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Effects of glycogen depletion on high intensity exercise performance and glycogen utilisation rates

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Summary

The aim of this study was to examine the effects of a decreased muscle glycogen concentration on metabolic and physiological responses to high intensity exercise. Six Thoroughbred geldings of age 5.8±0.5 years (mean±SEM) and body weight of 487±12 kg (mean±SEM) were randomly allocated to two treatments or control in a 3x3 Latin square repeated design. The experiment involved horses completing one of two different glycogen depletion runs, aimed at depleting mainly type I (Protocol A) and type II (Protocol B), with a run to exhaustion (RE) at 115% $\dot{V}O_2$max 5 hours after the glycogen depletion run.

For muscle glycogen concentration, there was a significant difference between the three treatments at the $p<0.05$ level, with the post hoc test revealing that the difference was between the control and both protocols A and B. There was no significant difference in muscle glycogen concentrations between protocols A and B. Total glycogen utilized during RE runs were 188±23.2, 204±12.3 and 146±43.6 mmol.kg^{-1} for protocols A, B and Control respectively. Mean rates of glycogen usage were 1.9±0.34, 2.2±0.25 and 1.7±0.28 mmol.kg^{-1}s^{-1} for protocols A, B and Control respectively. There were no significant differences between protocols A, B and control. Mean run times were 103±9.2, 101±14.6 and 97±12.0 s for protocols A, B and Control respectively and there was no significant difference between these values.

We concluded that muscle glycogen concentration in the horse can be reduced by 22% without having a significant effect on physical work capacity during high intensity exercise.

Keywords: Anaerobic, Carbohydrate, Glycogen, Muscle, Exhaustion

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Einfluß eines Glykogenverlusts auf die Leistung bei Belastungen hoher Intensität und die Glykogenutilisationsraten

Ziel dieser Studie war es, den Einfluß einer verminderten Glykogenkonzentration im Skelettmuskel auf metabolische und physiologische Reaktionen, die einer Belastungen hoher Intensität folgen, zu erfassen. Als Versuchsdesign wurde ein 6x3 Lateinisches Quadrat mit zufälliger Reihenfolge der Behandlungsarten gewählt (6 Vollblüter x 3 Behandlungsarten). Die Behandlungsarten waren: A: Belastung über 45 Minuten bei 30% VO_2max und 15 Minuten bei 50% VO2max; B: 6 x 1 Minute bei 115% VO_2max; C: keine Belastung (=Kontrolle). Die Untersuchung sah vor, daß die Pferde zunächst einer der drei Behandlungsarten unterzogen wurden. Behandlungsart A hatte zum Ziel, hauptsächlich den Glykogengehalt der Typ I-Fasern zu depletieren, Behandlungsart B die Depletion der Typ II-Fasern. 5 Stunden nach der Behandlung galoppierten alle Pferde bis zur Erschöpfung, bei einer Geschwindigkeit, die 115% VO_2max entspricht.

Die Glykogenkonzentration in der Skelettmuskulatur war nach Behandlung zwischen den drei Gruppen signifikant verschieden ($p<0,05$). Der Unterschied bestand zwischen der Kontrollbehandlung und beiden Behandlungsarten A und B. Zwischen den beiden Behandlungsarten A und B bestanden keine Unterschiede in der Muskelglykogenkonzentration. Durch die Belastung bis zur Erschöpfung wurde die Glykogenmenge um 118±23,2; 204±12,3 und 146±43,6 mmol/kg bei den Behandlungsarten A, B und C verringert. Die mittleren Glykogenutilisationsraten unterschieden sich nicht. Sie betrugen 1,9±0,34; 2,2±0,25 und 1,7±0,28 mmol/kg/s für die Behandlungsarten A, B und C. Die mittlere Laufzeit bis zur Erschöpfung war 103±9,2; 101±14,6 und 97±12,0 Sekunden für A, B und C, sie war zwischen den Gruppen nicht unterschiedlich. Daraus schließen wir, daß beim Pferd die Glykogenkonzentration in der Skelettmuskulatur um 22% verringert werden kann, ohne daß die Leistungsfähigkeit während Belastungen hoher Intensität signifikant beeinflußt wird.

