 1984) or when the additional infusion of 0.45 kg glucose over 6 hours was added to a high carbohydrate diet (Snow et al., 1987).

Based on the similar rates of resynthesis between the control group in the study by Davie (1996) and those reported by Hodgson (1984), it is suggested that even when feed is withheld, the capacity for glycogen resynthesis is similar to that found during normal feeding in the first 6 hours after exercise. For this to occur, gluconeogenesis must be adequate to maintain a plasma glucose concentration sufficient for glycogen resynthesis.

A glucose dose greater than 3 g/kg (bwt) administered intravenously can have an effect on glycogen replenishment rates. This finding may have potential practical implications for horses required to compete on successive days in endurance events. The provision of glucose intravenously overcomes the potential problems of gastrointestinal disturbances and laminitis which can be associated with oral glucose administration.
Conclusion

For events lasting longer than one hour, the general consensus is that initial muscle glycogen concentration plays a significant role in performance. There is no evidence that equine skeletal muscle glycogen concentration is increased by provision of extra dietary carbohydrate during recovery periods after exercise.

References

Glycogen Depletion and Repletion


Lindholm, A. 1974. “Glycogen depletion pattern and the biochemical response to


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