

A diet rich in carbohydrate does not compensate for the dramatic delay of glycogen resynthesis caused by eccentric exercise and sore muscles: a ¹³C MRS study

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Introduction

Delayed onset muscle soreness (DOMS) is caused by exercise of great vigor and intensity, involving eccentric contractions. Manifestations of DOMS are muscle damage including inflammation, loss of muscle strength, swelling, release of muscle proteins in the blood, and reduced motor control. In addition, it has been reported from muscle biopsies that the rate of muscle glycogen resynthesis is reduced after eccentric exercise compared to concentric exercise [1]. In our study, we were interested in finding out if a diet rich in carbohydrate (CHO) was capable to compensate the delay in the glycogen resynthesis.

Methods

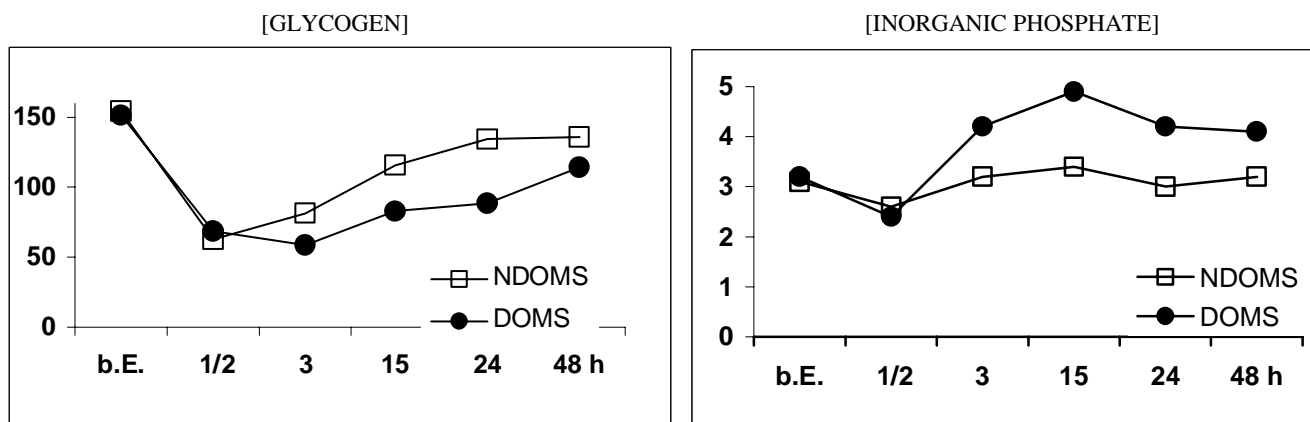
Experimental design: 20 well-trained, non-smoking men volunteered to participate in the study. One week before the experiment, each subject performed a Conconi test on a treadmill. On the test day, the subjects were allowed to eat and drink only the food prepared by us to ensure that every subject ingested at least 10 g CHO/kg-body-mass/day during recovery. Each subject performed several bouts of sprints in order to reduce glycogen stores in the fast twitch fibers. The 65 % velocity of the Conconi test was then used for a 1 hour tread-mill run, in order to reduce the glycogen stores in the slow twitch fibers. After this, the 12 subjects of the DOMS group performed a single-leg toe-raise exercise to induce muscle soreness. The subject lifted and lowered his body and an additional weight of 25 % of his body mass by flexion of the ankle joint. Each subject completed 10 bouts of 20 s toe-raise exercise (1 per sec.) followed by 40 s of rest.

Magnetic resonance spectroscopy: A 4.7 Tesla spectrometer (Varian) was used for the in vivo MRS measurements. Two concentric surface coils for ³¹P and for ¹³C were used to transmit and receive the signals. The coils were placed under the center of the right calf muscle. Small external reference samples of phenylphosphoric acid for ³¹P and of enriched formic acid for ¹³C were placed in the center of the concentric coils to optimize the rf-pulse power in order to achieve a precise 180° flip angle in the center of the RF coil. The Tr was 0.15 s and the partial saturation of the glycogen signal was corrected. 6000 FIDs were sampled. We fitted the data in the time- and in the frequency-domain. Glycogen quantification was achieved with a calibration phantom [2]: a 2-liter bottle filled with a 100 mmol/L glycogen solution buffered to a pH of 7.2.

Results

The left-hand figure shows the glycogen concentrations. Sprinting and running reduced glycogen by about 50 %. The concentration dropped further by 8 mmol/kg/h in the first 2 hours of recovery in the DOMS group but increased by 9 mmol/kg/h in the control group (NDOMS).

The right-hand figure depicts the concentrations of inorganic phosphate. Pi rose after exercise and remained significantly high for the DOMS but did not change markedly for the control group (NDOMS).



Discussion

Muscle glycogen concentration was equally lowered after running and sprinting irrespective of whether subjects rested or performed an additional eccentric exercise. Eccentric exercise of the DOMS group successfully induced muscle soreness. The carbohydrate-rich diet immediately consumed after exercise not only failed to prevent delayed glycogen resynthesis. Glycogen decreased even further over the first 2 hours of recovery in the DOMS subjects in contrast to the control group (NDOMS), where glycogen started to refill. This decrease in glycogen concentration after eccentric exercise is reported here for the first time and has not been seen in previous studies, which measured glycogen after 4 to 6 h of exercise. The additional glycogen loss during early recovery is especially disadvantageous, because the first 2 hours of exercise recovery are normally characterized by a rapid, insulin independent, glycogen resynthesis phase. Elevated Pi concentration persisted for at least 24 hours in the DOMS group. Increased Pi is an indicator of muscle cell damage. Thus, the delayed glycogen resynthesis is most likely an effect of muscle cell damage. In consideration of this fact, **it is probably impossible to eliminate the negative effect of muscle injury on the glycogen resynthesis after eccentric exercise, even with a large supply of carbohydrate during recovery.**

Ref.: 1. Komi PV and Viitasalo JT, Changes in motor unit activity Acta Physiol Scand, 100: 246-254, 1977.

2. Buchli R and Boesiger P, Comparison of methods for the determination..... Magn Reson Med, 30: 552-558, 1993.