Are our children being fruct? Sugar on the brain

Robert H. Lustig, M.D., M.S.L. Emeritus Professor, Division of Endocrinology Department of Pediatrics Institute for Health Policy Studies University of California, San Francisco

Smart Kids and Teens, Food for the Brain, April 26, 2025

Disclosures



<text>

Chief Medical Officer: BioLumen Ireneo Health Perfact Paid Advisor: Simplex Health Myka Labs Levels Health Unpaid Advisor: Kuwaiti Danish Dairy Blue Oak Nutriceuticals

I will not be speaking about any of these companies or products



56% of the food sold in America is ultraprocessed food Accounts for 62% of the sugar in the American diet And 67% of the sugar in kids' diets





FINTE & INCLUSION BUSIES B

NOV 20, 2011 DRAFT EMBARGOED & CONFIDENTIAL

SUGAR IN CHILDREN'S CEREALS: POPULAR BRANDS PACK MORE SUGAR THAN SNACK CAKES AND COOKIES

10 Worst Children's Cereals

Based on percent sugar by weight		Percent sugar by weight
	Kellogg's Honey Smacks	55.6%
	2 Post Golden Crisp	51.9%
	8 Kellogg's Froot Loops Marshmallow	48.3%
	Quaker Oats Cap'n Crunch's OOPS! All Berries	46.9%
	Ouaker Oats Cap'n Crunch Original	44.4%
	Quaker Oats Oh!s	44.4%
	Kellogg's Smorz	43.3%
	8 Kellogg's Apple Jacks	42.9%
	Quaker Oats Cap'n Crunch's Crunch Berries	42.3%
2	Kellogg's Froot Loops Original	41.4%

Source: EWG analysis of nutrition labels for 84 children's cereals.





Courtesy of M. Lustig



Courtesy of M. Lustig

Sugar is the 'alcohol of the child', yet we let it dominate the breakfast table Robert Lustig

With kids consuming half their sugar quota first thing, it's no wonder they're getting diabetes and liver disease. We have to fight corporate interests



'On average, cereal contains a whopping 12g of sugar, all added, in a typical serving.' Photograph: Stockbyte/Rex Features

Wednesday 4 January 2017 08.31 EST

The change in our global food supply

Addition of fructose

- palatability (esp. with decreased fat)
- browning agent

Removal of fiber

- shelf life
- freezing

High Fructose Corn Syrup is 42-55% Fructose; Sucrose is 50% Fructose



Fructose is not glucose

- Fructose is 7 times more likely than glucose to form Advanced Glycation End-Products (AGE's)
- Fructose does not suppress ghrelin
- Acute fructose does not stimulate insulin (or leptin)
- Hepatic fructose metabolism is different in liver and brain
- Chronic fructose exposure promotes the metabolic syndrome, including brain manifestations

Elliot et al. Am J Clin Nutr, 2002 Bray et al. Am J Clin Nutr, 2004 Teff et al. J Clin Endocrinol Metab, 2004 Gaby, Alt Med Rev, 2005 Le and Tappy, Curr Opin Clin Nutr Metab Care, 2006 Wei et al. J Nutr Biochem, 2006 Johnson et al. Am J Clin Nutr 2007 Rutledge and Adeli, Nutr Rev, 2007 Brown et al. Int. J. Obes, 2008









Sugar effects on the brain: mechanisms

- alter CNS energy metabolism (e.g. mitochondria)
- alter CNS neurotransmission (e.g. glutamate)
- alter CNS trophic factors (e.g. leptin, BDNF)
- alter CNS exposures (e.g. toxins, infections (Strep.)
- addiction (dopamine)

1. CNS energy metabolism: Mitochondrial dysfunction

REVIEWS

Impaired mitochondrial function in psychiatric disorders

Husseini Manji¹, Tadafumi Kato², Nicholas A. Di Prospero¹, Seth Ness¹, M. Flint Beal³, Michael Krams¹ and Guang Chen¹



Manji et al. Nat Rev Neurosci 2012

Fructose reduces liver mitochondrial function, while glucose stimulates it



Softic et al. Cell Metab 30:735, 2019

"The most important takeaway of this study is that high fructose in the diet is bad," says Dr. Kahn. "It's not bad because it's more calories, but because it has effects on liver metabolism to make it worse at burning fat. As a result, adding fructose to the diet makes the liver store more fat, and this is bad for the liver and bad for whole body metabolism."

