REGULATION OF CARBOHYDRATE OXIDATION DURING EXERCISE AND HEAT STRESS

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Muscle glycogenolysis and carbohydrate oxidation are both increased by heat stress during prolonged exercise (Febbraio et al., 1994). Glycogen phosphorylase and pyruvate dehydrogenase, the key enzymes which regulate glycolytic flux and carbohydrate oxidation respectively, are maximally activated 1 min after the onset of moderate intensity exercise (Howlett et al., 1998). Thus, the aim of this study was to examine the effect of heat stress on muscle metabolism at the onset of moderate intensity exercise.

Six active men (26.0 ± 5.0 yrs, 75.0 ± 6.7 kg, VO\textsubscript{2} peak = 3.55 ± 0.39 l min\textsuperscript{-1}, mean ± SD) reported to the laboratory on two occasions in the morning after an overnight fast. Subjects positioned a rectal thermometer, a basal blood sample was obtained after a catheter was positioned in the antecubital space of one arm, and a resting muscle temperature was obtained from the vastus lateralis. After this time, subjects entered an environmental chamber set at either 20\textdegree C (CT) or 40\textdegree C (HT) (relative humidity 35% for both trials) and rested for 20 min, before a pre-exercise blood sample, rectal (T\textsubscript{rec}) and muscle (T\textsubscript{mus}) temperature measures were made and a muscle biopsy was obtained. Subjects then commenced cycling exercise for 20 min at a power output eliciting ~70% VO\textsubscript{2} peak. Muscle biopsies were obtained at 1 min and 5 min of exercise. Together with the pre-exercise sample, these were analysed for adenosine-5'-triphosphate (ATP), creatine (C), phosphocreatine (PCr), glucose-6-phosphate (G-6-P), pyruvate (pyr), lactate (mLa\textsuperscript{-}) and acetyl coA (ACoA). Muscle glycogen (Gly) was measured pre-exercise and at 5 min. In addition to those obtained at rest, blood samples were obtained at 5 min intervals throughout exercise for subsequent measurement of plasma adrenaline (Adr). T\textsubscript{mus} and T\textsubscript{rec} were also measured at 1, 5 and 20 min of exercise. Although T\textsubscript{mus} was similar at rest, 20 min of passive exposure to heat increased (P<0.05) T\textsubscript{mus} in HT compared with CT. This difference was maintained throughout exercise. Neither T\textsubscript{rec} nor Adr were different at rest, pre-exercise or at 1 and 5 min but T\textsubscript{rec} was higher (P<0.05) and Adr tended to be higher (P=0.051) after 20 min of exercise in HT compared with CT. In addition, neither ATP nor Pyr were affected by treatment or exercise. Although C, G-6-P, mLa\textsuperscript{-} and ACoA increased (P<0.05) and PCr and Gly decreased (P<0.05) when comparing concentrations at 1 and 5 min with those pre-exercise, no differences were observed when comparing HT with CT.

The results of the present study demonstrate that heat stress does not alter intramuscular metabolism at the onset of exercise. It is likely, therefore, that as exercise in the heat progresses and the difference in circulating adrenaline is augmented, the activation of the enzymes which regulate carbohydrate utilisation are increased resulting in enhanced glycogen use.


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