Schlüsselwörter: Anaerob, Kohlenhydrate, Glykogen, Muskel, Erschöpfung

Introduction

The horse, in comparison to humans, has a high skeletal muscle glycogen concentration (*Lindholm* et al., 1974). The importance of muscle glycogen and blood glucose for energy supply during prolonged exercise in humans has been highlighted by the work of *Bergström* et al. (1967); *Ahlborg* et al. (1967) and *Ivy* et al. (1983). However, the muscle's initial glycogen concentration appears to be unimportant for performance of high intensity exercise (*Costill* et al., 1971; *Symons* and *Jacobs*, 1989). Further, the provision of a carbohydrate supplement prior to high intensity exercise, also has been shown not to be beneficial (*Snyder* et al., 1993), whereas the provision of a carbohydrate supplement during 4 hours of cycling, followed by a sprint ride to exhaustion at 100% $\dot{V}O_2$ max, resulted in enhanced high intensity performance capacity (*Hargreaves* et al., 1984). Glycogen supercompensation, which is used for performance enhancement in prolonged exercise, also has been shown not to 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. In their study, they utilized the distance the horse could drag a sled, multiplied by the weight of the horse plus sled, as a measure of relative work. However, no studies

have examined the effects of glycogen depletion on physiological responses to high intensity exercise.
The aim of this study was to examine the effects of a decreased muscle glycogen concentration on metabolic and physiological responses to high intensity exercise. The results of this investigation will assist in further understanding the role of muscle glycogen concentration in high intensity exercise, as well as further development of nutritional strategies before race day.

Materials and methods

Six Thoroughbred geldings of age 5.8±0.5 years (mean±SEM) and body weight of 487±12 kg(mean±SEM) were randomly allocated to two treatments or control in a 3x3 latin square repeated design. Testing was carried out over consecutive weeks, with all horses being tested at the same time and on the same day each week. All horses had been maintained on a constant diet; 2 kg pellets (*Coprice, Leeton*, Australia), 200 g lucerne and 800 g oaten chaff as morning and night feeds, with 2.5 kg lucerne hay at lunch, for the 10 weeks of training prior to and throughout the study. The experiment involved horses completing one of two different glycogen depletion runs, with a run to exhaustion (RE) at 115% $\dot{V}O_2$max 5 hours after the glycogen depletion run. The run to exhaustion was used as a measure of physical work capacity.
On the day of testing feed was withheld for a minimum of 10 hours before commencement of experiments and withheld during the 5 hours prior to RE. Horses were allowed free access to water during the 5 hour period between glycogen depletion run and RE.
Glycogen depletion and RE intensities for each horse were determined from sub-maximal and maximal treadmill tests conducted the week prior to the experiment. For all testing the treadmill was set at 10% slope as for training. Speeds for RE were obtained from a regression equation of speed and oxygen uptake values from the sub-maximal and max test.

Glycogen Depletion Runs
Two protocols were used to reduce the concentration of glycogen in skeletal muscle. All glycogen depletion runs were conducted between 7 am and 10 am, with testing carried out on consecutive weeks.

Protocol A: Horses ran on the treadmill set at 10% slope for 45 min at 30% $\dot{V}O_2$max (3.5 m.s^{-1}), then for 15 min at 50% $\dot{V}O_2$max (5.7 m.s^{-1}). The aim of this protocol was to deplete mainly type I fibres:

Protocol B: Horses trotted at 3.2 m.s^{-1} for 1000 m, followed by six one min sprints at 115% $\dot{V}O_2$max (12.8 m.s^{-1}) , with five min of walking at 1.5 m.s^{-1} between each sprint. The aim of this protocol was to deplete mainly type II fibres:

Control: in which horses only completed the RE run.

Run to Exhaustion (RE) Run
The RE tests were conducted 5 hours after glycogen depletion runs. Horses warmed-up for 5 min at 50% $\dot{V}O_2$max then stood on the treadmill for 3 min, followed by 2 min walking at 1.5 m.s^{-1}. The treadmill was then accelerated to the speed that would produce 115% $\dot{V}O_2$max. The acceleration time and total time of the run were recorded. Run time was taken from the time the horses reached the required speed until they could not maintain pace with the speed of the treadmill.

Muscle Biopsy
Muscle biopsies were taken using the needle biopsy technique of *Bergström* (1962) as modified by *Lindholm* and *Piehl* (1974). Biopsies were taken from the m. gluteus medius at a depth of 8 cm at the same locations in each horse. Samples were taken immediately before the glycogen depletion and RE and within 20 s of the completion of the RE, for protocols A and B. For control, samples were taken immediately before RE and within 20 s of the completion of the RE . Muscle samples for biochemical analysis were immediately frozen in liquid nitrogen. Muscle glycogen concentration was determined on freeze dried muscle specimens according to the method of *Harris* and *Hultman* (1984) as modified by *Snow* et al. (1987).