Dr. C. Ronald Kahn, CEO, Joslin Diabetes Center

Fructose and neuroinflammation in astrocytes and microglia



Johnson et al. Front Aging Neurosci 2020 | https://doi.org/10.3389/fnagi.2020.560865

Fructose induces reprogramming of microglia from M2 (anti-) to M1 (pro-inflammatory)



Ting et al. Front Immunology 2024 DOI 10.3389/fimmu.2024.1375453

2. CNS Neurotransmission: Glutamate—Glutamine—GABA

In vivo magnetic resonance spectra in humans after infusion of glucose or fructose: The human brain turns glucose into fructose



Hwang et al. JCI Insight 2:e90508, 2017

Fructose is Made in the in Brain in Response to Hyperglycemia



Blood glucose raised to 220 mg/dl (12mM) for 4 hrs

Hwang et al. JCI Insight. 2(4):e90508, 2017

CSF:blood ratio of glucose or fructose due to the insulin resistance of pregnancy



Hwang et al. PLoS One 10(6): e0128582, 2015

Glutamine synthetase — Glutamate to glutamine cycle in astrocytes



Hertz, Front Endocrinol 4:1, 2013

The relation between glutaminergic and GABA-ergic neurons is glutamine synthetase in astrocytes



Incubation of rat cortical synaptosomes with glucose or fructose



Hassel et al. J Neurochem 133:572, 2015

Fructose inhibits glutamine synthetase in immune cells



Jones et al. Nat Comm 12:1209, 2021

3. CNS Trophic effects: Insulin—Leptin—BDNF

Ultra-processed food consumption correlates with depression



Gomez-Ronoso et al. Eur J Nutrition 2019 doi:10.1007/s00394-019-01970-1

Insulin resistance is a driver of depression



Watson et al. Neurophamacology 136:327, 2018

Insulin blocks leptin signaling in the brain

Lee, Ann. NY Acad Sci 1243:15, 2011

Leptin regulates neural and glial protein levels Ahima et al. Endocrinology 140:2755, 1999

Leptin corrects cognitive defects in patients with leptin deficiency Paz-Filho et al. PLoS One 3:e3098, 2008

Sugar causes insulin resistance, and insulin resistance causes leptin resistance Lim et al., Nat Rev Gastro Hepatol 7:251, 2010 Lustig, Nat Rev Endocrinol Metab 2:447, 2006

Exercise, BDNF, and depression



Erickson et al. Neuroscientist 18:82, 2012

4. CNS exposures: Gut microbiome

Fructose affects the gut microbiome too

ABSTRACT: Increased sugar intake is implicated in Type-2 diabetes and fatty liver disease; however, the mechanisms through which glucose and fructose promote these conditions are unclear. We hypothesize that alterations in intestinal metabolite and microbiota profiles specific to each monosaccharide are involved. Two groups of six adult C57BL/6 mice were fed for 10-weeks with diets with glucose (G) or fructose (F) as sole carbohydrates, and a third group was fed with a normal chow carbohydrate mixture (N). Fecal metabolites were profiled by nuclear magnetic resonance (NMR) and microbial composition by real-time polymerase



chain reaction (qPCR). Although N, G and F mice exhibited similar weight gains (with slight slower gains for F) and glucose tolerance, multivariate analysis of NMR data indicated that F mice were separated from N and G, with decreased butyrate and glutamate and increased fructose, succinate, taurine, tyrosine, and xylose. The different sugar diets also resulted in distinct intestinal microbiota profiles. That associated with fructose seemed to hold more potential to induce host metabolic disturbances compared to glucose, mainly by promoting bile acid deconjugation and taurine release and compromising intestinal barrier integrity. This may reflect the noted nonquantitative intestinal fructose absorption hence increasing its availability for microbial metabolism, a subject for further investigation.

KEYWORDS: Fructose absorption, short-chain fatty acids, metabolic profiling, metabolomics, intestinal microbiota

Silva et al. J Proteome Res 17:2880, 2018
Tight junctions keep bad stuff out



Di Ciaula et al. J Clin Med 9:2648 2020

Fructose disrupts tight junctions, and lets bad stuff in



Cho et al. Hepatology 2019 Apr 8. doi: 10.1002/hep.30652.

