PHOSPHORYLATED HEXOSES IN HUMAN PLASMA INCREASE DURING EXERCISE

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Phosphorylated hexoses are thought to be 'locked' in the cell and normally cannot leave unless metabolized to nonphosphorylated compounds (Newsholme and Leech 1983). However, injury to muscle cells during exercise can result in cell membrane damage (Armstrong 1986). Such membrane damage provides the potential for 'leakage' of cell metabolites into the blood. Therefore we have measured the concentration of the hexose monophosphates, glucose 1-phosphate (G1P) and glucose 6-phosphate (G6P) in plasma before, during and after exercise.

Six subjects undertook an incremental bicycle VO_2 max exercise regime, starting at an exercise workload of 50 W, increasing 25 W every two minutes until volitional exhaustion (VO_2max = 44.96 ± 5.18 $ml.min^{-1}kg^{-1}$). Fingerprick blood samples were collected from the subjects just prior to the commencement of exercise, at one to three minute intervals during the exercise period (~18-23 min) and then every five to ten minutes for 60 minutes during recovery. Samples were centrifuged, deproteinised and the concentrations of G1P and G6P were determined using luminometric methods.

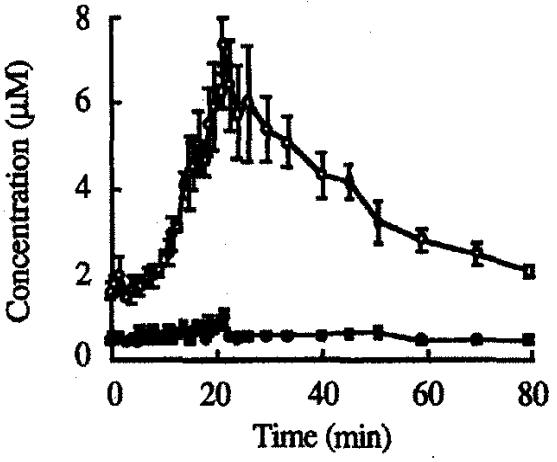

Fig 1. Plasma (mean ± S.E.) G6P (o) and G1P (●) in plasma before, during and after exercise.

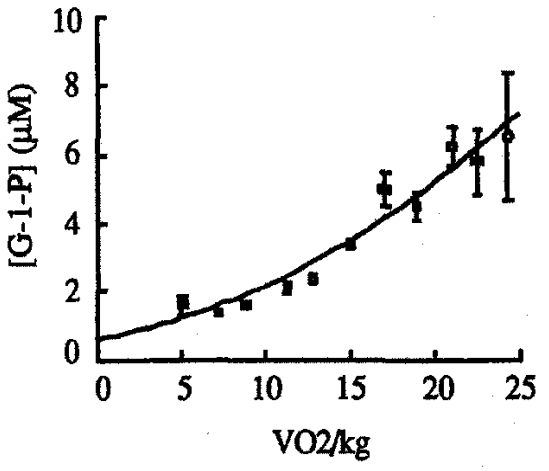

Fig 2. The change in G1P (o , mean±S.E.) versus the VO_2/kg body weight

The results show that G6P and G1P are normally present in plasma and that during exercise the concentration of these compounds increases by two and four fold, respectively (Fig. 1). The increase in G1P was closely correlated to VO_2/kg (r= 0.97; Fig 2.), and therefore glycogen utilisation. Immediately exercise ceased the concentration of G1P and G6P in the plasma decreased and the elimination rate for G1P was estimated to be 2.1 %/min.

We have shown for the first time that hexose monophosphates are normally present in the plasma. During muscle contraction when glycogen stores are utilised, it has been suggested that there is a rapid accumulation of G1P, and possibly G6P in the cell (Lee and Katz 1989). Thus the rapid increase in plasma G1P observed during exercise may be due to increased glycogenolysis resulting in an increased loss of G1P from the cell. If this is so the rapid disappearance after exercise may reflect the decreased utilisation of glycogen. Alternatively the increased G1P concentration could be explained by altered membrane function, whether it be cell lysis or an increase in the permeability of cell membranes. If this increase in plasma G1P is a result of altered membrane function of muscle cells, the concentration of G1P in the plasma may be a useful index for determining muscle cell damage and/or the rate of glycogen utilisation.

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