Statistics

All results are expressed as mean±SEM. For glycogen the major effects of time, horse, week and treatment were analysed by MANOVA, with time as a repeated measures (within group) factor. Where F values were significant, a post hoc test of least significant difference was used. The level of statistical significance used was $p< 0.05$.

Results

Muscle glycogen concentrations for before depletion runs and before and after the RE, for the three treatments are shown in Table 1. There was a significant difference between the three treatments at the $p<0.05$ level, with the post hoc test revealing that the difference was between the control and both protocols A and B. There was no significant difference in muscle glycogen concentrations between protocols A and B.

Table 1: Muscle glycogen concentrations (mmol.kg^{-1}) (mean± SEM) of the m. gluteus medius, for glycogen depletion protocols A, B and C, before glycogen depletion runs, and before and after the run to exhaustion (RE) (N=6).

Protocol	Before Depletion Run	Before RE Run	After RE Run
A	591±19.5	517±20.1	329±16.2 [a]
B	647±14.6	502±17.0	297±18.0 [a]
Control	608±53.8	608±53.8	462±26.2 [b]

Note: Superscripts in the same column not sharing the same letter were significantly different $p<0.05$

Total glycogen utilized during the RE runs were 188±23.2, 204±12.3 and 146±43.6 mmol.kg^{-1} (dry.wt d.w) for protocols A, B and Control respectively. Mean rates of glycogen usage were 1.9±0.34, 2.2±0.25 and 1.7±0.28 mmol.kg^{-1}s^{-1} for protocols A, B and Control respectively. There were no significant differences between the treatments and control at the $p<0.05$ level.
Mean run times for the three protocols were 103±9.2, 101±14.6 and 97±12.0 s for protocols A, B and Control respectively and there was no significant difference between these values.

Discussion

The results of this study have shown that muscle glycogen concentration in the horse, can be reduced by 22% without having a significant effect on physical work capacity during high intensity exercise. These results are in contrast to the findings of *Topliff* et al. (1985), who reported a decrease in the relative work capacity of the horse when pre-exercise muscle glycogen concentration was reduced by

approximately 41%. The relative work capacity, represented the distance the horse was able to drag a sled multiplied by the weight of the horse and sled, with the task of dragging the sled considered to be an anaerobic activity. The relative work performed by each horse following a 28 day training phase, a 5 day glycogen depletion phase and a 3 day glycogen repletion phase were compared. They reported a significant difference in relative work performed between the glycogen depletion phase, the end of the 28 day training phase and the 3 day repletion phase. There was no difference however, between the end of training and the glycogen repletion phase. A concern with this study is whether the task of dragging the sled, was a reliable measure of high intensity activity and because of the nature of the activity, which involves slow muscle contraction, it is difficult to relate the results to a high velocity muscle contraction activity as sprinting. The reported lactate concentrations of approximately 13 mmol.L^{-1} at fatigue, would also indicate, based on reported lactate concentrations from both treadmill and field testing (*Bayly* et al., 1987; *Harris* et al., 1987) that the intensity of exercise was not maximal.

The mean rate of glycogen utilization of 1.9±0.55 mmol.$kg^{-1}s^{-1}$ in this study is similar to that reported for humans and Thoroughbred horses at maximum intensity (*Snow* et al., 1985; *Vøllested* et al., 1992) but higher than that reported for Standardbred horses (*Hodgson,* 1984). The similar rates of glycogen utilization between this study and those reported for testing, validates use of the treadmill as an ideal method for investigating factors affecting performance of high intensity exercise in race horses. The lack of a significant difference in glycogen utilization rates is in constrast to that reported for rats. *Richter* and *Galbo* (1986) reported that increased muscle glycogen concentrarions resulted in an increased breakdown of glycogen and release of lactate within the muscle cells.

As certain metabolic changes in muscle, such as increased lactate and creatine phosphate reduction (*Sahlin,* 1992), and increased ammonia concentrations (*Miller* and *Lawrence,* 1986) have been associated with fatigue in high intensity exercise, the measurement of these variables may have provided more insight into the physiological responses to exercise with reduced concentration of glycogen in skeletal muscle.

In conclusion the results of this study suggest that the rate of glycogen usage and the physiological and metabolic responses to high intensity exercise, may not be affected by a reduced initial muscle glycogen concentration of up to 22%.

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