Sugar alters Th17 barrier in the intestine



Kawano et al., Cell 185, 1, 2022

Group A Streptococcus grow better with fructose than glucose



GAS responsible for psych disease:

1) Sydenham's chorea

2) PANDAS OCD tic disorders adult personality dis. mood disorder

Orlovska et al. JAMA Pediatr. 74:740, 2017

Dmitriev et al. J Bacteriol 188:7230, 2006

5. Addiction

Junk food addiction may be clue to obesity: study



(Reuters) March 28, 2010 - Bingeing on high-calorie foods may be as addictive as cocaine or nicotine, and could cause compulsive eating and obesity, according to a study published on Sunday.

Current Drug Abuse Reviews, 2011, 4, 146-162

Is Fast Food Addictive?

Andrea K. Garber^{*,1} and Robert H. Lustig²

¹Division of Adolescent Medicine, University of California San Francisco, San Francisco, CA. 94143, USA ²Division of Pediatric Endocrinology, University of California San Francisco, USA

146



Sugar and opioids



Sweet-Ease increases endogenous opioids to reduce pain, Even in neonates

RESEARCH ARTICLE

Dissociable Behavioral, Physiological and Neural Effects of Acute Glucose and Fructose Ingestion: A Pilot Study

Bettina Karin Wölnerhanssen^{1®}*, Anne Christin Meyer-Gerspach^{1®}, André Schmidt^{2,3}, Nina Zimak¹, Ralph Peterli⁴, Christoph Beglinger¹, Stefan Borgwardt^{2,3}

1 Department of Gastroenterology, University Hospital of Basel, Basel, Switzerland, 2 Medical Image Analysis Center, University Hospital of Basel, Basel, Switzerland, 3 Department of Psychiatry, University Hospital of Basel, Basel, Switzerland, 4 Department of Surgery, St. Clara Hospital, Basel, Switzerland

No satiety or fullness with fructose compared with glucose No insulin rise with fructose compared with glucose

fMRI: Glucose: caudate, putamen, precuneus, lingual gyrus Fructose: amygdala, hippocampus, parahippocampus, orbitofrontal cortex precentral gyrus

PLoS One 10(6):e0130280, 2014

Fructose Increases Hunger, Desire for Food, Increases Visual Cues, Reduces Will Power, and Reduces Recent Memory



Luo et al PNAS. 2015;112:6509 Purnell et al Diabetes Obes Metab. 2011;13:229 Page et al JAMA. 2013;309:63

Ultraprocessed food promotes tolerance and withdrawal

Current Addiction Reports https://doi.org/10.1007/s40429-022-00425-8

FOOD ADDICTION (E SCHULTE, SECTION EDITOR)



Preliminary Evidence that Tolerance and Withdrawal Occur in Response to Ultra-processed Foods

Lindsey Parnarouskis¹ · Ashley N. Gearhardt¹

Accepted: 28 June 2022 © The Author(s), under exclusive licence to Springer Nature Switzerland AG 2022

Adults

Depression

SCIENTIFIC REPORTS

Received: 21 November 2016 Accepted: 1 June 2017 Published online: 27 July 2017

 OPEN Sugar intake from sweet food and beverages, common mental disorder and depression:
²⁰¹⁶ prospective findings from the Whitehall II study

Anika Knüppel 💿, Martin J. Shipley, Clare H. Llewellyn & Eric J. Brunner

Intake of sweet food beverages and added sugars has been linked with depressive symptoms in several

Mental Health: Ultraprocessed food and depression



Ultra-processed food consumption and mental wellbeing outcomes

Rapid Report // September 2023

Global Mind Project (n = 227,000)

Figure 3: Impact of ultra-processed food consumption for different levels of exercise and income

Relationship between MHQ scores and frequency of ultra-processed food consumptions for individuals who (i) exercise several times a week (blue line) or less than once a week to never (red line) for the global sample (left) and (ii) for those who are low income (<\$40,000 annually; red line) versus high income (>\$100,000 annually; blue line) for respondents in the United States (right)



Cognitive Decline

22 Symptoms of Carbohydrate-Associated Reversible Brain (CARB) syndrome

- 1. Craving sweet and starchy food.
- 2. Excessive hunger.
- 3. Excessive mental & physical fatigue.
- 4. Difficulty concentrating and focusing.
- 5. Poor impulse control.
- 6. Depressed affect.
- 7. Excessive anxiety.
- 8. Excessive mood swings.
- 9. Insomnia.
- 10.Diminished sensory filtering.
- 11.Low self esteem.

