

THE EFFECT OF CO-INGESTION OF FAT ON METABOLIC RESPONSES
TO CARBOHYDRATE AND PROTEIN

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Much attention has been focussed recently on factors such as physical form and viscosity which affect postprandial glucose and insulin responses to different types of carbohydrate. In the Western diet carbohydrate is seldom eaten alone, but is consumed together with fat and/or protein. In an attempt to examine some of the interactions of these foods on postprandial metabolic responses, we measured the glucose, insulin and gastric inhibitory polypeptide (GIP) responses to carbohydrate (potato) or protein (low fat veal) in the presence and absence of fat (butter). Eight lean subjects (22 ± 2 years) consumed four test meals: 50g carbohydrate \pm 50g fat and 50g protein \pm 50g fat.

Postprandial glucose, insulin and GIP responses to the test meals
(areas under the curve)

	50g carbohydrate	50g carbohydrate + 50g fat	50g protein	50g protein + 50g fat
Glucose mmol l ⁻¹ min ⁻¹	89 \pm 5	43 \pm 13	7 \pm 3	3 \pm 4
Insulin μ U ml ⁻¹ min ⁻¹	1277 \pm 168	1144 \pm 166	368 \pm 75	393 \pm 71
GIP ng ml ⁻¹ min ⁻¹	21 \pm 9	171 \pm 32	6 \pm 4	87 \pm 24

The co-ingestion of fat resulted in a pronounced flattening of the glucose response to carbohydrate but no significant change in the insulin response. The insulin response to protein was similarly unaffected by fat ingestion. Fat ingestion resulted in a marked increase in the GIP response although this effect was less pronounced when fat was consumed with protein than when it was consumed with carbohydrate, which may be an indication that protein is inhibiting fat-induced GIP secretion.

Presumably the presence of fat was slowing gastric emptying and accounting for the flattening of postprandial glucose curves when fat was administered together with carbohydrate. The lack of a resultant suppression of the postprandial insulin response suggests that in the presence of fat insulin response to glucose is potentiated. Gastric inhibitory polypeptide, the secretion of which is strongly stimulated by the intestinal absorption of fat, is known to potentiate glucose-induced insulin secretion, and so could account for apparent increased responsiveness of the β cell to small changes in blood glucose when carbohydrate is consumed together with fat.

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