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Fructose consumption: considerations for future research on its effects on adipose distribution, lipid metabolism, and insulin sensitivity in humans.

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Abstract

Results from a recent study investigating the metabolic effects of consuming fructose-sweetened beverages at 25% of energy requirements for 10 wk demonstrate that a high-fructose diet induces dyslipidemia, decreases insulin sensitivity, and increases visceral adiposity. The purpose of this review is to present aspects of the study design which may be critical for assessment of the metabolic effects of sugar consumption. Collection of postprandial blood samples is required to document the full effects of fructose on lipid metabolism. Fasting triglyceride (TG) concentrations are an unreliable index of fructose-induced dyslipidemia. Differences in the short-term (24-h) and long-term (>2 wk) effects of fructose consumption on TG and apolipoprotein-B demonstrate that acute effects can differ substantially from those occurring after sustained fructose exposure. Investigating the effects of fructose when consumed ad libitum compared with energy-balanced diets suggest that additive effects of fructose-induced de novo lipogenesis and positive energy balance may contribute to dyslipidemia and decreased insulin sensitivity. Increases of intra-abdominal fat observed in subjects consuming fructose, but not glucose, for 10 wk indicate that the 2 sugars have differential effects on regional adipose deposition. However, the increase of fasting glucose, insulin, and homeostasis model assessment-insulin resistance at 2 wk and the lack of increase of 24-h systemic FFA concentrations suggest that fructose decreases insulin sensitivity independently of visceral adiposity and FFA. The lower postprandial glucose and insulin excursions in subjects consuming fructose and increased excursions in those consuming glucose do not support a relationship between dietary glycemic index and the development of dyslipidemia, decreased insulin sensitivity, or increased visceral adiposity.

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