- 12.Low self-image.
- 13.Diminished cognitive functioning.
- 14.Loss of empathy.
- 15.Chronic diffuse pain.
- 16.Diminished short term memory.
- 17.Internal restlessness, racing thoughts.
- 18.Poor listening skills.
- 19.Obsessive-compulsive tendencies.
- 20.Intestinal symptoms.
- 21.Increased communication lag time.
- 22.Consciously thinking about food & eating.

Ultraprocessed food inhibits cognition in older adults

Fig. 1 The relationship between A В detary UPF (as % of total energy intake) and cognitive 83 test scores: a CERAD total (P=0.308); b CERAD de taved CERAD delay moal 5.5 6 6.5 CERMD total score 10 20 recall (P=0.859); c animal fluency (P=0.010); d digit Symbol Substitution test (DSST) (P=0.005), Green area represents the 95% confidence intervals. UPF ultra-processed food, %keal % of total energy 靀 intake ЧÓ 21.140.8 52.1 63.3 89.1 21.140.8 52.1 63.3 80.1 Ultra-processed foods (%kcal) Ultra-processed foods (%kcal) C D 8 8 Animal Fluency 14 16 15 DSST 50 ΰ. 2 24 21.1 40.8 52.1 63.3 80.1 21.1 40.8 52.1 63.3 60.1 Ultra-processed foods (%kcal) Ultra-processed foods (%koal)



Pase et al Alzheimers Dement.13:955, 2017

Sugar consumption correlates with dementia, as well as other systemic diseases



Figure 1. Participants with chronic conditions of diabetes mellitus (DM, Figure 1A), stroke (Figure 1B), coronary heart disease (CHD, Figure 1C), and peripheral arterial disease (PAD, Fiure 1D) (red lines) had a significantly higher risk of AD than those without these conditions (black lines). There was an increasing trend of sugar intake (as indicated by x-axis) associated with an increase in incident AD rate (as indicated by y-axis) among those with (red line) and without these conditions (black line). Relative risk (RR) was adjusted for the same covariates in Model 3 of Table 4.

Liu et al. Nutr Neuroscience 25:2302, 2022

Does sugar cause dementia?

Obesity is associated with dementia Luchsinger et al. J Alz Dis Assoc Dis 2011

Obesity is associated with altered neural projections c/w dementia Bouret et al. Cell Metab 7:179, 2008

Sugar generates insulin resistance and hyperinsulinemia Seneff et al. Eur J Int Med 22:134, 2011

Insulin resistance and high insulin levels are associated with dementia Craft et al. Nat Rev Neurol 8:360, 2012

Western Diet correlates with dementia Barberger-Gateau et al. Neurology 69:1921, 2007

Fat or sugar both cause obesity in rats, but only sugar caused cognitive decline Jurdak et al. Nutr Neurosci 11:48, 2008

Causative data in animals; but to date no direct associative or causative data in humans



Stephan et al. J Gerontol 65:809, 2010

Anything that increases ROS production can damage cells and result in cognitive decline



Gomez-Pinilla, Nat Rev Neurosci 9:568, 2012

Effect of Dietary Fructose on the Rat Brain



Agrawal et al J Cereb Blood Flow Metab. 2016; 36:941

Insulin resistance, especially in the absence of omega-3's, means lack of neuron signaling



Agrawal and Gomez-Pinilla, J Physiol 590.10:2485, 2012

Dietary Fructose Increases Amyloid and Tau Protein in the Brain of Rats

Amyloid (Congo Red Positive)

Tau Protein (Brown Stain)



Mohamed et al Food Biochem. 2021;45:e13715, 2021 and Nutr Seurosci, 23:1, 27-36: 2020

High Fructose Levels and Low ATP in the Brains of Alzheimer's Patients



Xu et al Sci Rep. 2016 Jun 9;6:27524



Reduced hippocampus but more CSF in adolescents with metabolic syndrome



FIGURE 3

Lower QUICKI scores (more IR) were associated with smaller ICV-adjusted hippocampal volumes (n = 91) (A) and larger ICV-adjusted overall CSF volumes (n = 92) (B).

Yau et al. Pediatrics 130:e856, 2012

Reduced prefrontal cortical function in adolescents with metabolic syndrome

Table 2. Domain scores by MetS classification group.

