# Effects of the glycemic index on the insulin-like growth factor system

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Increased intake of refined carbohydrates has been associated with secular increases in height, weight and growth in groups such as the Eskimo (1). We hypothesised that acute postprandial hyperinsulineamia following the consumption high GI foods may cause changes in the insulin-like growth factor system that favour accelerated growth. Insulin-like growth factor-1 (IGF-1) is an important stimulator of growth and metabolism, and insulin-like growth factor binding protein-1 (IGFBP-1) is suppressed by acute and chronic elevation in insulin (2).

Two groups of young, lean, healthy subjects, 10 Caucasians and 10 South East Asian, were studied. The mean (± SD) age and BMI were 24 ± 4 y and 21 ± 2 respectively. They fasted overnight and consumed a low and high GI meal (50 g carbohydrate portions of pearled barley or instant potato respectively) in random order on separate occasions. On a third occasion they fasted over the same period. Finger prick blood samples were taken at regular intervals over four hours and analysed for glucose, insulin, free IGF, total IGF and IGFBP-1-3.

In all twenty subjects, IFGBP-1 levels were significantly decreased by 4 h post consumption of the low GI food (–44 ± 17 ng/mL) compared with little change after the high GI food (0 ± 16 ng/mL). However, in Caucasians, there were significantly greater increases in IGFBP-3 4 h after consumption of the low GI compared with the high GI food (0.3 ± 0.1 vs 0.1 ± 0.1 μ/mL, $p < 0.05$). No significant differences were found in serum IGFBP-2, free IGF-1 or total IGF- 1 levels in response to the two foods.

We also noted interesting racial differences during the extended fast. In SE Asian subjects, mean fasting levels of free IGF-1 over the 4 h were significantly higher than in Caucasian subjects (0.9 ± 0.01 vs 0.7 ± 0.02 ng/mL). Correspondingly, mean IFGBP-1 levels were lowest in SE Asian subjects (40 ± 3 vs 96 ± 5 ng/mL, $p < 0.01$). Fasting glucose levels were higher in the SE Asian groups (5.4 ± 0.1 vs 5.1 ± 0.03 mM, respectively, $p < 0.01$).

These results provide equivocal support for the hypothesis that the ingestion of high GI foods leads to alteration in the IGF system that collectively favours increased growth. Changes in IGFBP-3 were remarkable and unexpected and may indicate increased free IGF-1 available in the tissues. Changes in IGFBP-1, however, were the opposite of those hypothesised, suggesting that a low GI food would promote higher free IGF-1 levels. Racial differences in the glucose metabolism and the IGF system during extended fasting may be relevant to the documented differences in the prevalence of type 2 diabetes.

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2. Busby WH, Snyder DK, Clemmons DR. Radioimmunoassay of a 26,000 dalton plasma insulin-like growth factor binding protein: control by nutritional variables. J Clin Endo Metab 1988; 67; 1225–1230.