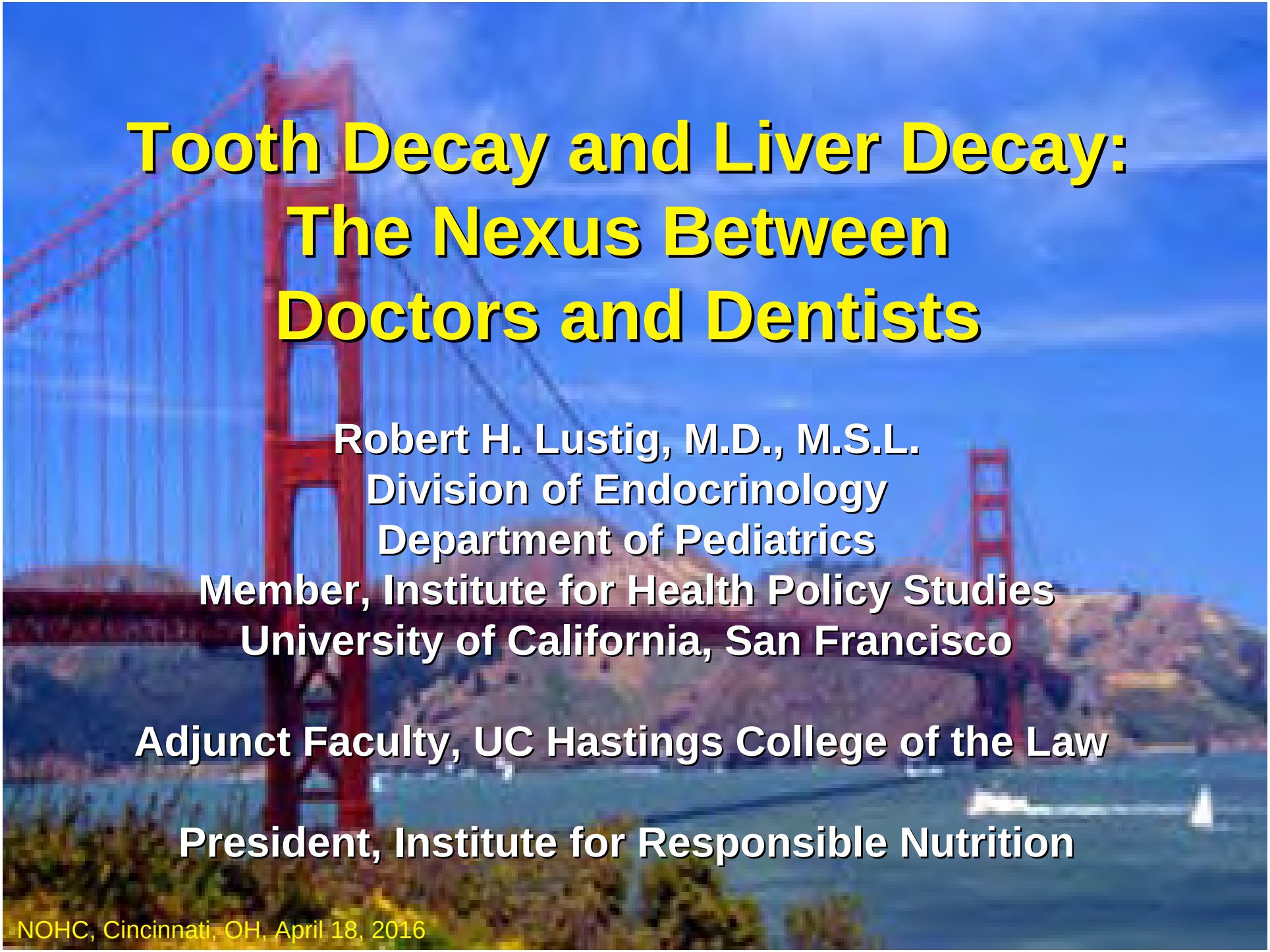


Tooth Decay and Liver Decay: The Nexus Between Doctors and Dentists

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

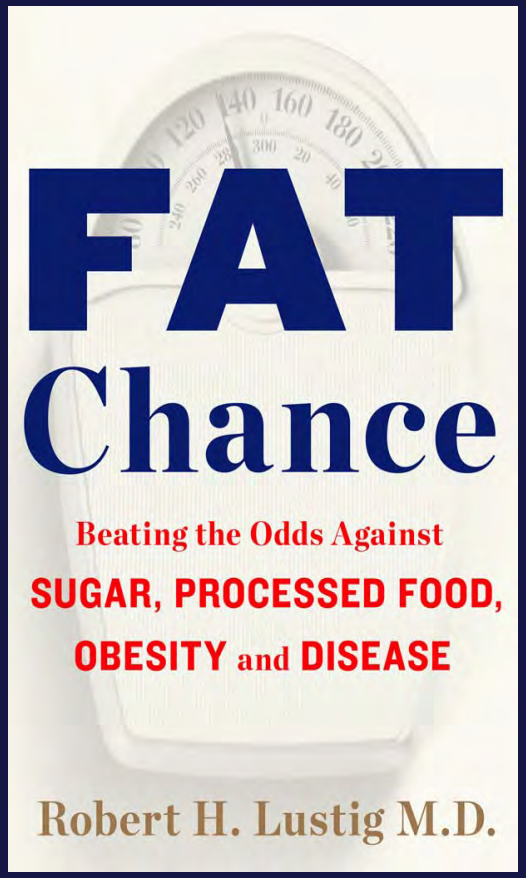
Adjunct Faculty, UC Hastings College of the Law

President, Institute for Responsible Nutrition

NOHC, Cincinnati, OH, April 18, 2016

- **No disclosures**

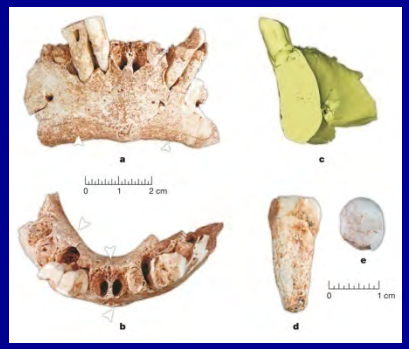
(except I wrote a book)



Tooth Decay

Dentists were the first anti-sugar advocates

"The worst mistake in the history of the human race" (Jared Diamond)

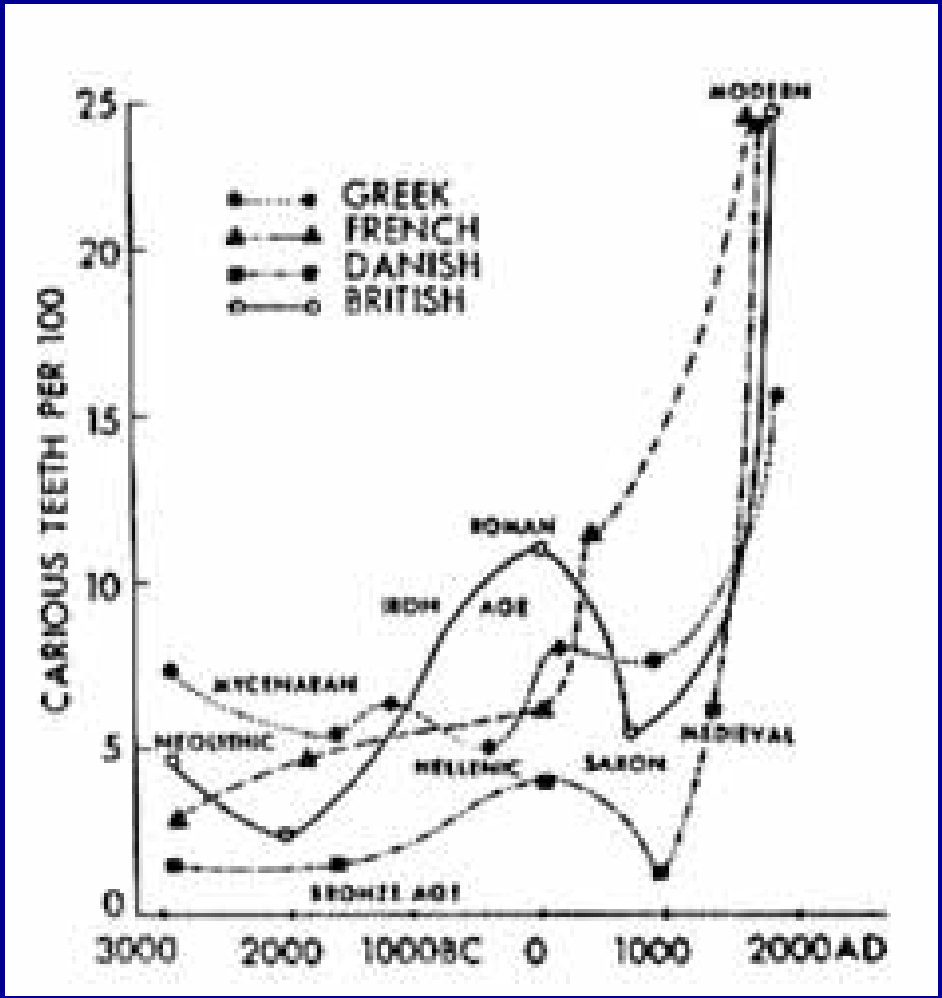


Virtually no evidence of caries in the human fossil record until 10-12k ya (i.e, after the invention of Agriculture)

5 million BCE

10,000 years

Prevalence of Dental Caries in European Populations



Kean, 1980

March 27, 1934
Hotel Pennsylvania, New York City

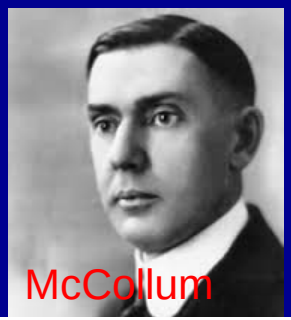
Conservative theory — Clean teeth do not decay:

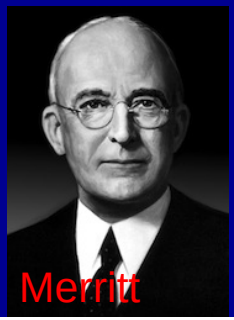
Dr. Thaddeus P. Hyatt, Metropolitan Life and New York University
Dr. Alfred Walker, New York University
Dr. Maurice William, Oral Hygiene Committee of Greater New York

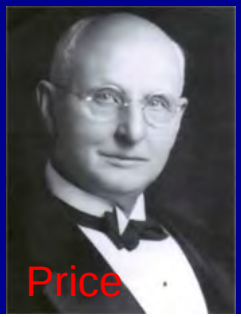
Nutritional dentistry — Caries are a manifestation of your internal metabolic milieu:

Dr. Elmer V. McCollum, Johns Hopkins University
Dr. Arthur H. Merritt, American Academy of Periodontics
Dr. Weston A. Price, Dental Research Laboratories, Cleveland, OH.