	No MetS (n = 204)	MetS (n = 84)	p	Effect Size (r)
Memory Processing Speed	100.88 (13.86) 100.57 (13.46)	97.94 (15.55) 100.64 (13.14)	0.195	0.08
Executive Function	101.47 (14.34)	96.97 (15.43)	0.020	0.15
Complex Attention	100.59 (15.69) 103.93 (10.17) 101.61 (14.20)	101.03 (12.81) 101.32 (11.25) 96.53 (16.00)	0.790	0.02 0.11 0.15
Verbal Memory Visual Memory	100.68 (14.07) 100.18 (14.92)	98.18 (15.19) 99.21 (14.41)	0.225 0.674	0.07 0.03

The 'Twinkie Defense':

Relationship between carbonated non-diet soft drinks and violence perpetration among Boston high school students

Adolescents who drank more than five cans of soft drinks per week (30%):

- more likely to have carried a weapon
- violent with peers, family members and dates.

•even after controlling for gender, age, race, BMI, sleep, tobacco use, alcohol use, and family dinners.

Solnick and Hemenway, Inj Prev 18:259, 2012

https://www.frontiersin.org/journals/ neuroscience

ORIGINAL RESEARCH published: 07 June 2021 doi: 10.3389/fnins.2021.670430



Long-Term Overconsumption of Sugar Starting at Adolescence Produces Persistent Hyperactivity and Neurocognitive Deficits in Adulthood

Kate Beecher¹, Ignatius Alvarez Cooper², Joshua Wang¹, Shaun B. Walters³, Fatemeh Chehrehasa^{2†}, Selena E. Bartlett^{1*†} and Arnauld Belmer^{1*†}

Beecher et al. Front Neurosci 15 : 670430, 2021

THE JOURNAL OF PEDIATRICS • www.jpeds.com

Soft Drinks Consumption Is Associated with Behavior Problems in 5-Year-Olds

ORIGINAL

ARTICLES

Shakira F. Suglia, ScD¹, Sara Solnick, PhD², and David Hemenway, PhD³

Suglia et al. J. Pediatr. 163: 1323-1328, 2013




Young children prefer sweet

a 3-point scale



Bobowski and Mennella, Child Obes 13:369, 2017

Commercial infant foods and health claims

PLOS ONE

Food claims and nutrition facts of commercial infant foods

	n	High sodium (>130mg)	p value	n	High sugar (>10%)	p value
Total	363	85 (23.4)		237	129 (54.4)	
Food daim						
Composition claim						
Yes	320	66 (20.6)	0.001	224	122 (54.5)	0.965
No	43	19 (44.2)		13	7 (53.8)	
Nutrition claim						
Yes	151	34 (22.5)	0.733	131	68 (51.9)	0.386
No	212	51 (24.1)		106	61 (57.5)	
Health claim						
Yes	182	61 (33.5)	< 0.001***	146	68 (46.6)	0.002**
No	181	24 (13.3)		91	61 (67.0)	
Salt related claim						
No added salt						
Yes	19	0 (0.0)	0.013*			
No	344	85 (24.7)				
No added seasoning						
Yes	78	4 (5.1)	< 0.001***			
No	285	81 (28.4)				
Sugar claim						
No added sugar						
Yes				48	35 (72.9)	0.004**
No				189	94 (49.7)	

Table 6. High sodium or high sugar content among products with different claims¹.

¹ Data are presented as the number (percentage).

**p* < 0.05;

***p* < 0.01;

*** *p* < 0.001 by chi-square test.

Koo et al. PLoS One 13:e0191982, 2018

Could this be the reason for obesity in 6-month olds?

INGREDIENTS (Powder) ((U) Pareve*)

43.2% Corn syrup solids,
14.6% soy protein isolate,
11.5% high oleic safflower oil,
10.3% sugar (sucrose),
8.4% soy oil,
8.1% coconut oil



Could this be the reason for obesity in 6-month olds?



100 MI Milk Shake

Nutrition Facts

Serving Size	100 ml	
Amount Per Serving Calories	129	
% Da	ily Values*	
Total Fat 4.68g	6%	
Saturated Fat 2.835g	14%	
Trans Fat -		
Polyunsaturated Fat 0.212g		
Monounsaturated Fat 1.286g		
Cholesterol 15mg	5%	
Sodium 65mg	3%	
Total Carbohvdrate 19.24g	7%	
Dietary Fiber 0.9g	3%	
Sugars 16.57g		
Totem 0.00g		
Vitamin D -		
Calcium 101mg	8%	
Iron 0.33mg	2%	
Potassium 165mg	4%	
Vitamin A 47mcg	5%	
Vitamin C 0.2mg	0%	

* The % Daily Value (DV) tells you how much a nutrient in a serving of food contributes to a daily diet. 2,000 calories a day is used for general nutrition advice.

