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A comparison of the insulinotropic and enterogastric response to ingestion of an equivalent quantity of maltodextran and whey protein

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In the absence of carbohydrate, the insulinotropic effect of whey protein ingestion induces a rise in plasma insulin and lowering of blood glucose during the first 30 min after ingestion (Power *et al.* 2009) ⁽¹⁾. It was hypothesized that the insulinotropic action of whey protein may, in part, be mediated via the secretion of the enterogastric hormones, represented by glucagon-like peptide-1 (GLP-1).

The present paper reports on the post-prandial response of plasma glucose, insulin and GLP-1 to feeding maltodextran and an equivalent quantity of whey protein. With ethical approval and informed consent four young, healthy subjects ($n = 4$, age 25(2.8) y, BMI 21.8(1.1) kg/m²) undertook a randomized trial of two treatments, either a protein (WHEY 0.33 g/kg, 10% w/v water) or maltodextran (MALT; 0.33 g/kg, 10% w/v water), each treatment separated by 5 days. Subjects fasted overnight (10 h) prior to participation. Serial blood samples were withdrawn prior to and post ingestion for a period of 2 h and batch analysed for glucose, insulin and total GLP-1. Data are presented as mean (SEM; $n = 4$). Area under the curve for plasma insulin or total GLP-1 (AUC_{0–120}) was calculated by trapezoidal integration. Difference in the mean response was analysed by paired Student's t-test.

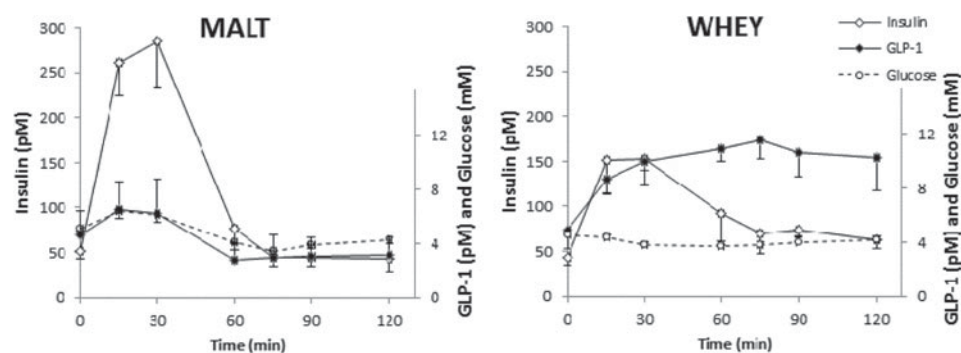


Figure 1. Mean plasma insulin, glucose and total GLP-1 for maltodextran (MALT) and whey protein concentrate (WHEY) during the post-prandial period. Values are mean (SEM; $n = 4$).

Following ingestion of the MALT, plasma insulin increased rapidly in the first 30 min and reached a maximal concentration (C_{max}) of 299(48) pM which directly correlated with the change in plasma glucose (Fig. 1). Ingestion of WHEY resulted in a C_{max} for plasma insulin of 182(23) pM within 30 min and was independent of a change in plasma glucose (Fig. 1). The differing insulin responses for MALT and WHEY were reflected in the AUC, 8506(2059) vs. 6512(1368) pM.2h ($P = 0.29$). In contrast, ingestion of MALT gave a transient increase in total GLP-1 in the first 30 min and a C_{max} of 7.4(3) pM but ingestion of WHEY gave a prolonged increase with a C_{max} of 11.4(2) pM at 75 min. Total GLP-1 AUC was 64(40) vs. 536(102) pM.2h ($P = 0.013$) for MALT and WHEY, respectively.

The enterogastric response, GLP-1, was greater following ingestion of an equivalent protein (WHEY) versus carbohydrate (MALT) load. This contrasts with previous reports that GLP-1 secretion is glucose dependent ⁽²⁾. There was a shift in the time course of maximal plasma insulin and GLP-1 response and this data does not support the hypothesis that GLP-1 directly mediates the insulinotropic action of protein. The magnitude of the protein mediated insulin response can be augmented by hydrolysis of the protein ⁽³⁾; therefore future studies are warranted to examine the effect of protein hydrolysis and those of peptide components upon GLP-1 secretion.

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