Clinical Insights

Fructose Research

Research From:

American Journal of Physiology & Endocrinology Metabolism; October; 2000

Dirlewanger M, Schneiter P, Jequier E, Tappy L. Institute of Physiology, University of Lausanne Medical School, Lausanne, Switzerland

Title of Research:

Effects of Fructose On Hepatic Glucose Metabolism in Humans

Conclusion of Research:

"These results indicate that fructose infusion induces hepatic and extrahepatic insulin resistance in humans."

Following are research article from National Library of Medicine

1: Am J Physiol Endocrinol Metab 2000 Oct;279 (4):E907-11

Effects of fructose on hepatic glucose metabolism in humans.

Dirlewanger M, Schneiter P, Jequier E, Tappy L.

Institute of Physiology, University of Lausanne Medical School, 1005 Lausanne, Switzerland.

Hepatic and extrahepatic insulin sensitivity was assessed in six healthy humans from the insulin infusion required to maintain an 8 mmol/l glucose concentration during hyperglycemic pancreatic clamp with or without infusion of 16.7 &mgr;mol. kg(-1). min(-1) fructose. Glucose rate of disappearance (GR(d)), net endogenous glucose production (NEGP), total glucose output (TGO), and glucose cycling (GC) were measured with [6,6-(2)H(2)]- and [2-(2)H(1)]glucose. Hepatic glycogen synthesis was estimated from uridine diphosphoglucose (UDPG) kinetics as assessed with [1-(13)C] galactose and acetaminophen. Fructose infusion increased insulin requirements 2.3-fold to maintain blood glucose. Fructose infusion doubled UDPG turnover, but there was no effect on TGO, GC, NEGP, or GR(d) under hyperglycemic pancreatic clamp protocol conditions. When insulin concentrations were matched during a second hyperglycemic pancreatic clamp protocol, fructose administration was associated with an 11.1 micromol. kg(-1). min(-1) increase in TGO, a 7.8 micromol. kg(-1). min(-1) increase in NEGP, a 2.2 micromol. kg(-1). min(-1) increase in GC, and a 7.2 micromol. kg(-1). min(-1) decrease in GR(d) ($P < 0.05$). These results indicate that fructose infusion induces hepatic and extrahepatic insulin resistance in humans.

Publication Types:

- Clinical Trial

PMID: 11001775 [PubMed - indexed for MEDLINE]

Research From:

American Journal of Physiology & Endocrinology Metabolism; October; 2000
Dirlewanger M, Schneiter P, Jequier E, Tappy L.
Institute of Physiology, University of Lausanne Medical School, Lausanne, Switzerland

Title of Research:

Effects of Fructose On Hepatic Glucose Metabolism in Humans

Conclusion of Research:

"These results indicate that fructose infusion induces hepatic and extrahepatic insulin resistance in humans."

Effects of fructose on hepatic glucose metabolism in humans.

Dirlewanger M, Schneiter P, Jequier E, Tappy L.

Institute of Physiology, University of Lausanne Medical School, 1005 Lausanne, Switzerland.

Hepatic and extrahepatic insulin sensitivity was assessed in six healthy humans from the insulin infusion required to maintain an 8 mmol/l glucose concentration during hyperglycemic pancreatic clamp with or without infusion of 16.7 &mgr;mol. kg(-1). min(-1) fructose. Glucose rate of disappearance (GR(d)), net endogenous glucose production (NEGP), total glucose output (TGO), and glucose cycling (GC) were measured with [6,6-(2)H(2)]- and [2-(2)H(1)]glucose. Hepatic glycogen synthesis was estimated from uridine diphosphoglucose (UDPG) kinetics as assessed with [1-(13)C] galactose and acetaminophen. Fructose infusion increased insulin requirements 2.3-fold to maintain blood glucose. Fructose infusion doubled UDPG turnover, but there was no effect on TGO, GC, NEGP, or GR(d) under hyperglycemic pancreatic clamp protocol conditions. When insulin concentrations were matched during a second hyperglycemic pancreatic clamp protocol, fructose administration was associated with an 11.1 micromol. kg(-1). min(-1) increase in TGO, a 7.8 micromol. kg(-1). min(-1) increase in NEGP, a 2.2 micromol. kg(-1). min(-1) increase in GC, and a 7.2 micromol. kg(-1). min(-1) decrease in GR(d) ($P < 0.05$). **These results indicate that fructose infusion induces hepatic and extrahepatic insulin resistance in humans.**