William

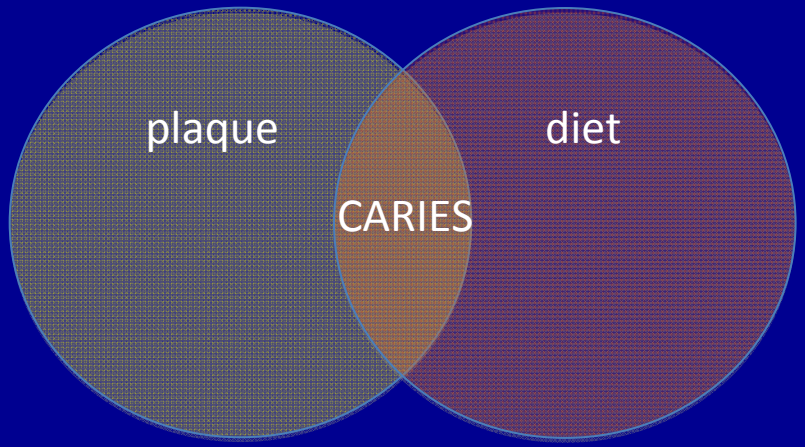

McCollum


Merritt


Price

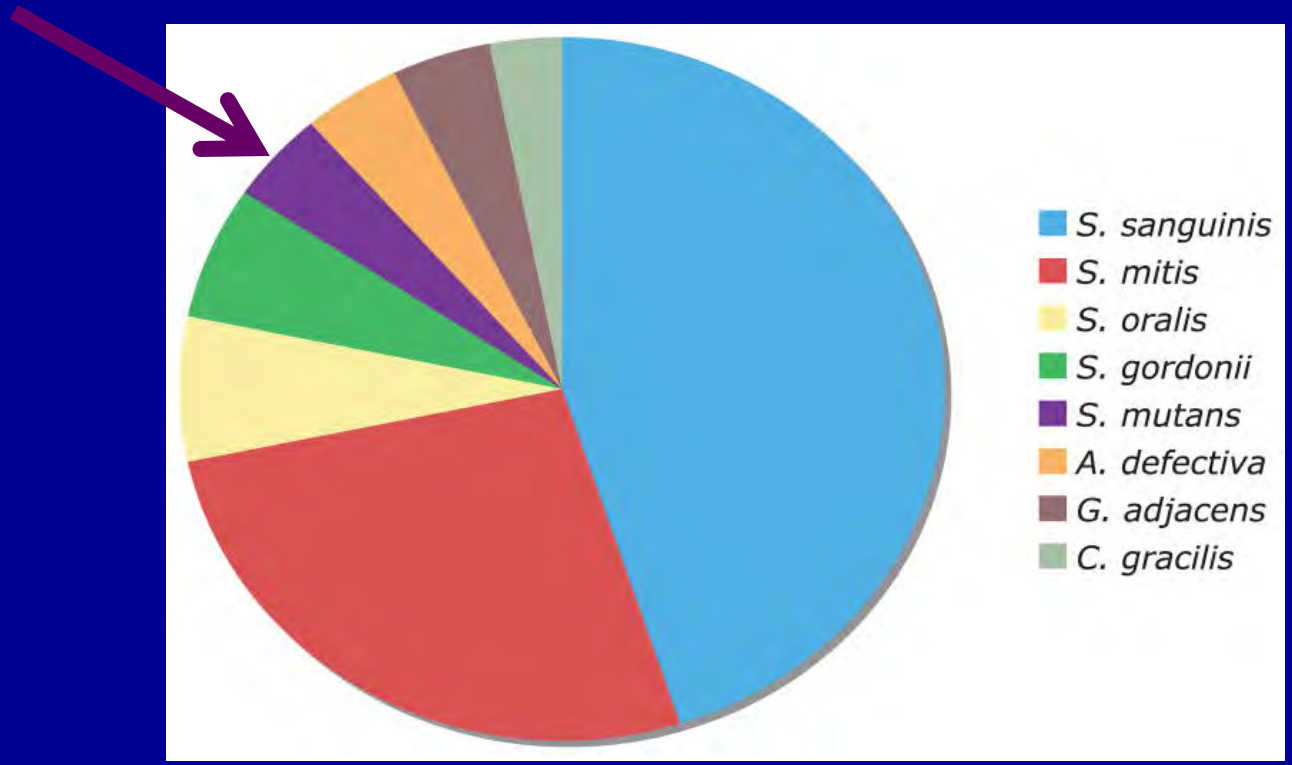
The pathogenesis of caries 1934

Hyatt
Walker
William



Keyes and Jordan, 1963

Current mouth flora



Peterson et al. Int Dent J 61:11, 2011

The Modern Rise of Strep. Mutans

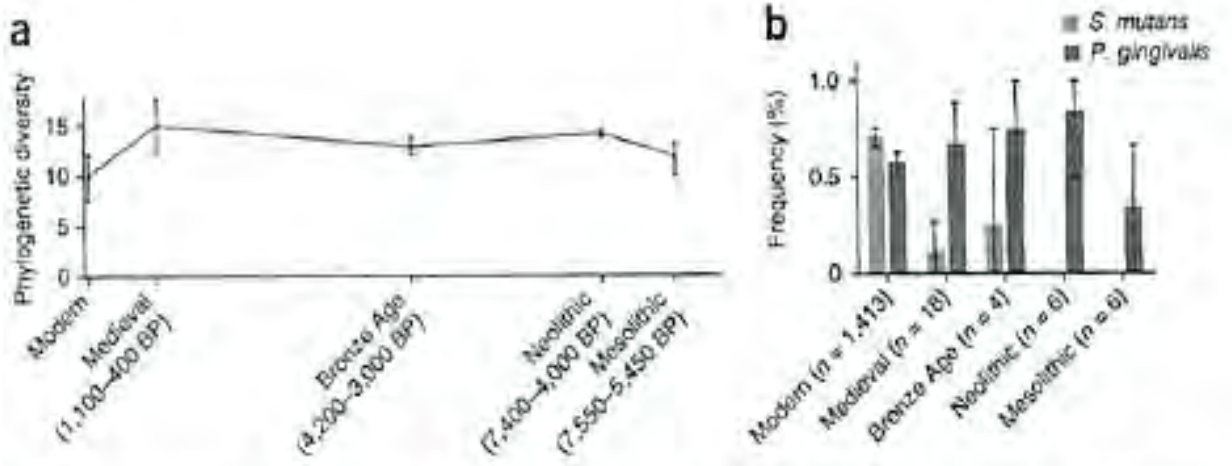
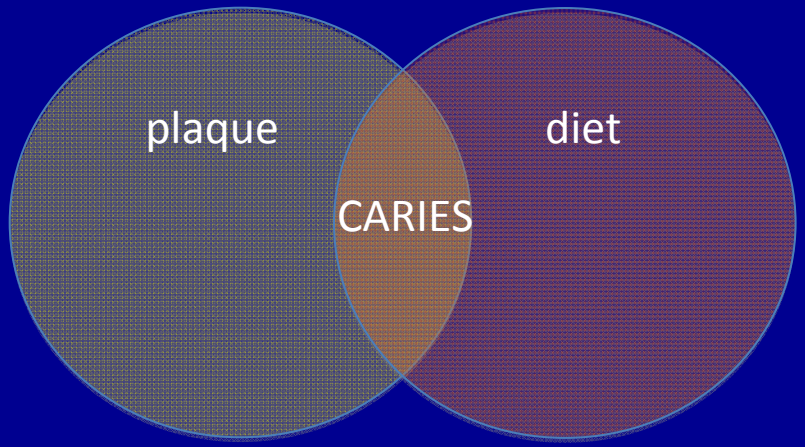


Figure 3 Changes in the diversity and composition of oral microbiota. (**a**) For the V3 region sequences, we estimated the phylogenetic diversity[50] (**Supplementary Note**) of the archaeological dental calculus samples ($n = 34$) and compared them to modern calculus ($n = 6$) and plaque ($n = 13$). We estimated phylogenetic diversity from only classified, Gram-positive bacterial sequences to minimize the influence of taphonomic bias (**Supplementary Note**). Diversity was calculated at a depth of 34 sequences and bootstrapped to assess the robustness of the pattern. Error bars represent bootstrapped diversity values generated by sampling 255 replicates without replacement. BP, years before the present. (**b**) Specific primers were used to amplify sequences unique to the oral pathogens *S. mutans* and *P. gingivalis*. Error bars represent bootstrapped frequencies generated by sampling 255 replicates without replacement.

Adler et al. Nat Genet 45:450, 2013

The pathogenesis of caries 1934

Hyatt
Walker
William



McCollum
Merritt
Price

Keyes and Jordan, 1963

More sugar + older teeth = More caries

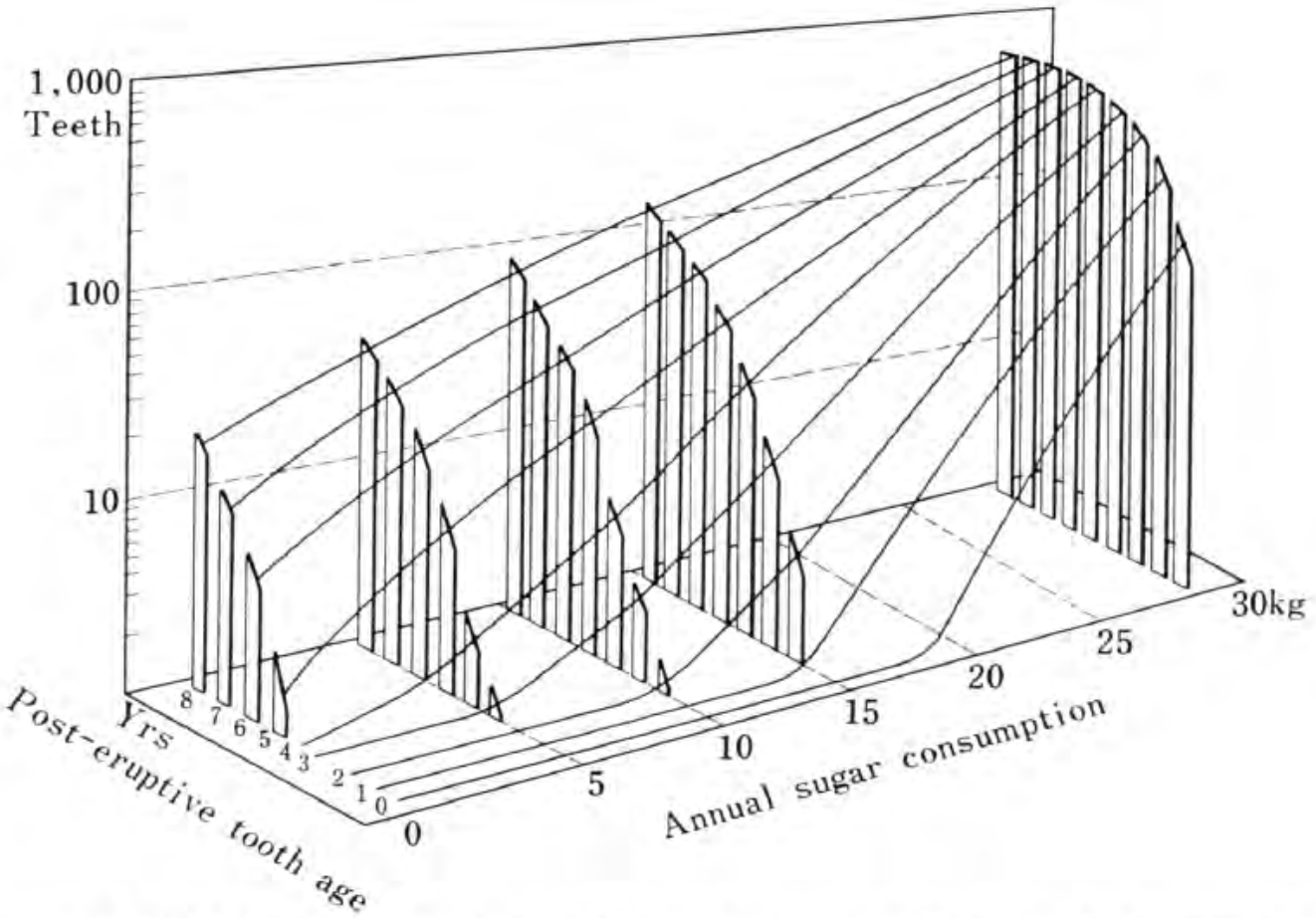


Figure 1 Three-dimensional model of the cumulative numbers of caries in upper central incisor teeth. Data were plotted on a log scale, by post-eruptive tooth age up to 8 years, and related to the average annual sugar consumption per head in Japan from 1935 to 1957 (Takeuchi et al. [14], with permission).

Sheiham and James, BMC Public Health 14:863, 2014

Log-linear relationship between sugar and caries

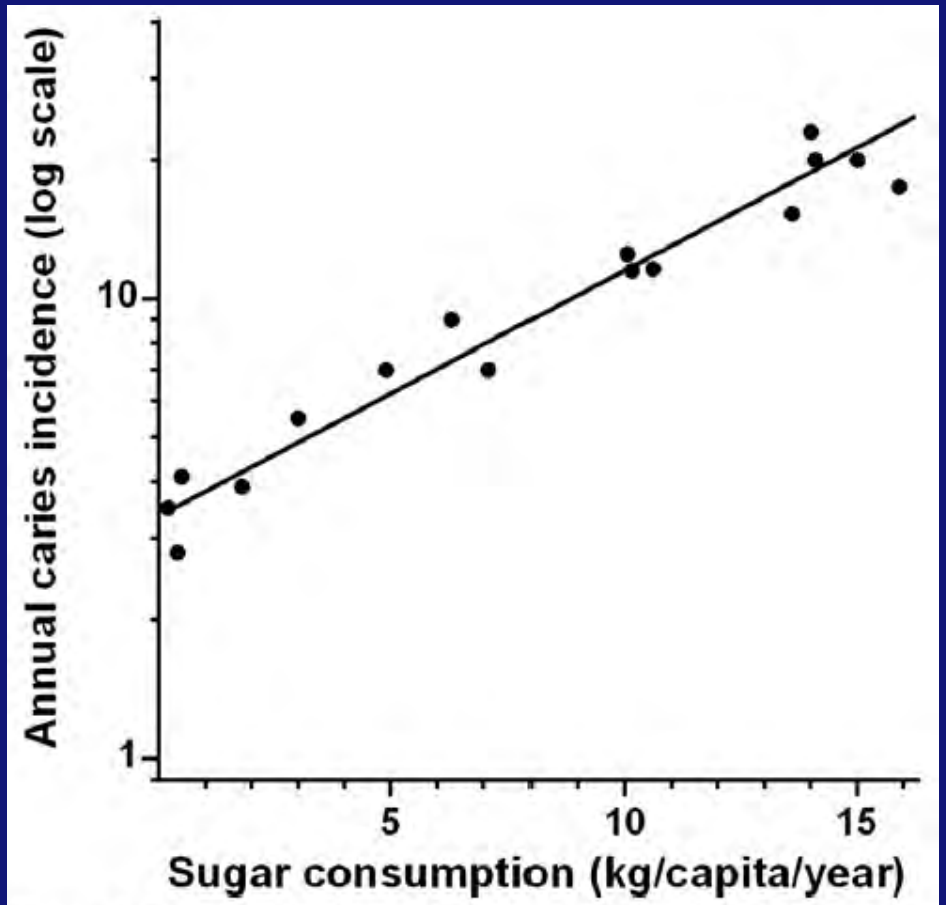
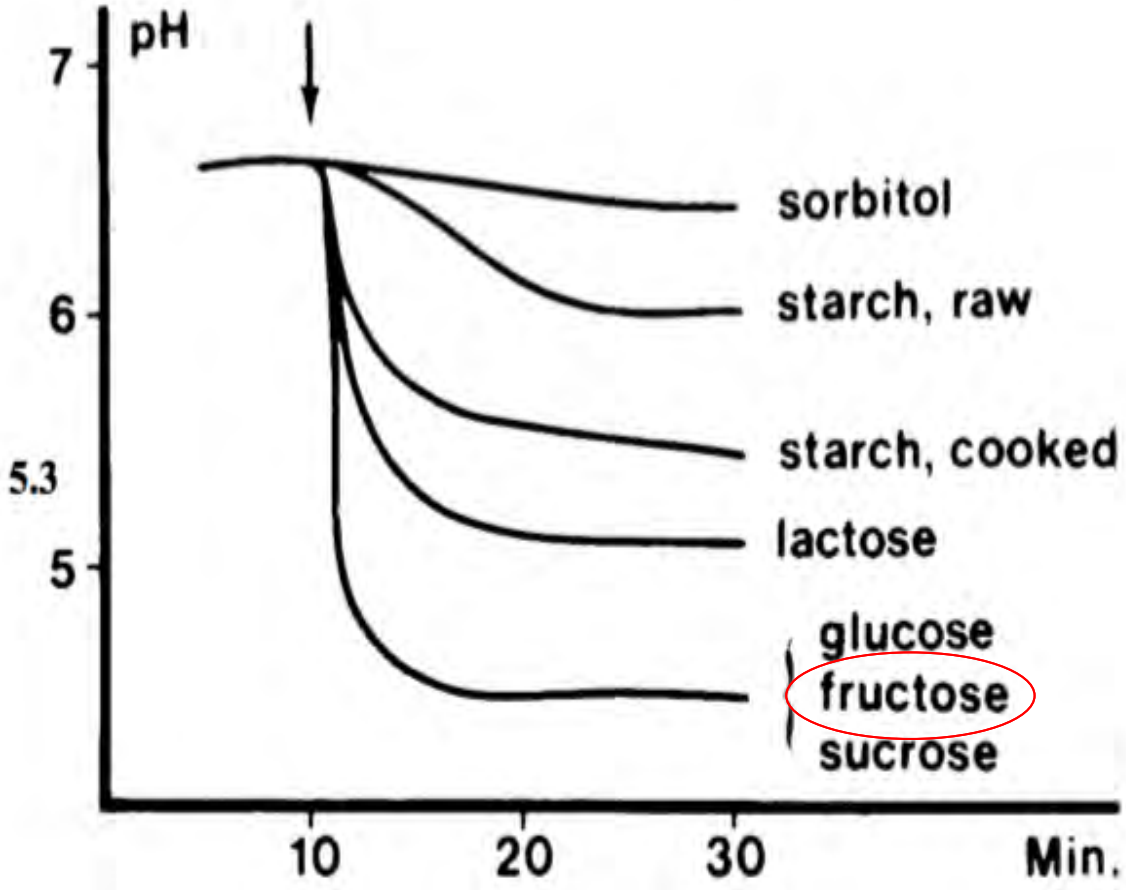


Figure 2 Relationship between annual per capita sugar consumption and annual caries incidence in lower first molar teeth. Data based on 10,553 Japanese children whose individual teeth were monitored yearly from the age of 6 to 11 years of age. Data plotted on a log scale. (Adapted from Koike [18]).

