COMMENTS AND RESPONSES

Comment on: Chen et al. Utilizing the Second-Meal Effect in Type 2 Diabetes: Practical Use of a Soya-Yogurt Snack. Diabetes Care 2010;33:2552–2554

In their brief report, Chen et al. (1) describe the impact of a nutrient preload (30 g soya beans + 75 g yogurt) on postbreakfast plasma glucose (PG), plasma free fatty acid (FFA), and serum insulin and C-peptide concentrations in 10 patients with type 2 diabetes. The soya-yogurt preload (given 2 h before breakfast) in itself elicited small rises in PG and serum insulin concentrations and moderate inhibition of plasma FFA concentrations (1). The preload had no impact on postbreakfast insulin levels, and FFA levels fell to the same nadir following breakfast with or without preload (1). Nevertheless, as shown before (2–5), the nutrient preload resulted in significant lower postbreakfast PG excursions (1). Since no effect of the preload on postprandial insulin secretion was observed, the authors argue—with reference to their previous studies (3,4)—that the mechanism behind the glucose-lowering effect of preloading is due to suppression of FFA (allegedly alleviating insulin resistance), allowing greater storage of glucose as muscle glycogen (1). However, as in previous preload studies from the group (3,4), the postbreakfast insulin response with nutrient preload, occurred at much lower PG levels compared with the postbreakfast insulin levels when no preload was given. This suggests that postbreakfast insulin secretion relative to PG indeed were increased by the preload. Recently Ma et al. (5) published data showing that protein preload elicits release of the insulinotropic glucagon-like peptide 1 (GLP-1) from endocrine L cells in the intestinal epithelium. GLP-1 is known to circulate for hours after nutrient ingestion, and the rather small protein preload used by Ma et al. resulted in significantly higher plasma GLP-1 levels during the following meal compared with the meal without protein preload (5). GLP-1 potently augments glucose-dependent insulin secretion, and, therefore, most likely contributes to the glucose-lowering effect of preloading (5). Also, GLP-1 is, like other nutrient-released gut hormones (e.g., cholecystokinin), known to decelerate gastric emptying—a critical determinant of postprandial PG concentrations. Interestingly, Ma et al. were also able to demonstrate that protein preload significantly slowed gastric emptying during the following meal (5). Thus, the presence of nutrients (from the preload) in the small intestine seems to induce the release of gut-derived signals that increase glucose-induced insulin secretion and slow gastric emptying—two major determinants of postprandial PG excursions. Furthermore, a role of the gastrointestinal tract in determining the impact of preloading on postprandial glycemia may also explain why priming with intravenous glucose (known to suppress plasma FFA concentrations) does not seem to elicit a "second-meal"–like effect in patients with type 2 diabetes (3). Thus, even though the mechanism by which Chen et al. (1) explain their results is of interest, it does not seem to carry the full explanation of the glucose-lowering effect of preloading; gut-derived mechanisms need to be considered as well.

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