Publication Types:

- Clinical Trial

PMID: 11001775 [PubMed - indexed for MEDLINE]

Research From

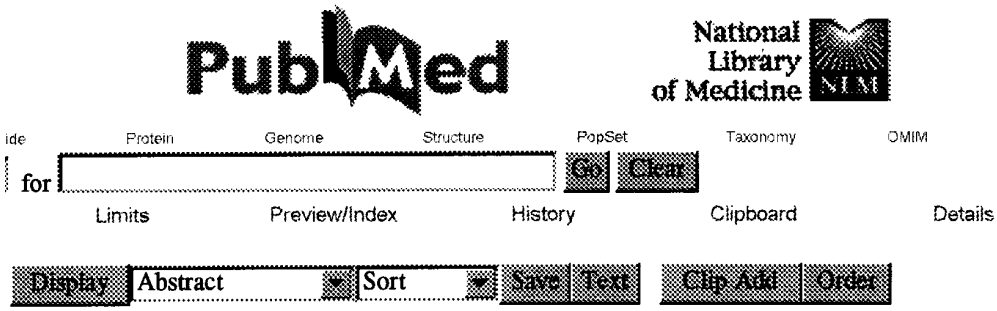
Am J Physiology Endocrinol Metab 2000 April 278(4):E677-83
Suga A, Hirano T, Kageyama H, Osaka T, Namba Y, Tsuji M, Miura M, Adachi M, Inoue S.
Department of Internal Medicine, Showa University School of Medicine, Tokyo, Japan

Title of Research:

Effects of Fructose and Glucose on Plasma Leptin, Insulin and Insulin Resistance in Lean and Obese Rats.

Conclusion of Research:

"Dietary fructose does not alter leptin levels, although this sugar brings about hyperinsulinemia and insulin resistance."



1: Am J Physiol Endocrinol Metab 2000 Apr;278 (4):E677-83

Related Articles, Books, LinkOut

Effects of fructose and glucose on plasma leptin, insulin, and insulin resistance in lean and VMH-lesioned obese rats.

Suga A, Hirano T, Kageyama H, Osaka T, Namba Y, Tsuji M, Miura M, Adachi M, Inoue S.

First Department of Internal Medicine, Showa University School of Medicine, Shinagawa-ku, Tokyo 142-8666, Japan.

To determine the influence of dietary fructose and glucose on circulating leptin levels in lean and obese rats, plasma leptin concentrations were measured in ventromedial hypothalamic (VMH)-lesioned obese and sham-operated lean rats fed either normal chow or fructose- or glucose-enriched diets (60% by calories) for 2 wk. Insulin resistance was evaluated by the steady-state plasma glucose method and intravenous glucose tolerance test. In lean rats, glucose-enriched diet significantly increased plasma leptin with enlarged parametrial fat pad, whereas neither leptin nor fat-pad weight was altered by fructose. Two weeks after the lesions, the rats fed normal chow had marked greater body weight gain, enlarged fat pads, and higher insulin and leptin compared with sham-operated rats. Despite a marked adiposity and hyperinsulinemia, insulin resistance was not increased in VMH-lesioned rats. Fructose brought about substantial insulin resistance and hyperinsulinemia in both lean and obese rats, whereas glucose led to rather enhanced insulin sensitivity. Leptin, body weight, and fat pad were not significantly altered by either fructose or glucose in the obese rats. These results suggest that dietary glucose stimulates leptin production by increasing adipose tissue or stimulating glucose metabolism in lean rats. Hyperleptinemia in VMH-lesioned rats is associated with both increased adiposity and hyperinsulinemia but not with insulin resistance. Dietary fructose does not alter leptin levels, although this sugar brings about hyperinsulinemia and insulin resistance, suggesting that hyperinsulinemia compensated for insulin resistance does not stimulate leptin production.

PMID: 10751202 [PubMed - indexed for MEDLINE]