Sheiham and James, BMC Public Health 14:863, 2014

Stephan Curve

pH changes in plaque following application of different carbohydrate solutions



Starch vs. sucrose vs. both

Epidemiologic data:

Starch + low sugar → low incidence of caries

Starch + high sugar → high incidence of caries

Lingstrom et al., Crit Rev Oral Biol Med 11:366, 2000

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Table 1. Biomass (dry-weight), total amount of protein, and EPS in *Streptococcus mutans* UA159 biofilms formed in the presence of starch and sucrose, alone or in combinations

Experimental groups	Dry-weight (mg)	Total amount of protein (mg)	Total amount of EPS (μg)
Starch	0.75 (0.27)[1]	0.1 (0.08)[1]	88.98 (17.84)[1]
Starch + sucrose	6.25 (0.69)[2]	1.3 (0.12)[2]	1747.99 (146.62)[2]
Sucrose	5.50 (0.45)[3]	1.5 (0.32)[3]	1411.28 (256.45)[3]
Starch + glucose + fructose	1.25 (0.42)[1]	0.2 (0.12)[1]	126.37 (16.58)[4]
Sucrose + glucose	3.92 (0.92)[4]	1.4 (0.21)[2]	850.31 (190.79)[5]

Values (SD, $n = 12$) in the same column followed by the same superscript numbers are not significantly different from each other ($P > 0.05$, ANOVA, comparison for all pairs using Tukey test).

Duarte et al.,
Oral Micro Immunol 23:206, 2008

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Table 1. Analysis of dental biofilm pH according to treatment

(Mean values with their standard deviation and number of determinations)

	Treatment*											
	Water			Starch			Sucrose			Starch + Sucrose		
	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*
Baseline pH	7·5[a]	0·4	14	7·4[a]	0·5	15	6·6[b]	0·6	14	6·7[b]	0·8	15
$pH_{5\,min}$	7·2[a]	0·4	14	6·4[b]	0·7	13	5·3[c]	0·3	14	5·2[c]	0·3	14
cH^+ area (μmol/l × min)	0·1[a]	0·2	14	2·0[b]	2·0	13	18·3[c]	11·7	14	22·7[c]	13·3	14

$pH_{5\,min}$, pH at 5 min; cH^+ area, hydrogen ionic concentration between the times of 0 and 5 min (see text, p. 49 for details of procedures).
*T1, H_2O; T2, 2% starch; T3, 10% sucrose; T4, 2% starch + 10% sucrose.
[a,b,c] Mean values with unlike superscript letters were significantly different ($P<0.05$).

Table 4. Analysis of dental enamel according to treatment

(Mean values with their standard deviation and number of determinations)

	Treatment*											
	Water			Starch			Sucrose			Starch + Sucrose		
	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*
Mineral loss	447·9[a]	169·0	14	420·0[a]	160·1	15	955·6[b]	543·6	14	1421·8[c]	653·8	14

*T1, H_2O; T2, 2% starch; T3, 10% sucrose; T4, 2% starch + 10% sucrose.
[a,b,c] Mean values with unlike superscript letters were significantly different ($P<0.05$).

Duarte et al.,
Oral Micro Immunol 23:206, 2008

Ribeiro et al., Br J Nutr 94:44, 2005

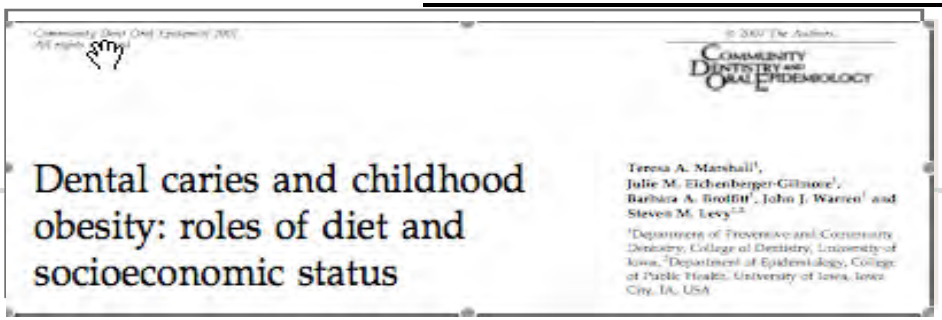
“Dentist Does Diet”

"It seems that were we to turn to a low sugar, high fat type of diet, such as is prescribed for diabetic patients, we might expect a prompt and marked reduction in caries susceptibility. This type of diet is practicable in many countries, but fats are in many regions considerably more expensive to produce than are starches and sugars. At any rate, we now know how to produce good teeth as respects structure and how to preserve them in considerable measure from decay. “

Elmer V. McCollum, Newer Knowledge of Nutrition, 1939


SIP ALL DAY
GET DECAY

CONSUMER PRICE INDEX



Price Index

300.0
250.0
200.0
150.0
100.0
50.0

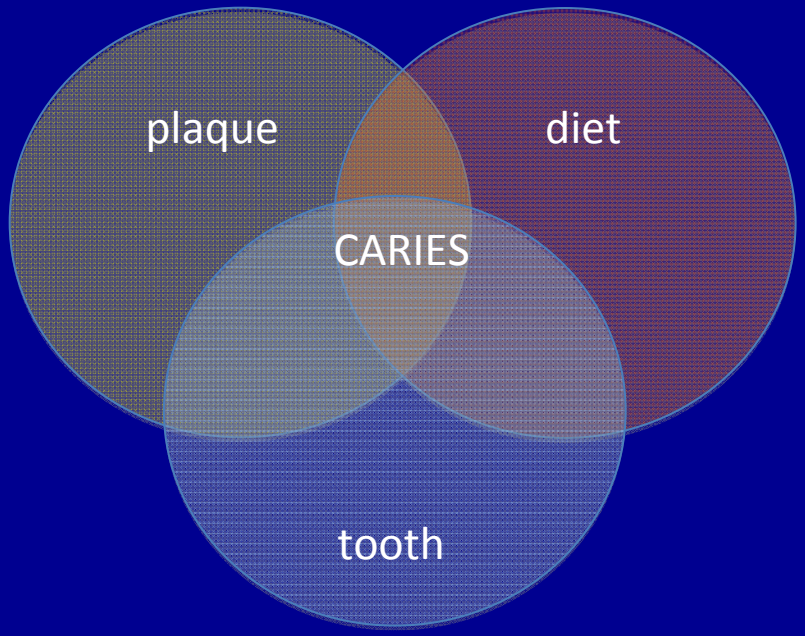
1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998 2000 2002

Hoping for a miracle

"We realize very well, however, that if sugar is the great offender in the cause of dental caries, as seems to be the case, we have a very difficult task ahead in making much progress in its control by the reduction of sugar intake so far as the mass of people is concerned. Most people would prefer some decay rather than to eliminate the sweets… We should keep up the admonition and give the evidence as to its harmful effect on teeth. At the same time, let us hope our research workers discover a more practical means of controlling or preventing dental decay."

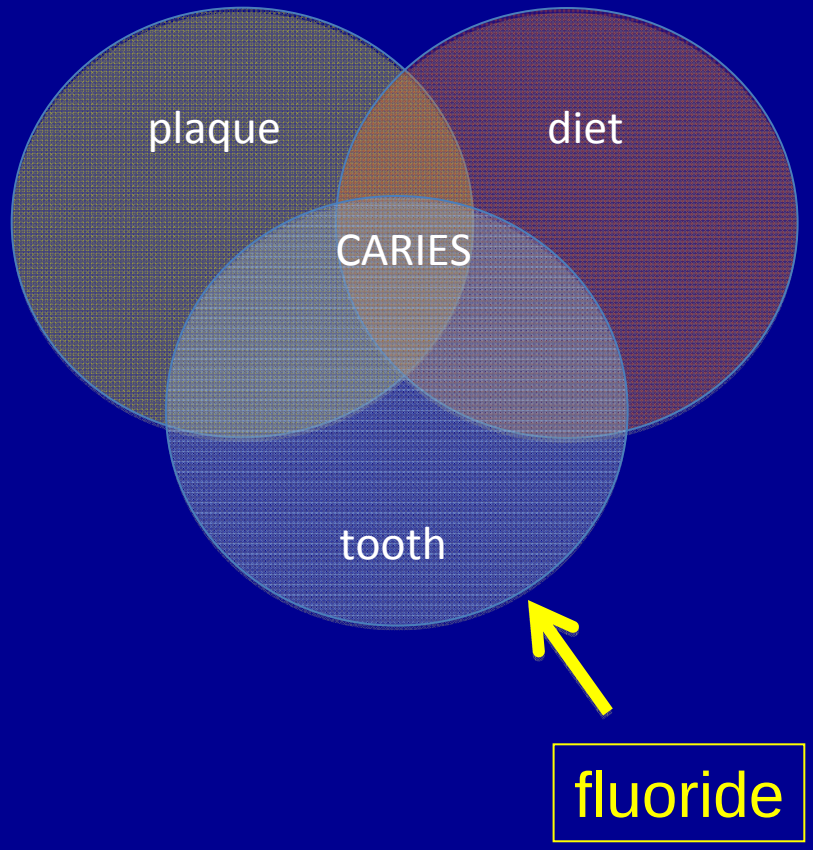
William Davis, Am J Public Health, 1941

The pathogenesis of caries 1947



Keyes and Jordan, 1963

The pathogenesis of caries 1947



Keyes and Jordan, 1963

Mechanisms of action of fluoride

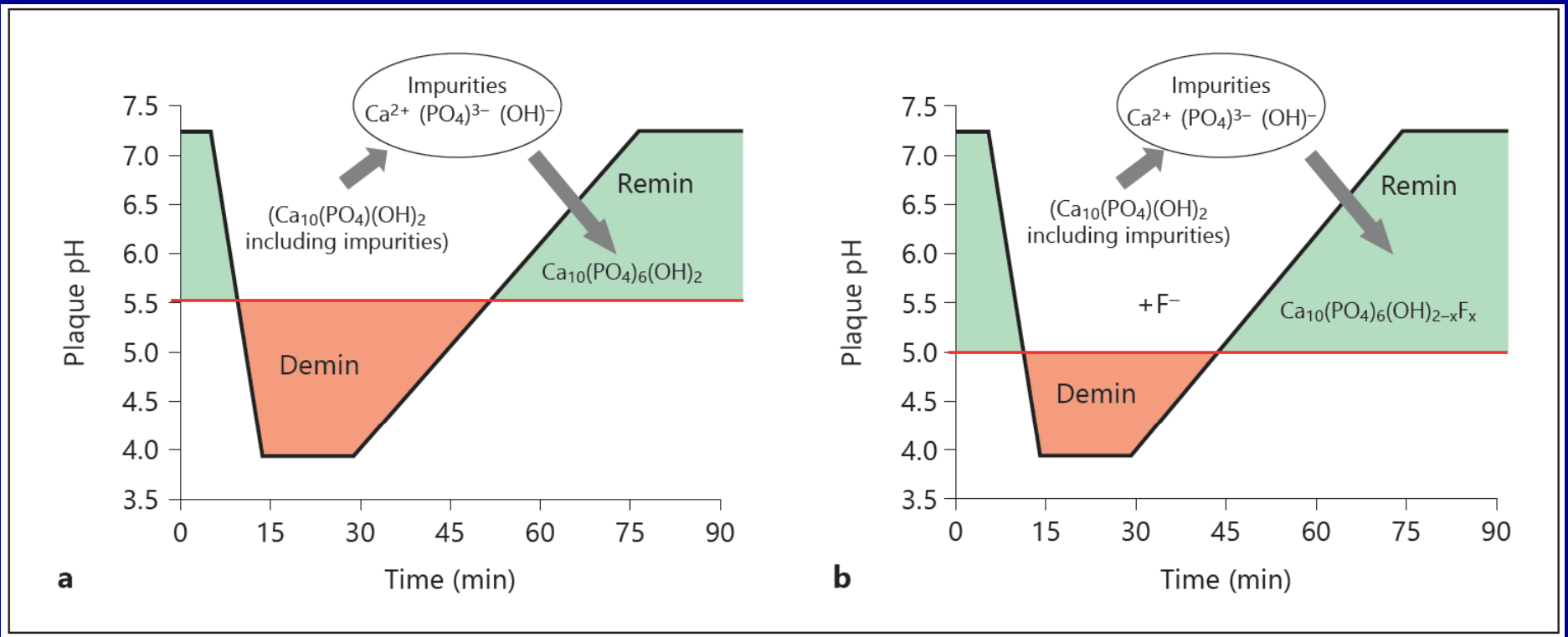


Fig. 1. Caries attack in the absence of fluoride (**a**) and in the presence of fluoride (**b**). In the presence of fluoride, the risk period (red area) is smaller than in the absence of fluoride as a result of a lower critical pH (pH 5.0 vs. 5.5). During remineralization, fluoridated hydroxyapatite is formed which is less soluble than the hydroxyapatite formed in the absence of fluoride.

