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## Dietary fructose reduces circulating insulin and leptin, attenuates postprandial suppression of ghrelin, and increases triglycerides in women.

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### Abstract

Previous studies indicate that leptin secretion is regulated by insulin-mediated glucose metabolism. Because fructose, unlike glucose, does not stimulate insulin secretion, we hypothesized that meals high in fructose would result in lower leptin concentrations than meals containing the same amount of glucose. Blood samples were collected every 30-60 min for 24 h from 12 normal-weight women on 2 randomized days during which the subjects consumed three meals containing 55, 30, and 15% of total kilocalories as carbohydrate, fat, and protein, respectively, with 30% of kilocalories as either a fructose-sweetened [high fructose (HFr)] or glucose-sweetened [high glucose (HGI)] beverage. Meals were isocaloric in the two treatments. Postprandial glycemic excursions were reduced by 66 +/- 12%, and insulin responses were 65 +/- 5% lower (both  $P < 0.001$ ) during HFr consumption. The area under the curve for leptin during the first 12 h (-33 +/- 7%;  $P < 0.005$ ), the entire 24 h (-21 +/- 8%;  $P < 0.02$ ), and the diurnal amplitude (peak - nadir) (24 +/- 6%;  $P < 0.0025$ ) were reduced on the HFr day compared with the HGI day. In addition, circulating levels of the orexigenic gastroenteric hormone, ghrelin, were suppressed by approximately 30% 1-2 h after ingestion of each HGI meal ( $P < 0.01$ ), but postprandial suppression of ghrelin was significantly less pronounced after HFr meals ( $P < 0.05$  vs. HGI). Consumption of HFr meals produced a rapid and prolonged elevation of plasma triglycerides compared with the HGI day ( $P < 0.005$ ). Because insulin and leptin, and possibly ghrelin, function as key signals to the central nervous system in the long-term regulation of energy balance, decreases of circulating insulin and leptin and increased ghrelin concentrations, as demonstrated in this study, could lead to increased caloric intake and ultimately contribute to weight gain and obesity during chronic consumption of diets high in fructose.

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