Last updated: 21 Aug 07 07:33 AM Source: FatSecret Platform API

INGREDIENTS (Powder) ((U) Pareve*)

43.2% Corn syrup solids, 14.6% soy protein isolate, 11.5% high oleic safflower oil, 10.3% sugar (sucrose), 8.4% soy oil,

8.1% coconut oil

Coca-Cola:10.5% sucrose (fiberless) Milk shake:4% sucrose (fiberless) Breast Milk:7.1% lactose (fiberless), no sucrose, EXCEPT:

Courtesy of M. Walker





PREPARATION OF MIXTURE BABY FEEDING ON WOODEN BACKGROUNI

Nestlé Adds Sugar to Baby Milk and Cereal in Poorer Nations

The report calls out Nestlé's "double standard" for adding sugar to baby food products in developing countries.

ARMANI SYED

APR 17, 2024 9:16 AM PDT

ARMANI SYED IS A WORLD AFFAIRS REPORTER AT TIME. SHE COVERS GLOBAL AFFAIRS, WITH A FOCUS ON THE SWANA REGION, ARTS AND CULTURE, AND ROYAL INSTITUTIONS.

Added sugar content of the same Cerelac wheat product in different countries

Country	Grammes per portion	
Thailand		6,0 g
Ethiopia		5,2 g
South Africa		4,0 g
Pakistan		2,7 g
India		2,2 g
Bangladesh		1,6 g
United Kingdom		0,0 g
Germany		0,0 g

Source: Public Eye and IBFAN (2024) • Analysis of the nutritional information appearing on the products, or, when the added sugar content is not declared, results of laboratory analyses.

Fructose in breast milk predicts weight and fat mass at 6 mos of age



Goran et al. Nutrients 9:146, 2017



Endocrine Updates Series Editor: Shlomo Melmed

Robert H. Lustig *Editor* Obesity Before Birth Maternal and Prenatal Influences on the Offspring

Obesity obeys the First Law of Thermodynamics. The routine assumption is that obesity is the result of a mismatch between calories in and calories out; in other words, the result of two divergent behaviors. However, there is mounting evidence that biochemical forces can drive obligate weight gain, and that the observed behaviors of increased energy intake and decreased energy expenditure are secondary to these processes. Furthermore, many of these biochemical forces are determined in utero; resulting in a developmental drive toward obesity and disease in later life. Four distinct prenatal forces have thus far been identified: 1) genetics; 2) epigenetics; 3) developmental programming; and 4) environmental obesogens. This volume explores the evidence for each of these in detail in human and animal models, and attempts to provide a cohesive analysis of the biochemical bases of obesity.

This volume will appeal to geneticists, developmental biologists, endocrinologists, epidemiologists, toxicologists, obstetrician/gynecologists, nutritionists, veterinary scientists, animal husbandry researchers, domestic species researchers, and obesity researchers and practitioners.

This very timely volume provides an in-depth scholarly or aview of a critical challenge facing our society—the obesity epidemic. Dr. Robert H. Listigh is a nimiled expert authors to address the fundamental contribution of preprogrammed general disorders leading to obesity, as well as the role of very early environmental influences. The chapters range from classic genetic mechanistic understanding through intra-sterine epigenetic influences, factors determining developmental programming, and the new clinical science of perinatal obesogens.

Obesity Before Birth: Maternal and prenatal influences on the offspring, brings easily accessible, cutting edge information to geneticists, pediatricians, endocrinologists, as well as those clinicians and scientists pursuing the complex yet elusive causes ofchildhood obesity and related disorders.