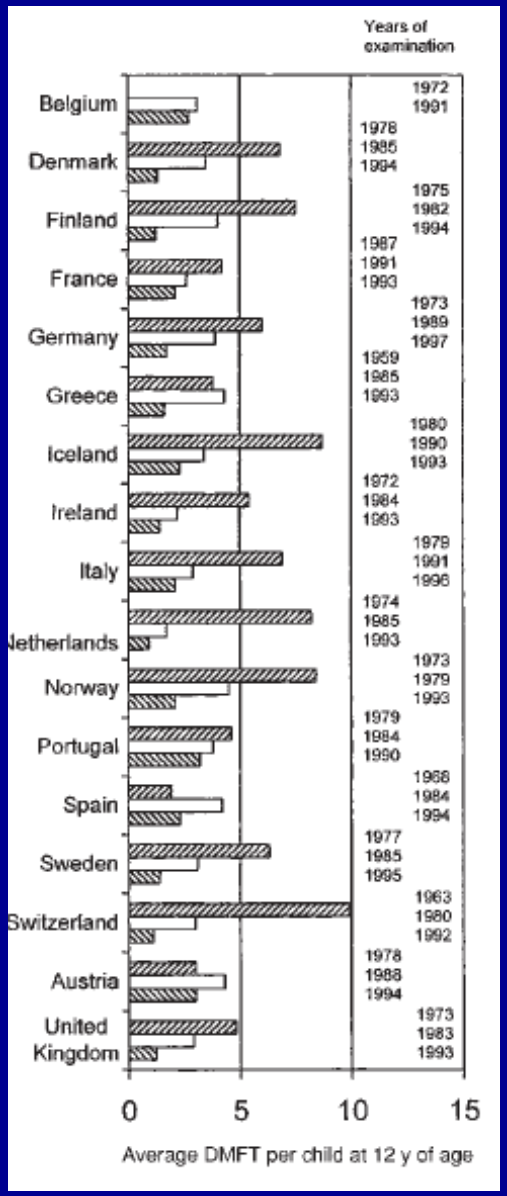
Amaechi and van Loveren, Monogr Oral Sci, Karger 2013, pp. 15-26

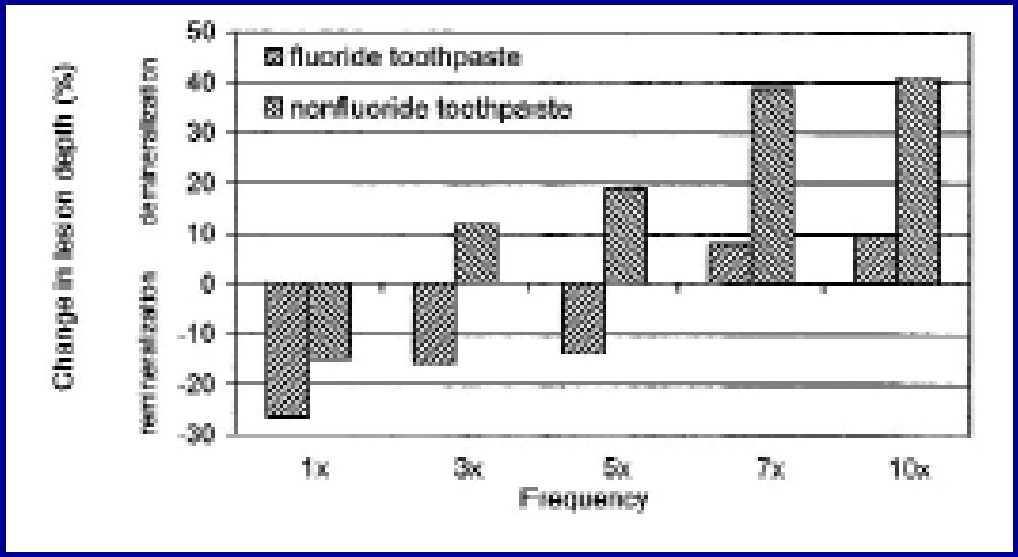
Water fluoridation becomes the standard

In 1945, Grand Rapids became the first city in the world to fluoridate its drinking water… During the 15-year project, researchers monitored the rate of tooth decay among Grand Rapids' almost 30,000 schoolchildren. After just 11 years, [Dr. H. Trendley] Dean - who was now director of the NIDR-announced an amazing finding. The caries rate among Grand Rapids children born after fluoride was added to the water supply dropped more than 60 percent. This finding, considering the thousands of participants in the study, amounted to a giant scientific breakthrough that promised to revolutionize dental care, making tooth decay for the first time in history a preventable disease for most people.

FJ McClure: Water Fluoridation, the Search and the Victory, NIDR, 1970

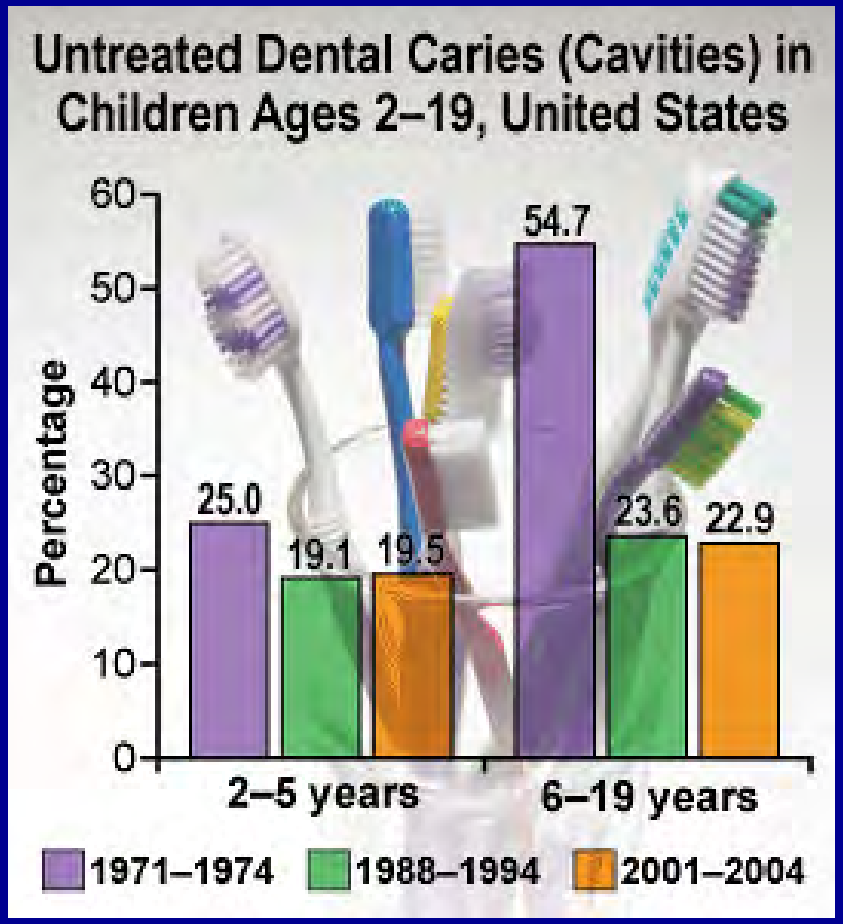
Fluoride in water or toothpaste cuts cavities





Touger-Decker and van Loveren, Am J Clin Nutr 78:881S, 2003

But we've reached an equilibrium – no further reduction in prevalence in caries



Untreated Dental Caries (Cavities) in Children Ages 2-19, by Sex, Race and Hispanic Origin, and Percent of Poverty Level, United States

	1971–1974	1988–1994	2001–2004
2–5 years			
Male	26.4%	19.3%	20.0%
Female	23.6%	18.9%	19.1%
6–19 years			
Male	54.9%	22.8%	23.9%
Female	54.5%	24.5%	22.0%
Race and Hispanic Origin			
2–5 years			
Not Hispanic or Latino			
White only	23.7%	13.8%	14.5%
Black or African American	29.0%	24.7%	24.2%
Mexican	—	34.9%	29.2%
6–19 years			
Not Hispanic or Latino			
White only	51.6%	18.8%	19.4%
Black or African American	71.0%	33.7%	28.1%
Mexican	—	36.5%	30.6%
Percent of Poverty Level			
2–5 years			
Below 100% of poverty level	32.0%	30.2%	26.1%
100%–less than 200%	29.9%	24.3%	25.4%
200% or more	17.8%	9.4%	12.1%
6–19 years			
Below 100% of poverty level	68.0%	38.3%	31.5%
100%–less than 200%	60.3%	28.2%	32.7%
200% or more	46.2%	15.1%	14.7%

Dye et al. NCHS, Vital and Health Statistics, National Health Survey, 2007

AAPD Leadership Perspective on the AAPD Foundation's Collaboration with the Coca-Cola Foundation (March 4, 2003)

A Brief Summary of Actions from AAPD Foundation President Joel H. Berg

…This commitment from the Coke Foundation is a large, unrestricted gift to the AAPD Foundation's endowment to fund independent research. Universities or other independent university-related entities selected by the AAPD (after a competitive process using an RFP-type protocol) will conduct the research. The gift does not involve endorsements, sponsorships or other relationships or affiliations. The Coca-Coca Foundation distributes millions of dollars annually to non-profit entities, including large grants to Habitat for Humanity and the Boys and Girls Clubs of America. We hope to leverage this interest on the part of the Coca-Cola Company and its Foundation in the oral health of children.

A Position Statement from AAPD Executive Director John S. Rutkauskas

The AAPD and AAPDF leadership firmly believes that this collaboration is in the best interest of children. Both AAPD members and parents should be assured that we have never and will never – endorse any consumer product from any corporate sponsor. That would not be in the best interests of the AAPD, parents or the children we serve. The Foundation's research topics and protocol and its choice of consumer education messages have always been chosen by its Board, comprised primarily of pediatric dentists. This is a donation from Coca-Cola's Foundation to our Foundation. We genuinely believe that we can make a big difference in promoting responsible choices for parents regarding their children's dental health and overall health.

HEALTH DIET/NUTRITION

The Sugar Industry Shaped Government Advice On Cavities, Report Finds

Alexandra Sifferlin @acsifferlin

March 10, 2015

Internal sugar industry documents reveal how it influenced national research priorities for tooth decay

Getty Images

A new report reveals that the sugar industry heavily influenced federal research—as well as the guidelines that resulted from that research.

Tooth decay remains a

PLOS | MEDICINE

RESEARCH ARTICLE

Sugar Industry Influence on the Scientific Agenda of the National Institute of Dental Research's 1971 National Caries Program: A Historical Analysis of Internal Documents

Cristin E. Kearns[1,2,3], Stanton A. Glantz[1,2,4,5]*, Laura A. Schmidt[1,2,6,7]

Portland, OR says “no” to fluoride

Guideline Summary

Guideline Title

Guideline on caries-risk assessment and management for infants, children and adolescents.

Bibliographic Source(s)

American Academy of Pediatric Dentistry (AAPD). Guideline on caries-risk assessment and management for infants, children and adolescents. Chicago (IL): American Academy of Pediatric Dentistry (AAPD); 2011. 8 p. [63 references]

Example of a Caries Management Protocol for 1-2 Year Olds

Risk Category	Diagnostics	Interventions		Restorative
		Fluoride	Diet	
Low risk	• Recall every 6-12 months • Baseline mutans streptococci (MS)[a]	• Twice daily brushing	Counseling	• Surveillance[e]
Moderate risk parent engaged	• Recall every 6 months • Baseline MS[a]	• Twice daily brushing with fluoridated toothpaste[b] • Fluoride supplements[d] • Professional topical treatment every 6 months	Counseling	• Active surveillance[c] of incipient lesions
Moderate risk parent not engaged	• Recall every 6 months • Baseline MS[a]	• Twice daily brushing with fluoridated toothpaste[b] • Professional topical treatment every 6 months	Counseling, with limited expectations	• Active surveillance[c] of incipient lesions
High risk parent engaged	• Recall every 3 months • Baseline and follow up MS[a]	• Twice daily brushing with fluoridated toothpaste[b] • Fluoride supplements[d] • Professional topical treatment every 3 months	Counseling	• Active surveillance[c] of incipient lesions • Restore cavitated lesions with interim therapeutic restorations (ITR)[f] or definitive restorations
High risk parent not engaged	• Recall every 3 months • Baseline and follow up MS[a]	• Twice daily brushing with fluoridated toothpaste[b] • Professional topical treatment every 3	Counseling, with limited expectations	• Active surveillance[c] of incipient lesions • Restore cavitated lesions with interim therapeutic restorations[f] or definitive restorations

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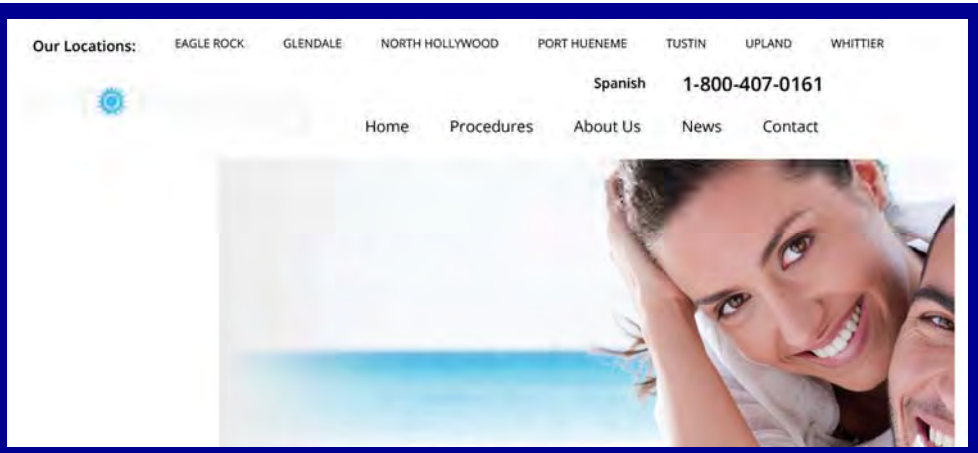
No dietary advice for any age group

CALIFORNIADENTAL group



IS HIGH SUGAR INTAKE AFFECTING YOUR ORAL HEALTH?
March 14, 2014

The World Health Organization may cut their recommended daily sugar intake in half. Should you follow their advice?

