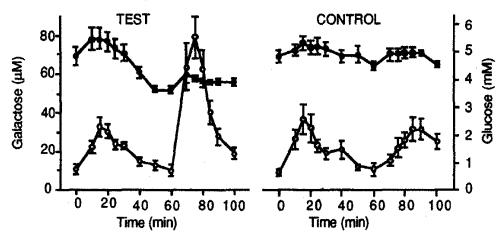
THE EFFECT OF MODERATE EXERCISE ON THE PLASMA RESPONSE TO A DOSE OF GALACTOSE

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The monosaccharides glucose and galactose, which form an important part of the diet, are metabolized in different ways. The liver metabolizes 90% of the galactose present in the portal blood, but glucose passes through the liver and is largely metabolized by the peripheral tissues, including skeletal muscle. However, by measuring the low concentrations of galactose remaining in the peripheral plasma, it is possible to monitor the clearance of galactose from the blood and to determine if galactose metabolism is altered under different physiological conditions, such as exercise.

Seven adult volunteers (6 males; 1 female) underwent a test protocol involving the ingestion of two doses of 5g galactose at 0 min (T1) and at 60 min (T2). Subjects were asked to exercise at 50% of their $\dot{V}O_2$ max, commencing at 40 min. Fingerprick blood samples were collected at 5-10 min intervals over a period of 100 min. Thus we were able to monitor the plasma response to the same amount of ingested galactose under resting (T1) and exercising (T2) conditions. In addition, each subject underwent a control protocol, which was identical to the test except that both the first (C1) and the second (C2) doses were carried out under resting conditions in order to determine the effect of two consecutive doses of galactose. Areas under the curves (AUC) for the galactose and glucose profiles (see figure) were calculated over a period of 40 min after ingestion of the dose.



Plasma galactose (o) and glucose (e) for test (n=7) and control (n=6) protocols (mean ± SEM). Galactose (5g) was ingested at both 0 and 60 min. Exercise started at 40 min for the test protocol.

The peak galactose concentration occurred 15 min after dose ingestion for both T1 and T2. Both the galactose peak and AUC were significantly higher after T2 than after T1 ($P \le 0.01$). The galactose AUC after T2 was 98% higher than that after T1. By 30 min after dose ingestion, the galactose concentrations for T1 and T2 were no longer significantly different (P > 0.05). There was no significant difference in peak or AUC between T1, C1 and C2. Both the peak glucose concentration and the AUC were significantly lower after T2 than after T1 ($P \le 0.01$) but there was no significant difference between T1, C1 and C2.

It is unlikely that the elevated peak in galactose was due to an increased rate of intestinal uptake, since glucose and galactose are absorbed by a common intestinal transport system and previous studies on the oral ingestion of glucose have shown that exercise has no effect on gastric emptying or its absorption from the intestine (Fordtran and Saltin 1967). Since the elevated plasma galactose which occurred after T2 reached resting levels within 15 min of the peak, the rate of clearance of galactose does not appear to be slower during exercise. There is evidence that peripheral tissues, although unable to metabolize galactose, may increase their uptake of galactose in response to higher insulin levels (Levine et al. 1950). Thus it is possible that under resting conditions, when plasma glucose and therefore circulating insulin levels were higher than during exercise, a greater amount of galactose was taken up by the peripheral tissues, decreasing the amount of galactose detectable in the peripheral blood.

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