COMMENT ON JAKUBOWICZ ET AL.

Fasting Until Noon Triggers Increased Postprandial Hyperglycemia and Impaired Insulin Response After Lunch and Dinner in Individuals With Type 2 Diabetes: A Randomized Clinical Trial. Diabetes Care 2015;38:1820–1826

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More than 100 years ago, it was shown that oral ingestion of two successive carbohydrate meals results in an improved glucose tolerance in healthy people, a phenomenon known as the "Staub-Traugott" effect. In 1970, Metz and Friedenberg (1) reported that an improvement in glucose tolerance to sequential intravenous glucose loads could be seen in both subjects with normal glucose tolerance and with glucose intolerance after a second or even a third isocaloric glucose load. Much work was subsequently carried out to study the metabolic and hormonal mediators of this effect (2). While the precise mechanism of this effect remained unclear, the Staub-Traugott effect was subsequently demonstrated after oral and intravenous glucose administration in both healthy people and those with type 2 diabetes, well summarized by the elegant studies of Bonuccelli et al. (3) and Lee et al. (4).

The excellent clinical trial by Jakubowicz et al. (5) is the latest mechanistic study in patients with type 2 diabetes to demonstrate that the reverse is also true; i.e., omission of breakfast in patients with type 2 diabetes results in worsening of postprandial hyperglycemia after lunch and after dinner. Although the authors prefer the term "second-meal effect," their study certainly rekindles the interest in the historical Staub-Traugott phenomenon. Although this second-meal phenomenon after skipping breakfast was accompanied by a significant increase in glucose, free fatty acid, and glucagon levels and a decrease in insulin and glucagon-like peptide 1 levels, additional studies are needed to see if these changes in metabolites and hormonal levels provide the full explanation for the hyperglycemic effects observed. In some earlier studies, the increase in insulin levels (priming of the β-cells) did not explain the improvement in glucose tolerance. Particularly noteworthy are the physiologic studies of Bonuccelli et al. (3), where the glucose potentiation of insulin secretion and a marked suppression in endogenous hepatic glucose release, partly mediated by glucagon-like peptide 1—not the glucose clearance or the peripheral insulin sensitivity enhancement after the initial glucose challenge—were the predominant mechanisms underlying an improved tolerance to sequential glucose load.

Recent large observational studies (6) and a meta-analysis (7), showing a 15–21% increase in the risk of diabetes, after adjustments for known risk factors, in those who regularly skip breakfast and even a significant increase in weight gain in such people (7), support the work by Jakubowicz et al. (5), with obvious implications for diabetes prevention.

Duality of Interest. No potential conflicts of interest relevant to this article were reported.

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