From: [redacted]
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Subject: HFCS
Date: Sat, 23 Apr 2011 00:24:08 +0000

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yesterday you were reading me something from columbia or princeton about high fructose corn syrup. where did you get it? just want to make sure bases covered.

check these out:

Pediatr Ann. 2006 Dec;35(12):898-902, 905-7.

The 'skinny' on childhood obesity: how our western environment starves kids' brains.

Lustig RH.

Division of Endocrinology, University of California at San Francisco Center for Obesity Assessment, Study and Ttreatment, 94143-0434, USA. [redacted]

Comment in:

- Pediatr Ann. 2007 Apr;36(4):176.

Abstract

In this review, the mechanism of our "toxic environment's" effects on insulin and weight gain in the genesis of obesity is elaborated. The composition of our diet is highly insulinogenic. The insulin drives energy into fat, and interferes with leptin signaling in the VMH. This results in weight gain and the sense of starvation, which results in decreased SNS activity, reducing energy expenditure and physical activity; and increased vagal activity, which promotes yet further insulin release and energy storage. Thus, hyperinsulinemia turns the leptin negative feedback system into a "vicious cycle" of obesity (see Figure 3, page 905). Externally, this appears as "gluttony and sloth" but it is biochemically driven. How does this work? A thin, insulin-sensitive, 13-year-old boy might consume a daily allotment of 2,000 kcal, and burn 2,000 kcal daily (or 50 kcal/kg fat-free mass) in order to remain weight-stable, with a stable leptin level. However, if that same 13-year-old became hyperinsulinemic and/or insulin resistant, perhaps as many as 250 kcal of the daily allotment would be shunted to storage in adipose tissue, promoting a persistent obligate weight gain. Due to the obligate energy storage, he now only has 1,750 kcal per day to burn. The hyperinsulinemia also results in a lower level of leptin signal transduction, conveying a CNS signal of energy insufficiency. The remaining calories available are lower than his energy expenditure; the CNS would sense starvation. Through decreased SNS tone, he would reduce his physical activity, resulting in decreased quality of life; and through increased vagal tone, he would increase caloric intake and insulin secretion, but now at a much higher level. Thus, the vicious cycle of gluttony, sloth, and obesity is promulgated. Is this personal responsibility, when a kid's brain thinks it's starving? Is it personal responsibility when the American Academy of Pediatrics still recommends juice for toddlers? Is it personal responsibility when the Women, Infant and Children program subsidizes fruit juice but not fruit? Is it personal responsibility when the first ingredient in the barbecue sauce is high-fructose corn syrup? Is it personal responsibility when high-fiber fresh produce is unavailable in poor neighborhoods? Is it personal responsibility when the local fast food restaurant is the only neighborhood venue that is clean and air-conditioned? Is it personal responsibility when in order to meet the criteria for No Child Left Behind, the school does away with physical education class? Is it personal responsibility when children are not allowed out of the house to play for fear of crime? We must get the insulin down. Fixing the "toxic environment" by altering the food supply and promoting physical activity for all

children can't be done by government, and won't be done by Big Food. This will require a grassroots, bottom-up effort on the part of parents and community leaders. We as pediatricians must lead the way.

PMID: 17236437 [PubMed - indexed for MEDLINE]

Acta Physiol (Oxf). 2011 Jan;201(1):55-62. doi: 10.1111/j.1748-1716.2010.02167.x.

Dietary fructose, salt absorption and hypertension in metabolic syndrome: towards a new paradigm.

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Abstract
The worldwide increase in the incidence of metabolic syndrome correlates with marked increase in total fructose intake in the form of high-fructose corn syrup, beverage and table sugar. Increased dietary fructose intake in rodents has been shown to recapitulate many aspects of metabolic syndrome by causing hypertension, insulin resistance and hyperlipidaemia. Recent studies demonstrated that increased dietary fructose intake stimulates salt absorption in the small intestine and kidney tubules, resulting in a state of salt overload and thus causing hypertension. The absorption of salt (sodium and chloride) in the small intestine is predominantly mediated via the chloride/base exchangers DRA (Down Regulated in Adenoma) (SLC26A3) and PAT1 (Putative Anion Transporter 1) (SLC26A6), and the Na(+) /H(+) exchanger NHE3 (Sodium Hydrogen Exchanger3) (SLC9A3). PAT1 and NHE3 also co-localize on the apical membrane of kidney proximal tubule. Luminal fructose stimulated salt absorption in the jejunum and kidney tubules, responses that were significantly diminished in PAT1 null mice. These studies further demonstrated that Glut5 (SLC2A5) is the major fructose-absorbing transporter in the small intestine (and kidney proximal tubule) and plays an essential role in the systemic homeostasis of fructose. Increased dietary fructose intake for several weeks upregulated the expression of NHE3, PAT1 and Glut5 in the intestine and resulted in hypertension in wild-type mice, a response that was almost abolished in PAT1 null mice and abrogated in Glut5 null mice. This article will discuss the interaction of Glut5 with salt-absorbing transporters and review the role of dietary fructose in enhanced salt absorption in intestine and kidney as it relates to the pathogenesis of hypertension in metabolic syndrome.

Kidney Int. 2008 Aug;74(4):438-47. Epub 2008 May 21.

Fructose-induced hypertension: essential role of chloride and fructose absorbing transporters PAT1 and Glut5.

Singh AK, Amlal H, Haas PJ, Dringenberg U, Fussell S, Barone SL, Engelhardt R, Zuo J, Seidler U, Soleimani M.

Department of Gastroenterology, Hepatology and Endocrinology, Hannover Medical School, Hannover, Germany.

Comment in:

- Kidney Int. 2008 Aug;74(4):409-10.

Abstract

Increased dietary fructose in rodents recapitulates many aspects of the Metabolic Syndrome with hypertension, insulin resistance and dyslipidemia. Here we show that fructose increased jejunal NaCl and water absorption which was significantly decreased in mice whose apical chloride/base exchanger Slc26a6 (PAT1, CFEX) was

knocked out. Increased dietary fructose intake enhanced expression of this transporter as well as the fructose-absorbing transporter Slc2a5 (Glut5) in the small intestine of wild type mice. Fructose feeding decreased salt excretion by the kidney and resulted in hypertension, a response almost abolished in the knockout mice. In parallel studies, a chloride-free diet blocked fructose-induced hypertension in Sprague Dawley rats. Serum uric acid remained unchanged in animals on increased fructose intake with hypertension. We suggest that fructose-induced hypertension is likely caused by increased salt absorption by the intestine and kidney and the transporters Slc26a6 and Slc2a5 are essential in this process.

PMID: 18496516 [PubMed - indexed for MEDLINE]