Effects of maltodextrin on liver and muscle glycogen synthesis during short-term recovery and on post-recovery cycling performance

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Introduction:

Glycogen is central to muscular performance and is depleted with repeated contractions. Muscle glycogen is supplemented during exercise by increased hepatic glucose output. In recovery from exercise muscle and liver glycogen are repleted with carbohydrate intake. The rate of glycogen recovery in muscle is dependent upon carbohydrate supply and the actions of insulin, following an initial insulin independent phase. Co-ingestion of protein with carbohydrate may improve insulin efficacy through an additional protein stimulated insulin release. The aim of this study was to evaluate the effect of protein supplementation during the recovery from exercise upon muscle and liver glycogen recovery and the subsequent effects upon muscle performance.

Methods

Endurance-trained individuals (n = 7) visited the Newcastle Magnetic Resonance Centre on three occasions over a 3-week period. During the initial visit, subjects performed a continuous graded exercise test to volitional exhaustion to determine maximal oxygen uptake ($\dot{V}O_{2max}$). Approximately a week later subjects returned to the centre. Following an overnight fast, participants cycled for 45 minutes at 70% of their $\dot{V}O_{2max}$. Participants then performed 6 x 1min bouts at 120% $\dot{V}O_{2max}$ each followed by 2min recovery at 50% $\dot{V}O_{2max}$. Participants then cycled for 45min at 70% of their $\dot{V}O_{2max}$. Following exercise they were randomly supplied with either a recovery drink containing maltodextrin + galactose with or without protein. (Carbohydrate was supplied at 1g/kg body mass and were consumed at 30, 60, 90, 120, 150, 180 and 210 minutes. After the full 4.5hour recovery period participants cycled to exhaustion at 85% of their VO_{2MAX}. During exercise and in recovery blood samples were collected from an intravenous cannula and expired air samples collected to evaluate changes in substrate use during exercise. ^{1}H-decoupled ^{13}C spectra and volume-localised ^{1}H spectra were acquired to measure glycogen and lipid content in thigh muscle and liver immediately after the first exercise period, after the 240 recovery period, and immediately after the second cycle. Spectra were acquired on a 3T Achieva whole body scanner (Philips, Best, The Netherlands) equipped with a PulseTeq ^{13}C/^{1}H leg coil and a home-built ^{13}C/^{1}H liver coil (^{13}C coil diameters = 6cm and 12cm respectively). Glycogen content was determined from the magnitude of natural abundance C1-glycogen signal at 100.5 ppm, quantitation was performed by comparison of peak magnitudes to spectra from leg- and liver-shaped phantoms containing glycogen solutions of known concentrations. For ^{1}H spectroscopy data the magnitude of resonances originating from intramuscular triglyceride were compared to the magnitude of the water proton resonance from the same voxel. All data were analysed with jMRUI software.

Results and Discussion

Figure 1 shows a typical pre-exercise ^{13}C spectrum acquired from one of the athletes. The C1 resonance of glycogen can be clearly seen at 100.5 ppm (circled). Plasma glucose availability during recovery (AUC) was significantly higher without protein than with ($p < 0.01$). The change in muscle and liver glycogen during recovery and following the exercise trial to exhaustion is shown in Figure 2 and Figure 3 respectively. Although the maltodextrin + galactose recovery drinks produced a robust increase in muscle and liver glycogen during recovery ($p<0.01$), there was no effect of added protein on recovery rate ($p>0.05$). Liver glycogen recovery was associated with plasma glucose availability (AUC) during recovery (r = -0.906; $p = 0.005$) and substrate oxidation during the cycle to exhaustion (r = 0.821; $p = 0.024$). There was a sustained decrease in intramuscular lipid over the examinations. There was no effect of protein upon intramuscular lipid levels. Intramuscular lipid levels were negatively associated with plasma lactate levels (r = 0.-790; $p = 0.034$). These results demonstrate that the addition of protein to a matodextrin and galactose recovery drink does not enhance muscle or liver glycogen recovery. The strong relationship between liver glycogen and substrate oxidation during exercise suggests that optimisation of liver glycogen recovery may play a significant role muscle metabolism and muscular performance.

Acknowledgements and References

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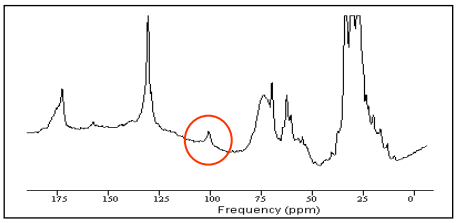

Figure 1 – Typical ^{13}C spectrum acquired from the thigh of a subject prior to exercise. The C1 peak of glycogen can be clearly seen at 100.5 ppm (circled).

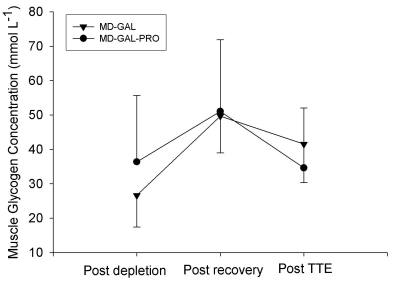

Figure 2 – Mean (±SD) muscle glycogen concentrations (n = 7) for the MD-GAL and MD-GAL-PRO treatment.

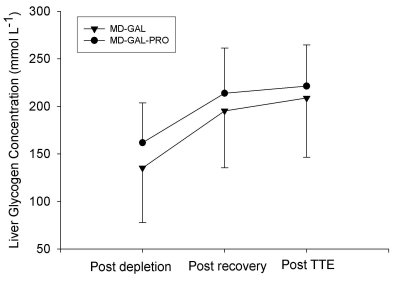

Figure 3 – Mean (±SD) liver glycogen concentrations (n = 7) for the MD-GAL and MD-GAL-PRO treatment.