Shlomo Melmed, M.D. Series Editor

Internal Medicine



springer.com

Endocrine Updates Series Editor: Shlomo Melmed

Robert H. Lustig Editor

Obesity Before Birth

Maternal and Prenatal Influences on the Offspring



Obesity Before Birth

Postulated effect of maternal fructose consumption on the offspring





Experimental maternal fructose ingestion: effects on the placenta



Experimental maternal fructose ingestion: effects on the offspring





Sugar and epigenetics

Table 3. Overall Unadjusted and Adjusted Associations Between Dietary Exposure and GrimAge2

	β (95% CI)	
Dietary exposure	Unadjusted (n = 342) ^a	Adjusted (n = 325) ^b
Alternate Mediterranean diet score	-0.62 (-0.91 to -0.34) ^c	-0.41 (-0.69 to -0.13) ^c
Alternate Healthy Eating Index -2010 score	-0.10 (-0.13 to -0.06) ^c	-0.05 (-0.08 to -0.01) ^c
Epigenetic Nutrient Index score	-0.19 (-0.29 to -0.08) ^c	-0.17 (-0.29 to -0.06) ^c
Added sugar, g	0.02 (0.01 to 0.04) ^c	0.02 (0.01 to 0.04) ^c



Original Investigation Genetics	and Genomics
Essential Nutrients,	Added Sugar Intake, and Epigenetic Age
in Midlife Black and	White Women
NIMHD Social Epiger	nomics Program

Dorothy T. Chiu, PhD; Elissa June Hamlat, PhD; Joshua Zhang, PhD; Elissa S. Epel, PhD; Barbara A. Laraia, PhD, MPH, RD

Table 4. Overall Adjusted Associations for Diet Quality Scores and Added Sugar With GrimAge2 Where Dietary Exposures Are Combined Among 325 Participants

Dietary exposure model ^a	Adjusted β (95% CI) ^b	
Model 1: aMED and added sugar		
aMED score	-0.29 (-0.58 to -0.00) ^c	
Added sugar, g	0.02 (0.00 to 0.03) ^c	
Model 2: AHEI-2010 and added sugar		
AHEI score	-0.03 (-0.07 to 0.01)	
Added sugar, g	0.02 (0.00 to 0.03) ^c	
Model 3: ENI and added sugar		
ENI score	-0.12 (-0.25 to 0.01)	
Added sugar, g	0.02 (0.00 to 0.03) ^c	
Abbreviations: AHEI-2010. Alternate Healthy Fating Index 2010: aMED. Alternate Mediterranean Diet: ENI. Enigenetic		

Abbreviations: AHEI-2010, Alternate Healthy Eating Index 2010; aMED, Alternate Mediterranean Diet; ENI, Epigenetic Nutrient Index.

HOME > NEWS > ALL NEWS > BRITAIN'S POSTWAR SUGAR CRAZE CONFIRMS HARMS OF SWEET DIETS IN EARLY LIFE

NEWS | BIOLOGY

Britain's postwar sugar craze confirms harms of sweet diets in early life

End of sugar rationing boosted diabetes, hypertension rates years later

31 OCT 2024 · 2:00 PM ET · BY CATHERINE OFFORD



UK sugar rationing ended in 1953 offspring born after rationing with higher diabetes and hypertension







Untested hypothesis: fructose alters epigenetics to alter prenatal brain development



Dufault et al. World J Clin Pediatr 12:25, 2023



New York Times, April 17, 2011

Nature 487:27-29, Feb 1, 2012



ECOLOGY Komodo dragons and elephants could reduce fire risk in Australia **p.30** NEUROSCIENCE The source of the self is in the brain's wiring, not individual neurons p.31

LITERATURE How Charles Dickens drew on science, but left room for wonder **p.32** OBITUARY Philip Lawley and the discovery that DNA damage can cause cancer **p.36**



The toxic truth about sugar

Added sweeteners pose dangers to health that justify controlling them like alcohol, argue Robert H. Lustig, Laura A. Schmidt and Claire D. Brindis.

Summary

- Fructose, the sweet molecule of sugar, is preferentially turned into liver fat
- That fat precipitates in the liver, leading to insulin resistance and NAFLD
- Fructose also generates advanced glycation endproducts, which inhibit mitochondrial function
- Fructose effects gut permeability, inflammation, and autoimmunity
- Fructose (and insulin) affect the brain, leading to addiction, depression, dementia, cognitive decline, and irritability
- Kids' and baby food are oversweetened on purpose
- Maternal fructose ingestion can cross into breast milk, and possibly increase infant weight gain and body fat
- Fructose can also cross the placenta, leading to placental insufficiency, and induction of fat-making enzymes in the fetus, and possibly epigenetic changes that could lead to long-term brain alterations