What Kind of Sugars Are You Consuming?

How Often Do You Indulge?

How Often Do You Brush?

"To sum up, while reducing overall sugar intake can help promote better oral health, it is not necessarily the most effective step to take. The best way to prevent tooth decay is to brush as quickly as possible after eating any kind of food, not just sugar."

Liver Decay

The Fiction

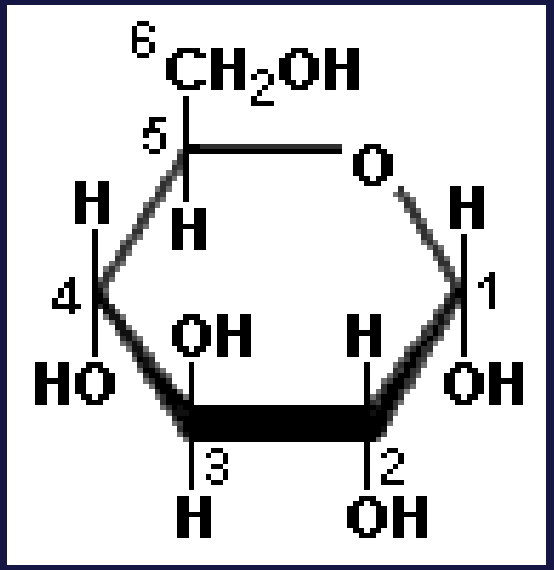
"Beating obesity will take action by all of us, based on one simple *common sense* fact: All calories count, no matter where they come from, including Coca-Cola and everything else with calories…"

-The Coca Cola Company, "Coming Together", 2013

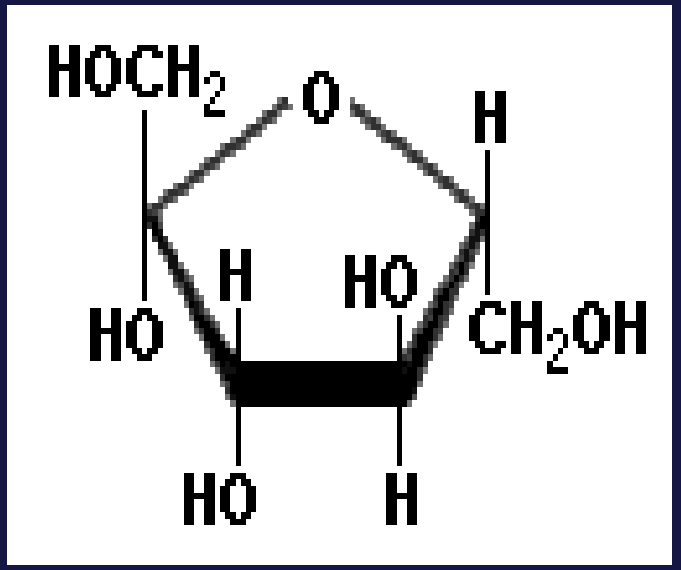
The Science

- Some Calories Cause Disease More than Others
- Different Calories are Metabolized Differently
- A Calorie is Not A Calorie
  - Fiber
  - Protein
  - Fat
  - Fructose

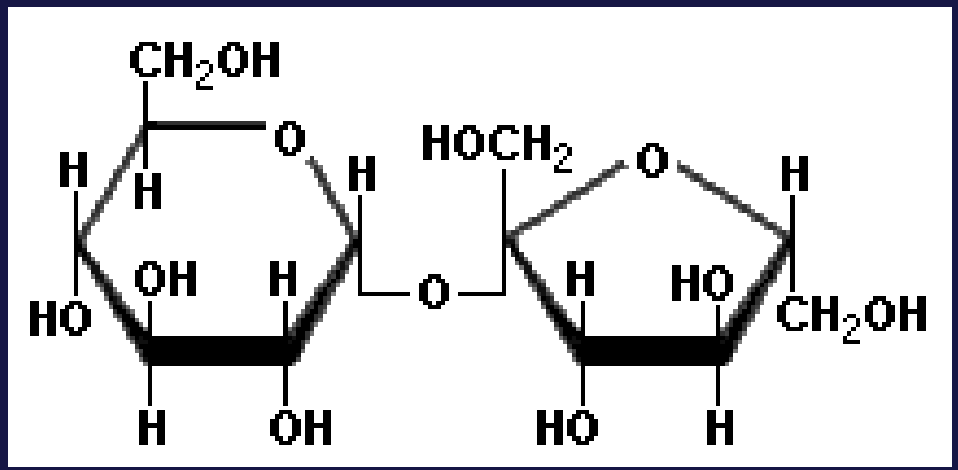
High Fructose Corn Syrup is 42-55% Fructose; Sucrose is 50% Fructose



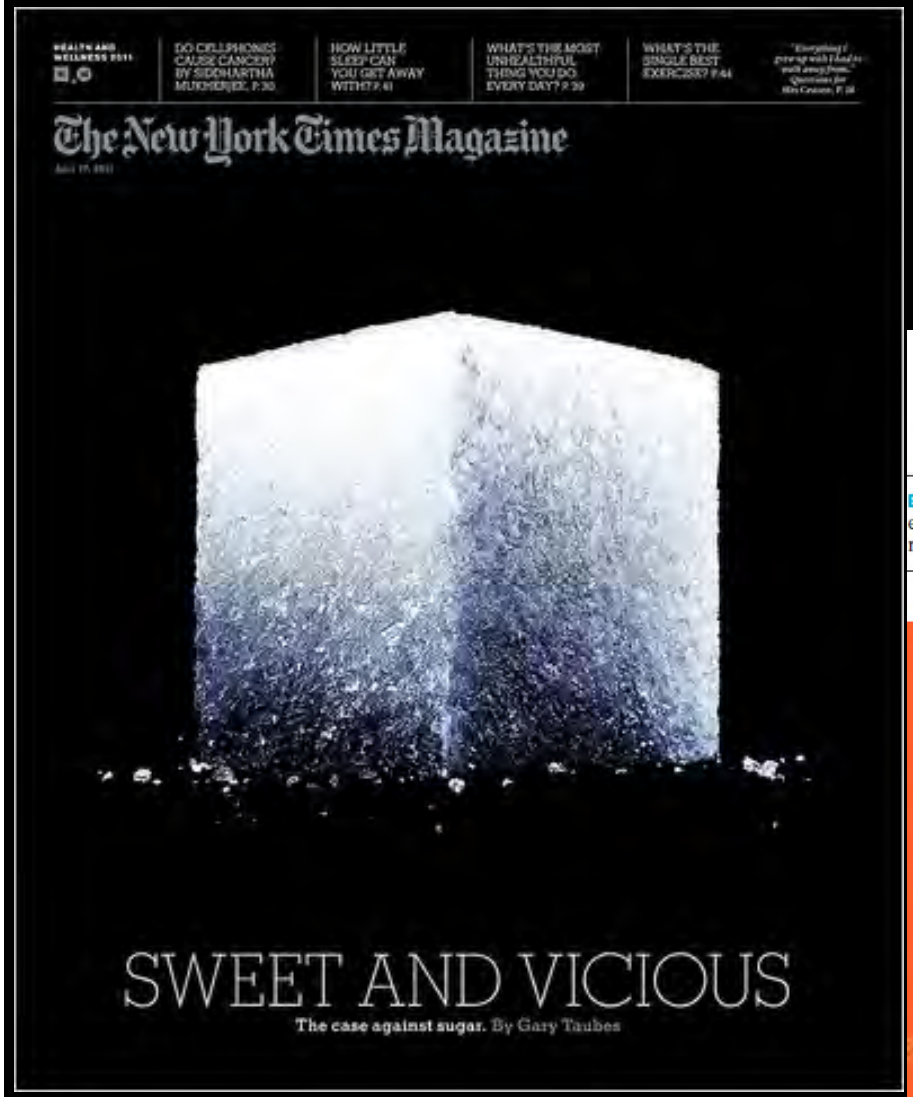
Glucose



Fructose



Sucrose



New York Times, April 17, 2011

Nature 487:27-29, Feb 1, 2012

COMMENT

ECOLOGY Komodo dragons and elephants could reduce fire risk in Australia **p.30**

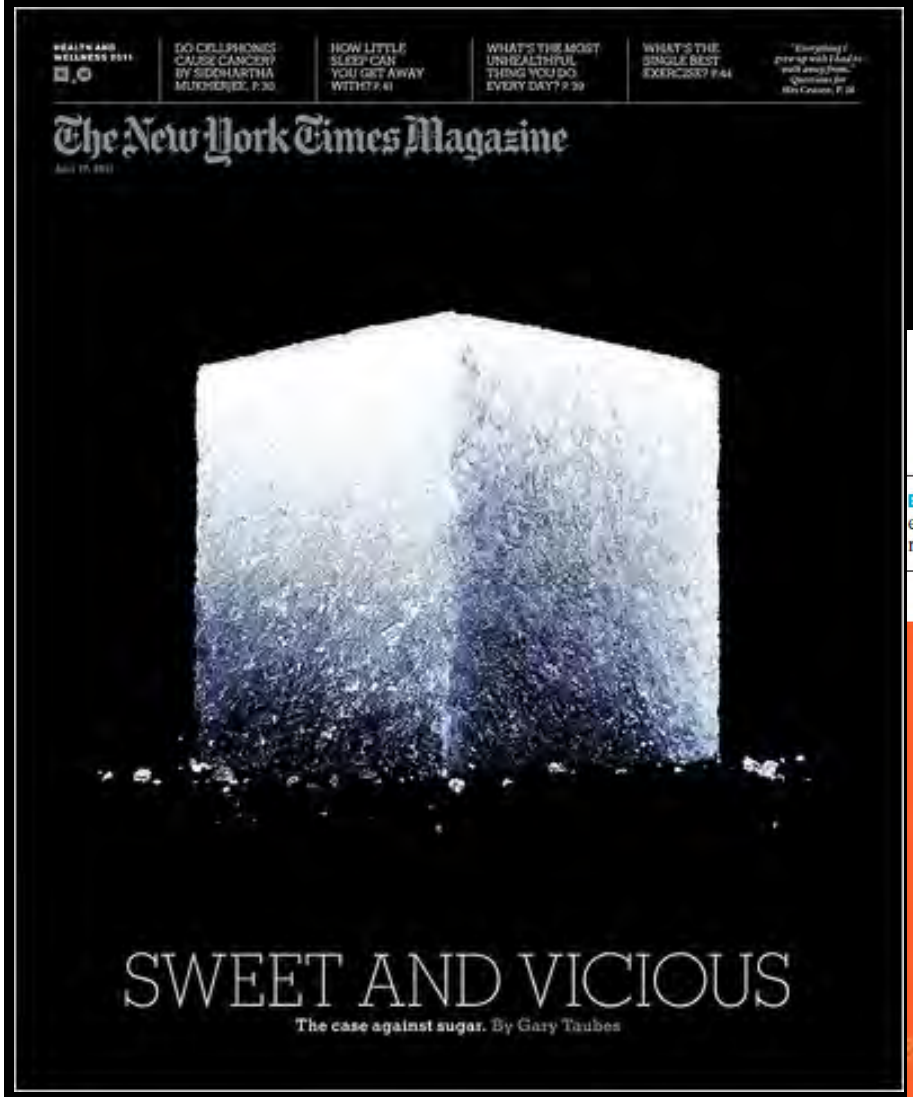
NEUROSCIENCE The source of the self is in the brain's wiring, not individual neurons **p.31**

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.



New York Times, April 17, 2011

Hyperbole?

Nature 487:27-29, Feb 1, 2012

Toxicity:

The degree to which a substance can damage an organism

- Does not distinguish acute vs. chronic toxicity

Requisites:

- Must be an “independent risk factor”
- Must establish causation
- Exclusive of calories
- Exclusive of obesity

Criticisms of Fructose Toxicity

- Animal models, not human studies
- Administration of excessive doses of fructose

Criticisms of Fructose Toxicity

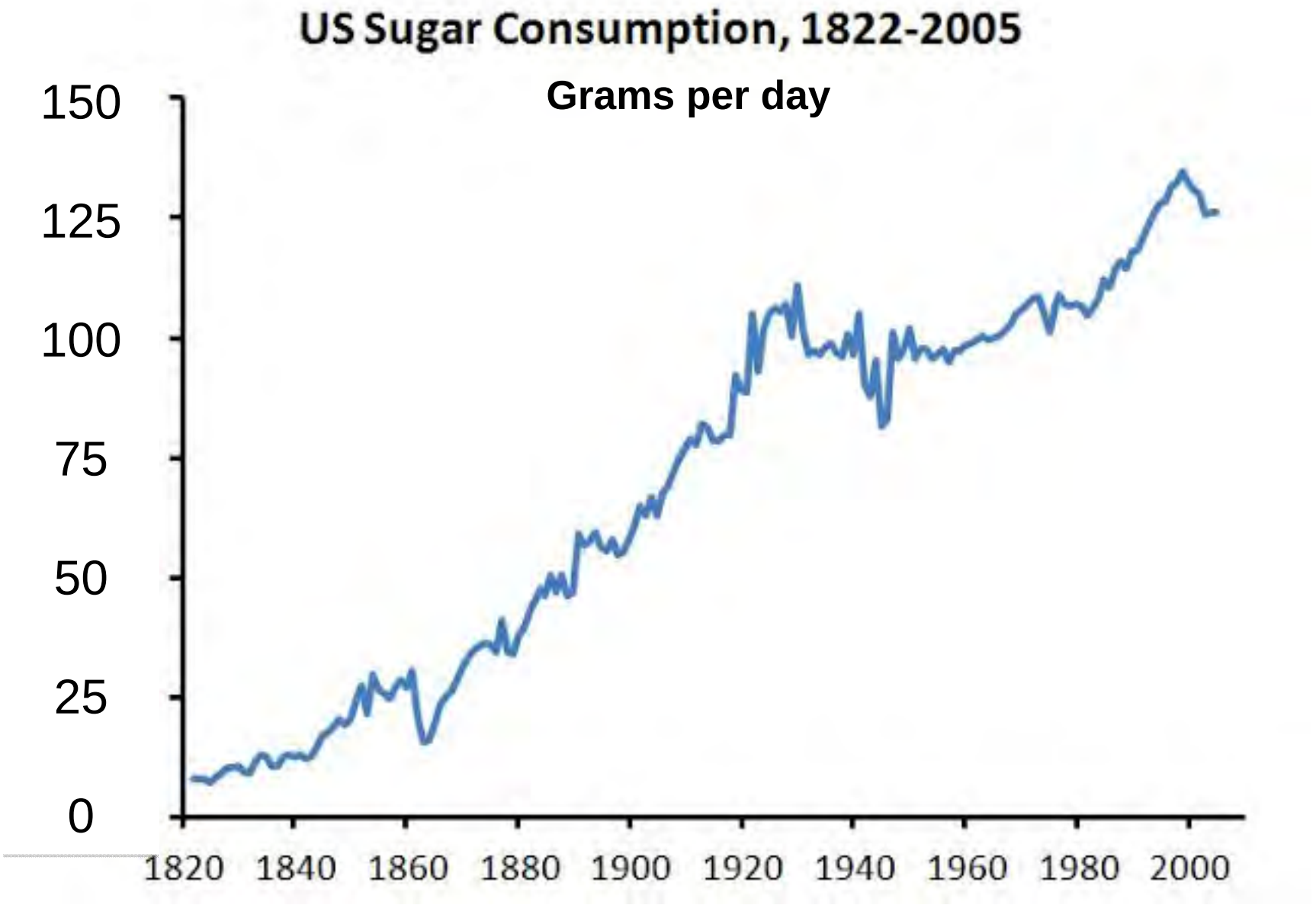
- Animal models, not human studies
- Administration of excessive doses of fructose

WILL LIMIT DISCUSSION TO:

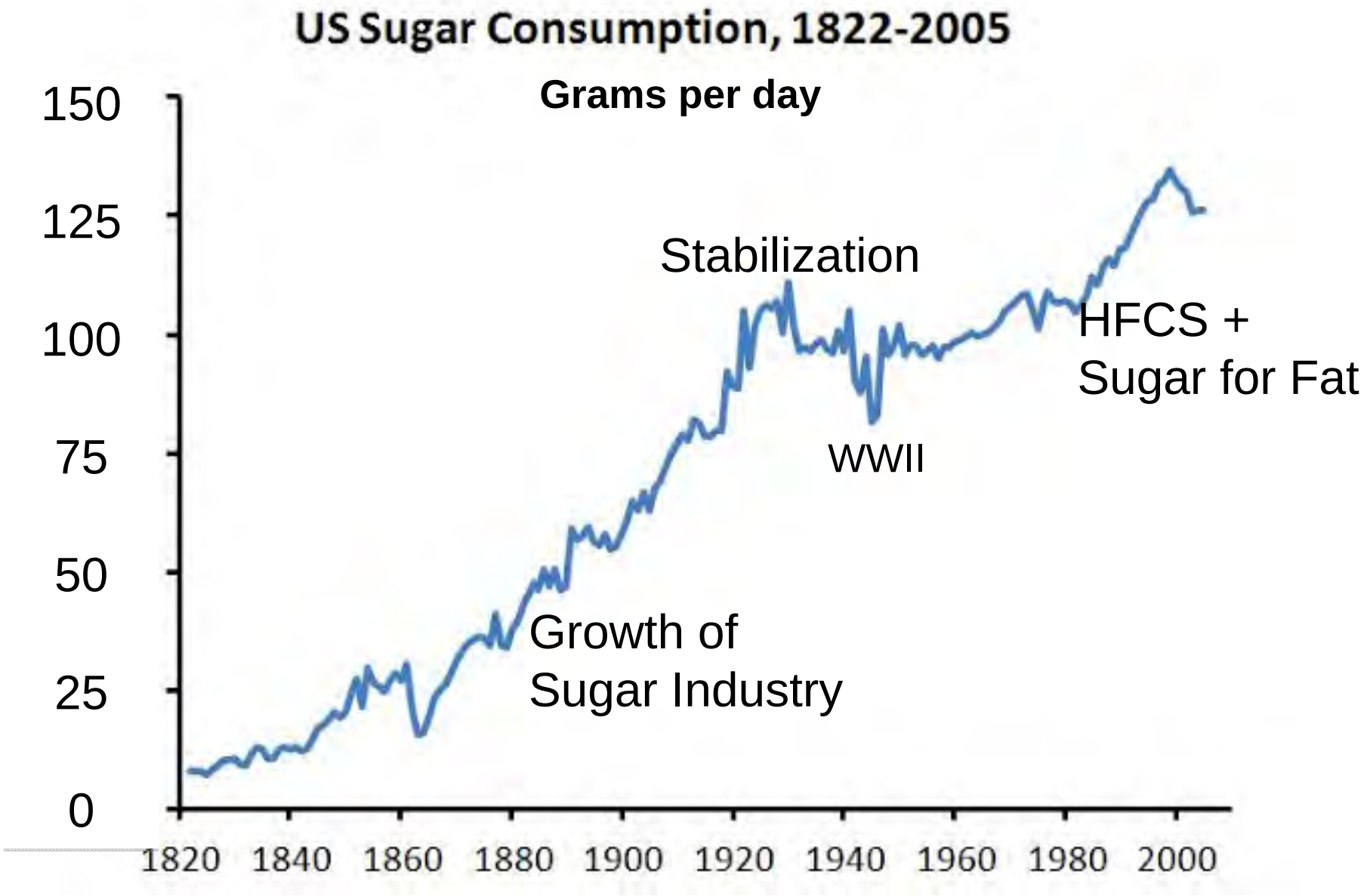
HUMAN DATA,

HUMAN CONSUMPTION,

AND IN DOSES ROUTINELY INGESTED



U.S. Commerce Service 1822-1910, combined with Economic Research Service, USDA 1910-2010



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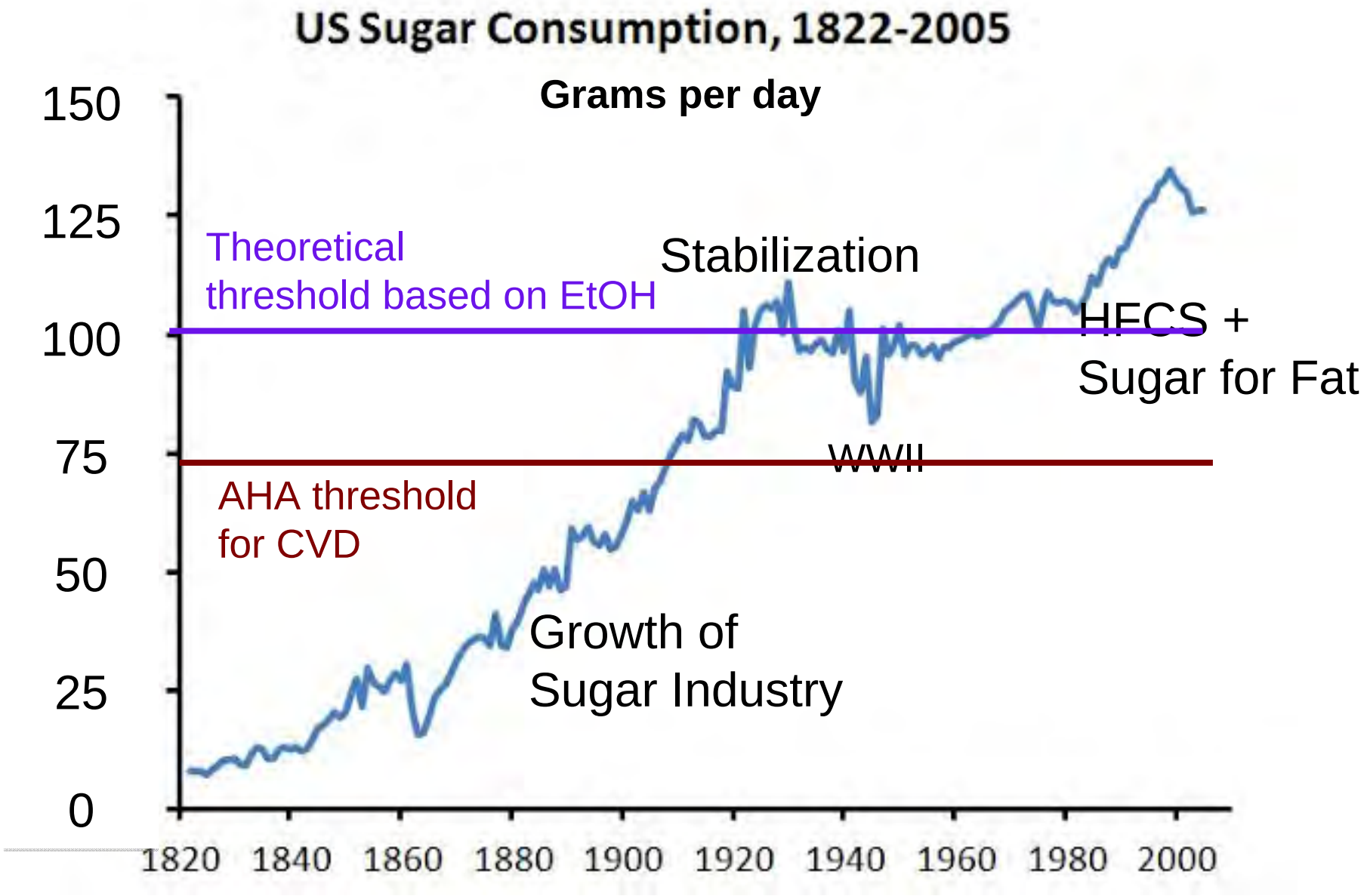

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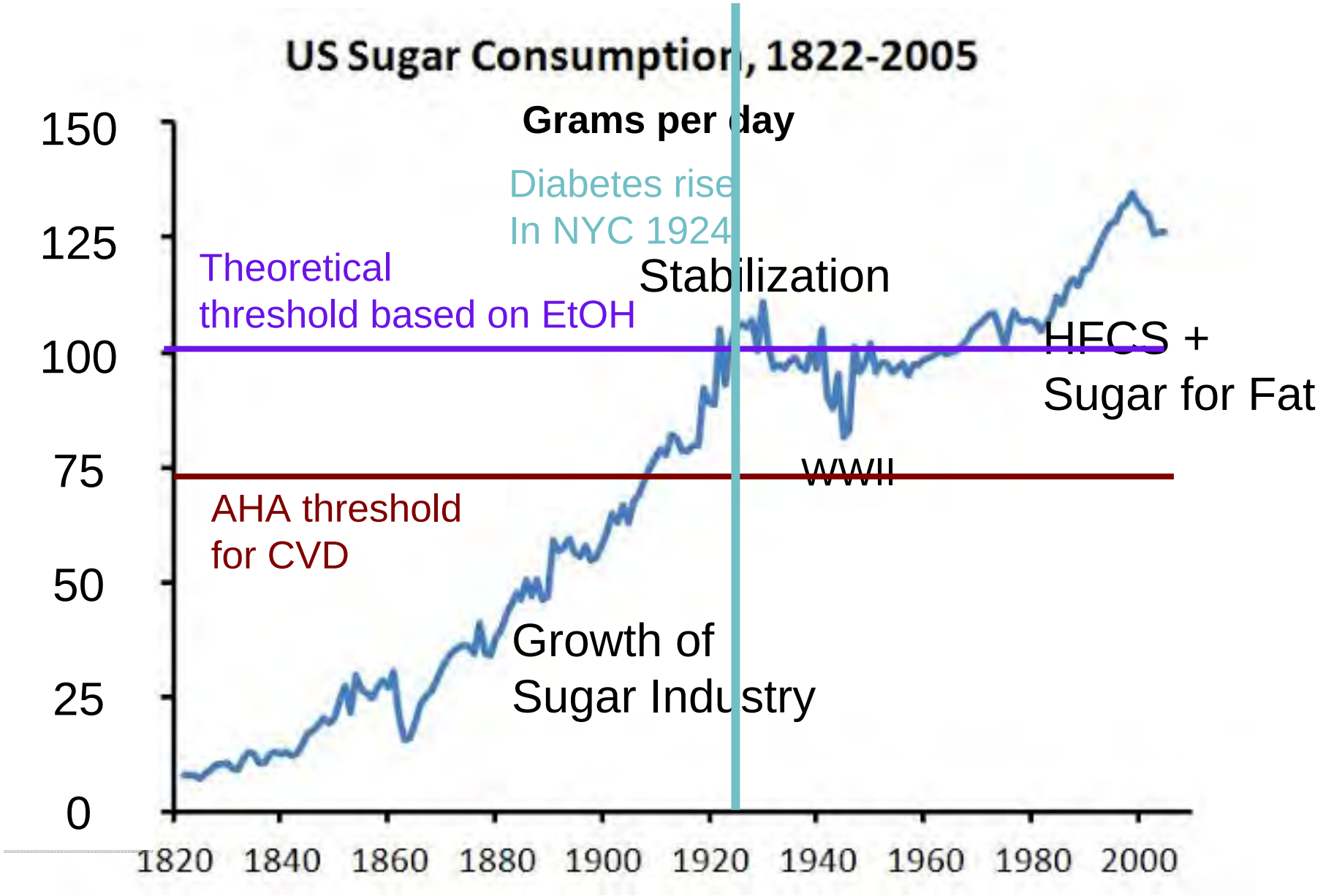
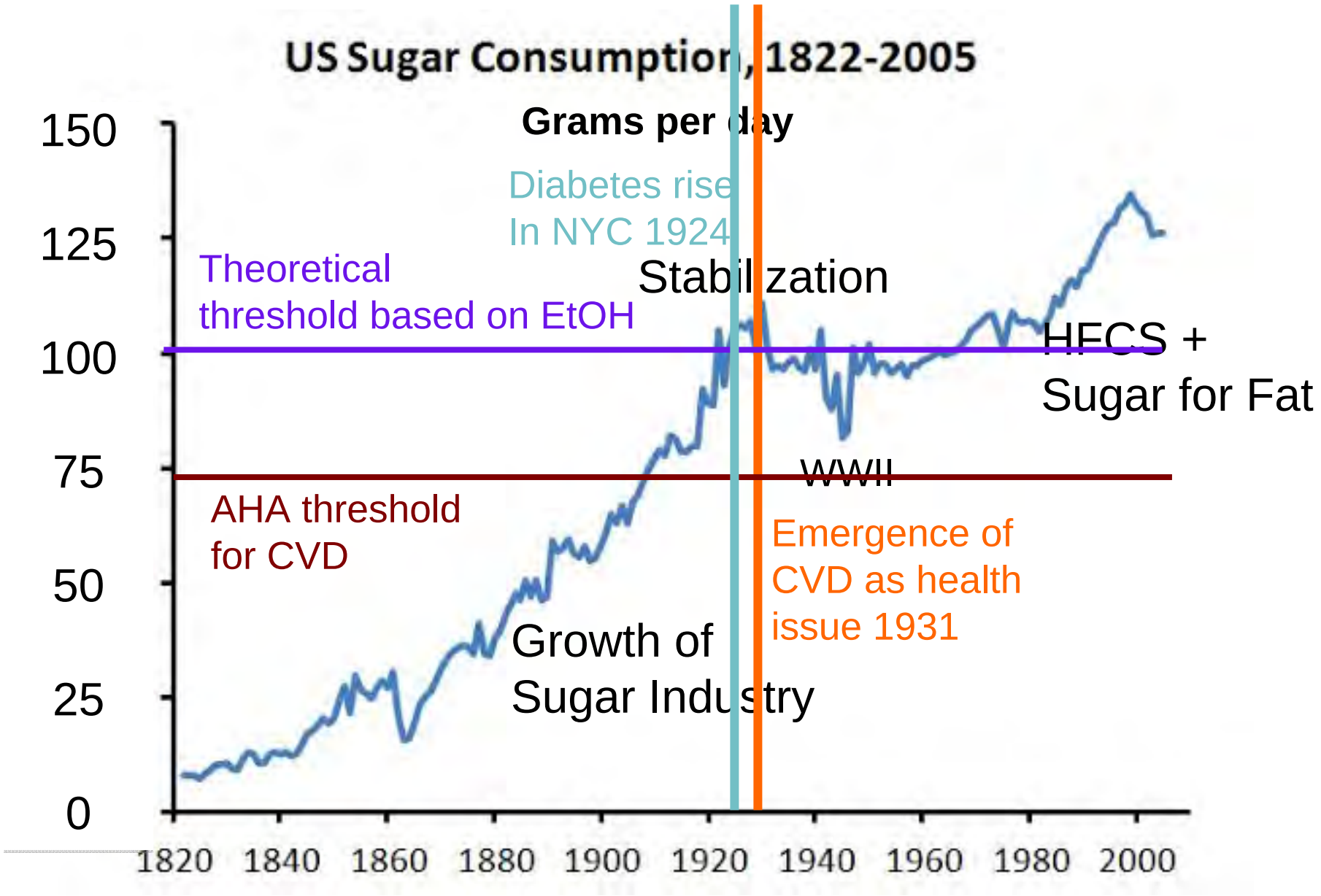
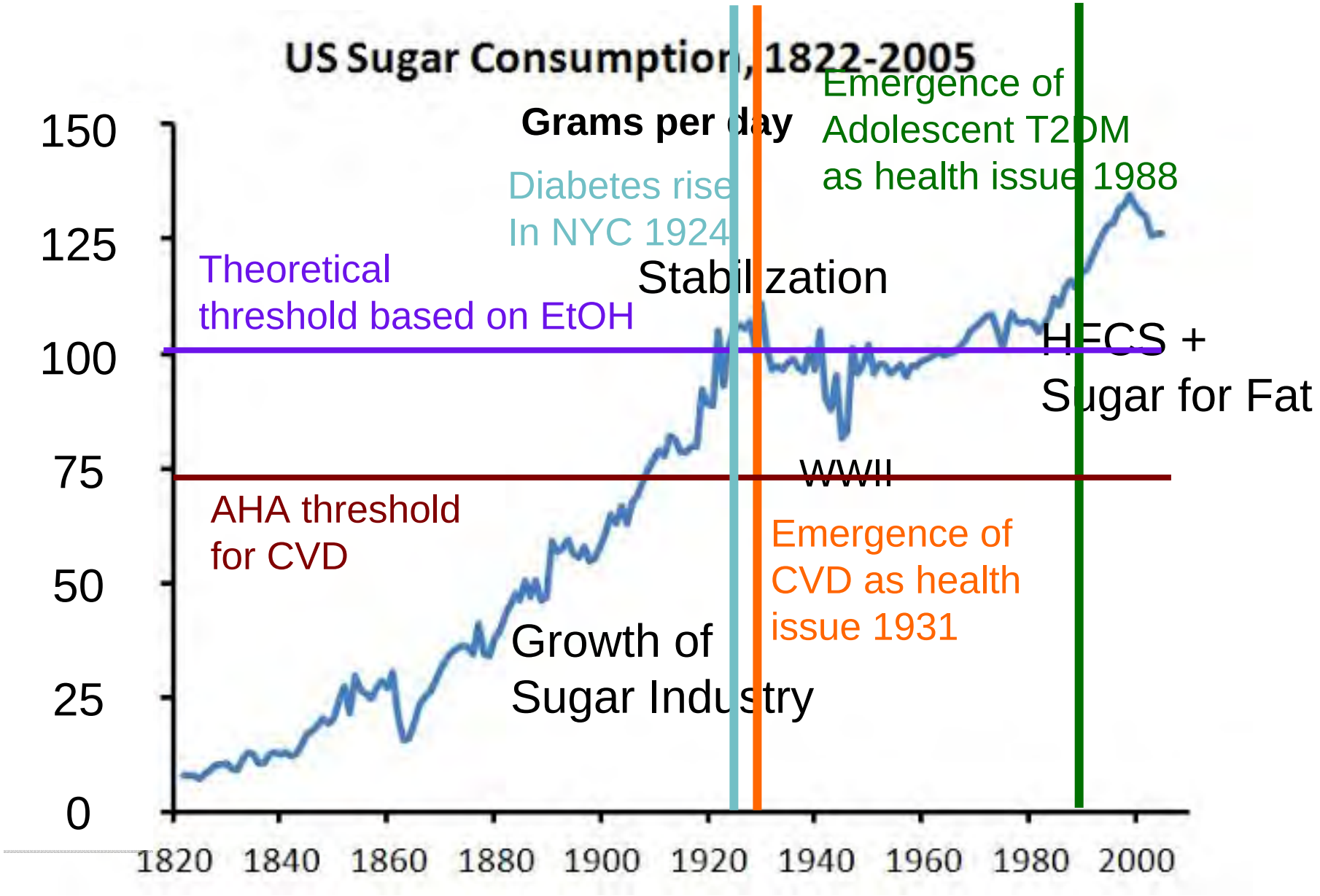


U.S. Commerce Service 1822-1910, combined with Economic Research Service, USDA 1910-2010



U.S. Commerce Service 1822-1910, combined with Economic Research Service, USDA 1910-2010



U.S. Commerce Service 1822-1910, combined with Economic Research Service, USDA 1910-2010

Sugar and Heart Disease

Variation of HDL and triglyceride levels based on consumption of added sugars in NHANES adults

Figure 1. Multivariable-Adjusted Mean HDL-C Levels by Level of Added Sugar Intake Among US Adults, NHANES 1999-2006

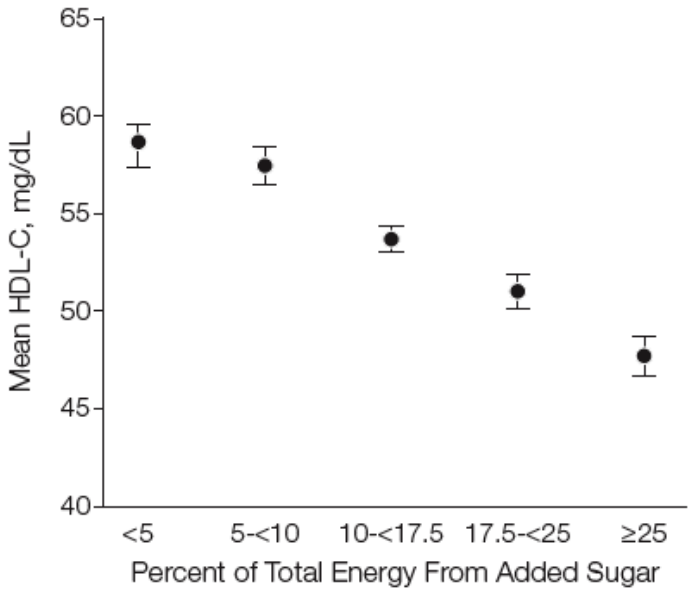
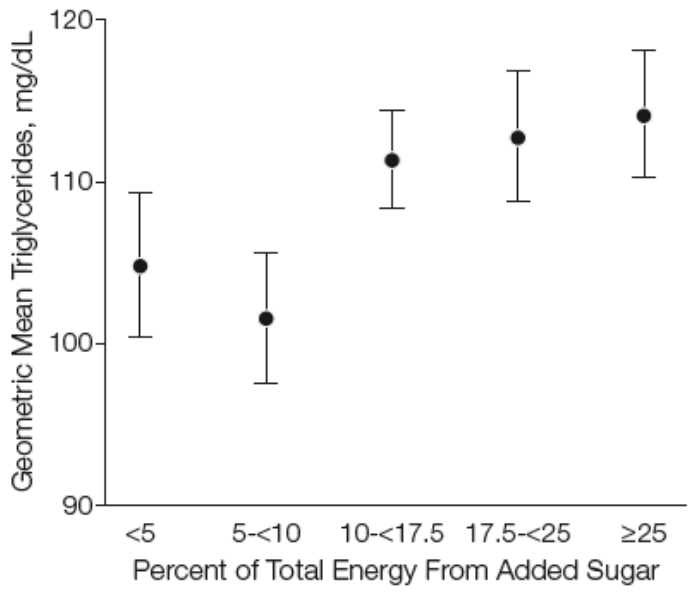
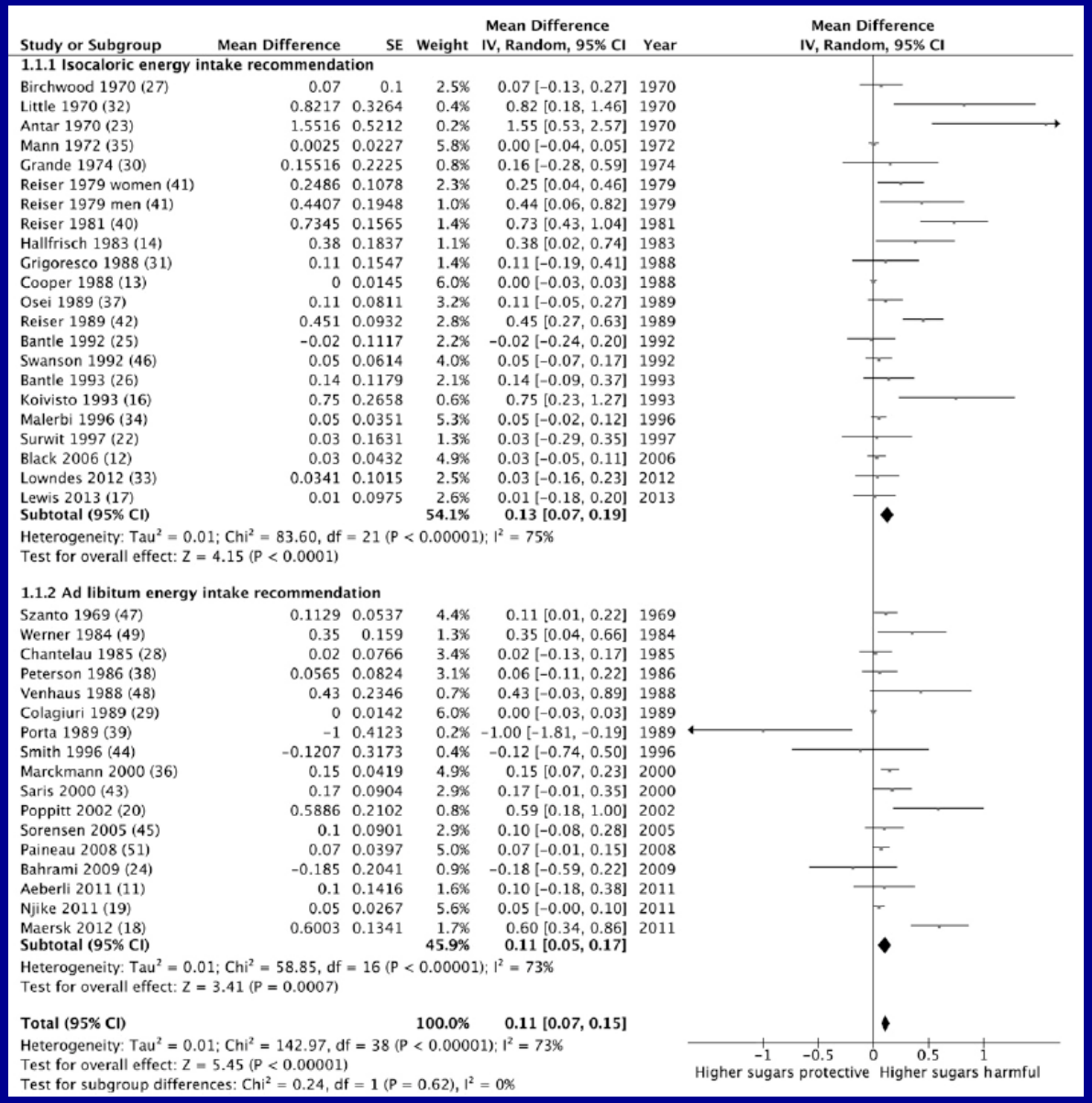


Figure 2. Multivariable-Adjusted Geometric Mean Triglyceride Levels by Level of Added Sugar Intake Among US Adults, NHANES 1999-2006



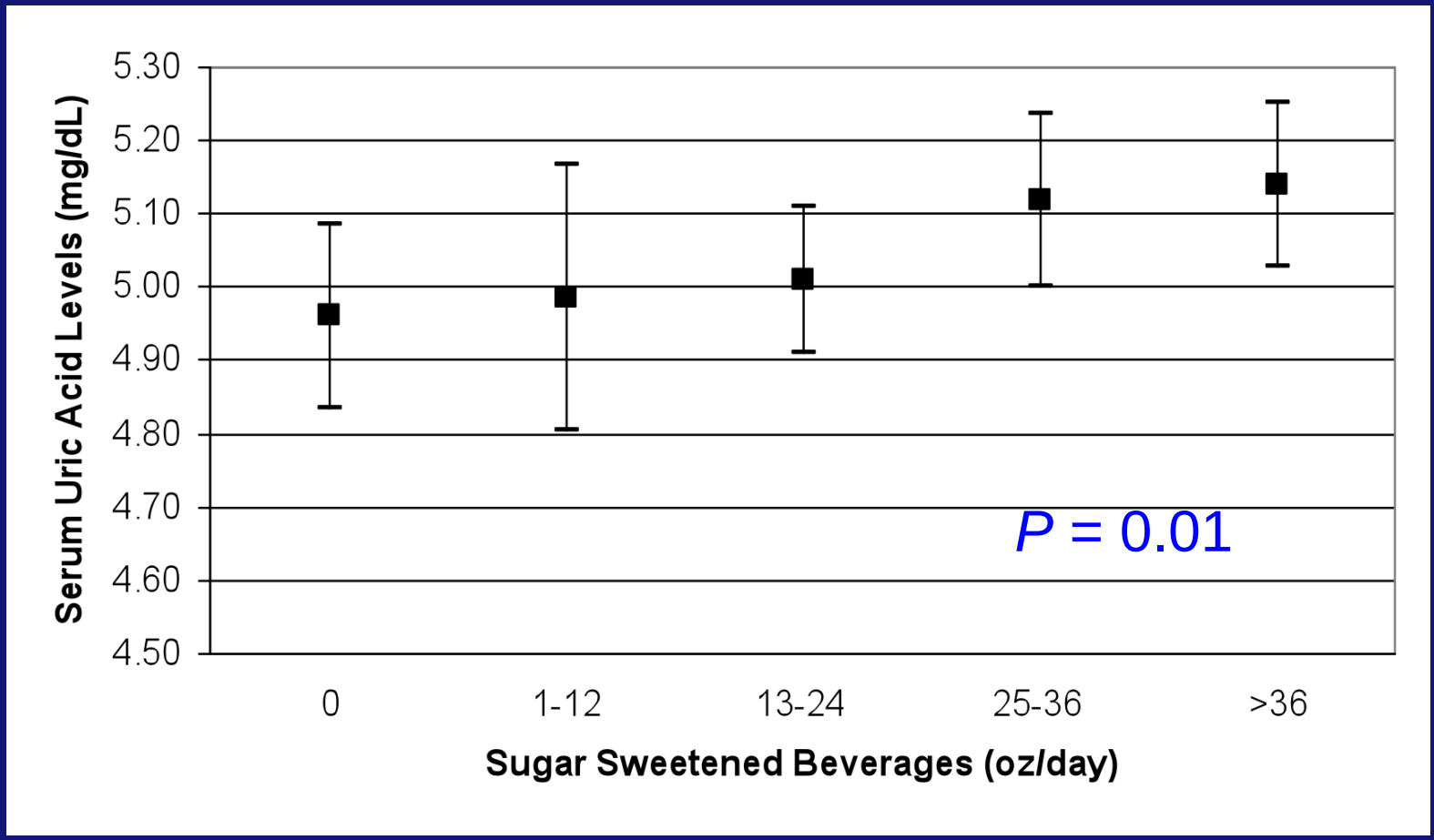
Welsh et al. JAMA 303:1490, 2010

Meta-Analysis of Effects of Sugar on Triglycerides



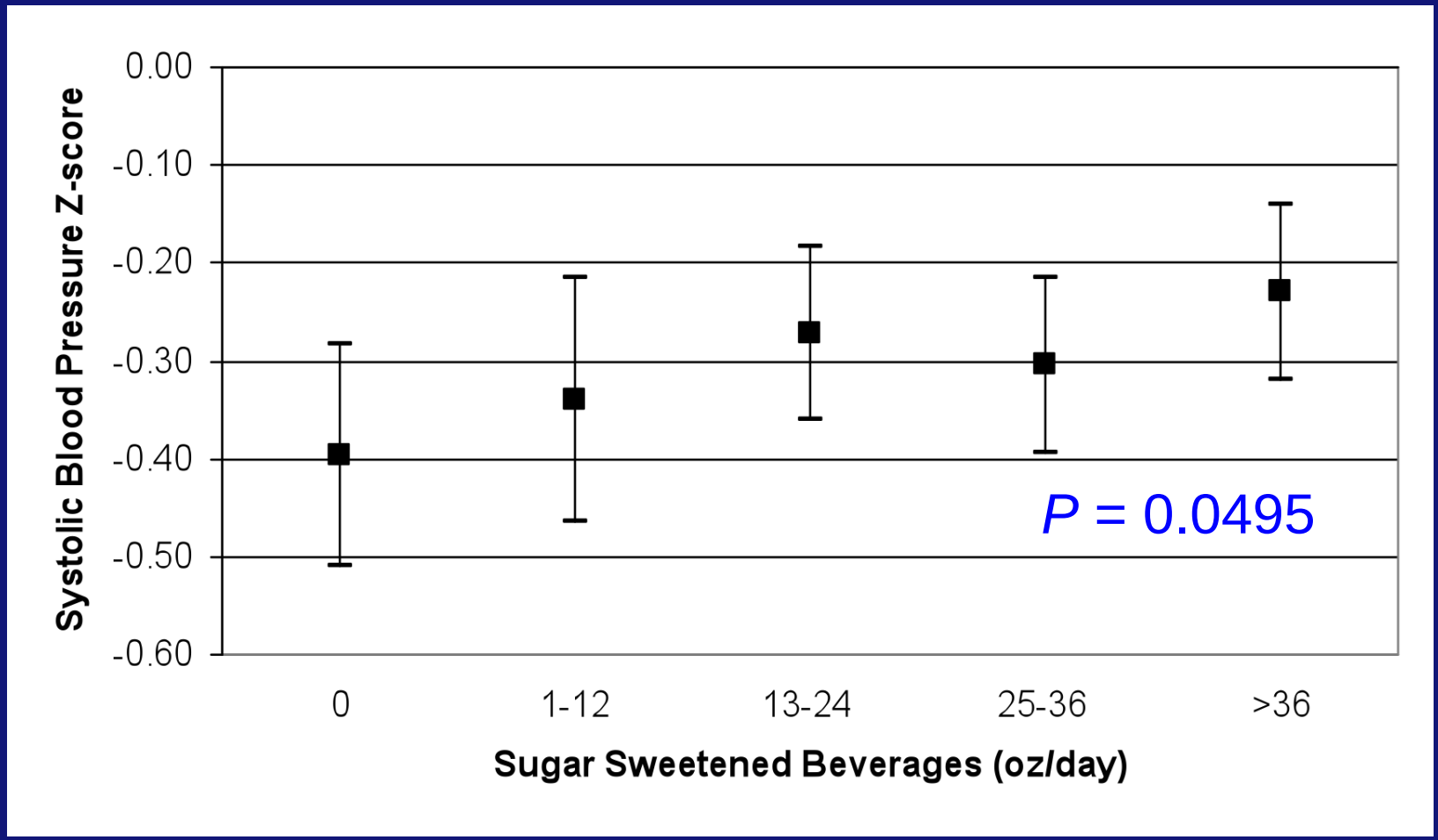
Te Morenga et al. Am J Clin Nutr doi: 10.3945/ajcn.113.081521, May 7, 2014

Relations between fructose, uric acid and hypertension in NHANES IV adolescents



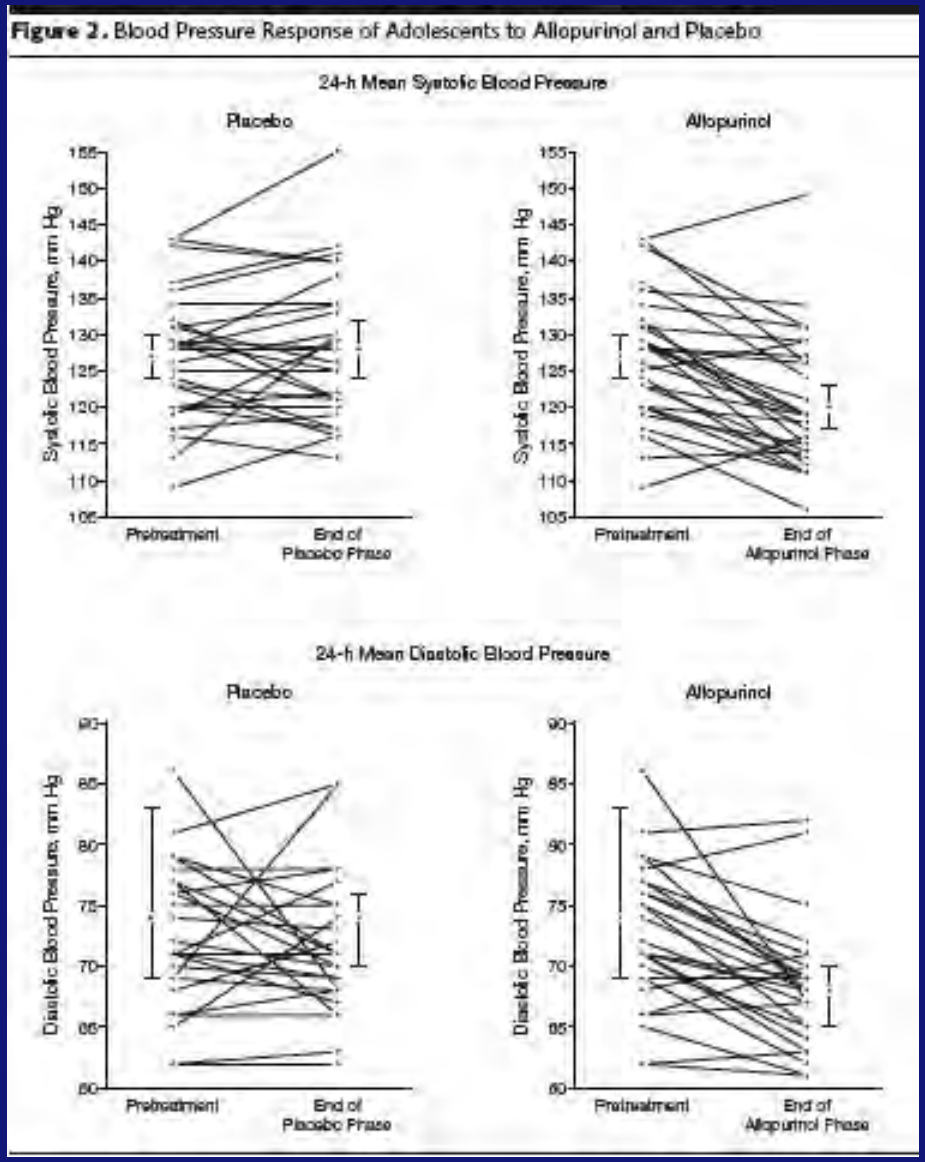
S Nguyen et al. J Pediatr 154:806, 2009

Relations between fructose, uric acid and hypertension in NHANES IV adolescents



S Nguyen et al. J Pediatr 154:806, 2009

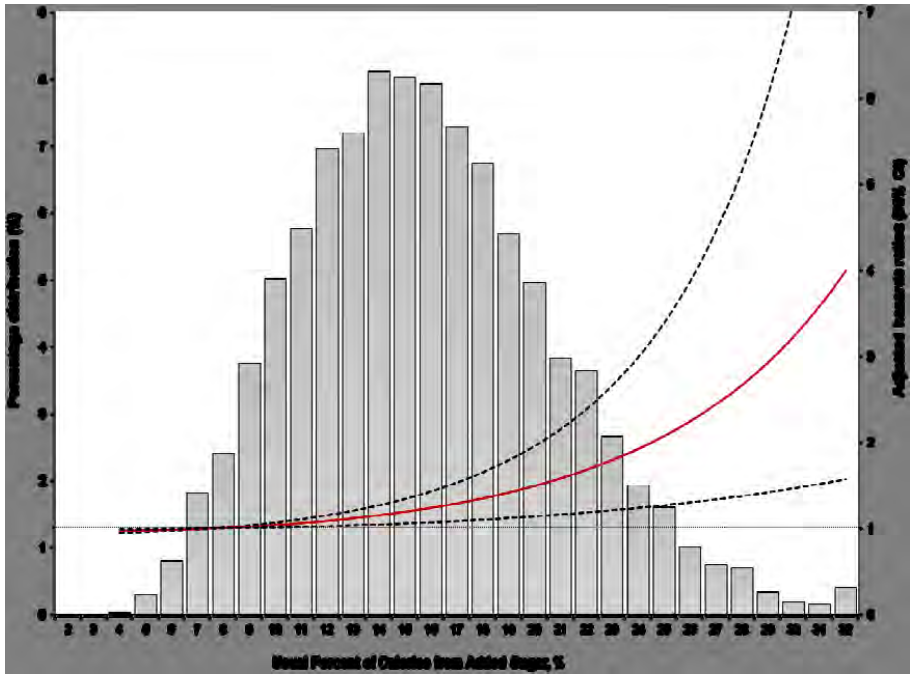
Allopurinol lowers BP in obese adolescents with essential hypertension



Feig et al. JAMA 300:924, 2008

Hazard ratio for CV mortality based on percent calories as sugar for US adult population, 1988-2006

Figure 1. Adjusted Hazard Ratio of the Usual Percent of Calories from Added Sugar for CVD Mortality Among US Adults Aged ≥20 Years – NHANES Linked Mortality Files, 1988-2006



Histogram is the distribution of usual percent of calories from added sugar in population. Lines show the adjusted HRs from Cox models. Mid-value of quintile 1 (7.5%) was the reference standard. Model was adjusted for age, sex, race/ethnicity, educational attainment, smoking status, alcohol consumption, physical activity level, family history of CVD, antihypertensive medication use, health eating index score, body mass index, systolic blood pressure, total serum cholesterol and total calories. Solid line indicates point estimates; dashed lines indicate 95% CIs. CVD indicates cardiovascular disease; HR, hazard ratio; NHANES, National Health and Nutrition Examination Survey.

Yang et al. JAMA Int. Med epub Feb 3, 2014

Sugar and Diabetes

— Confound by Obesity

— Plausibility

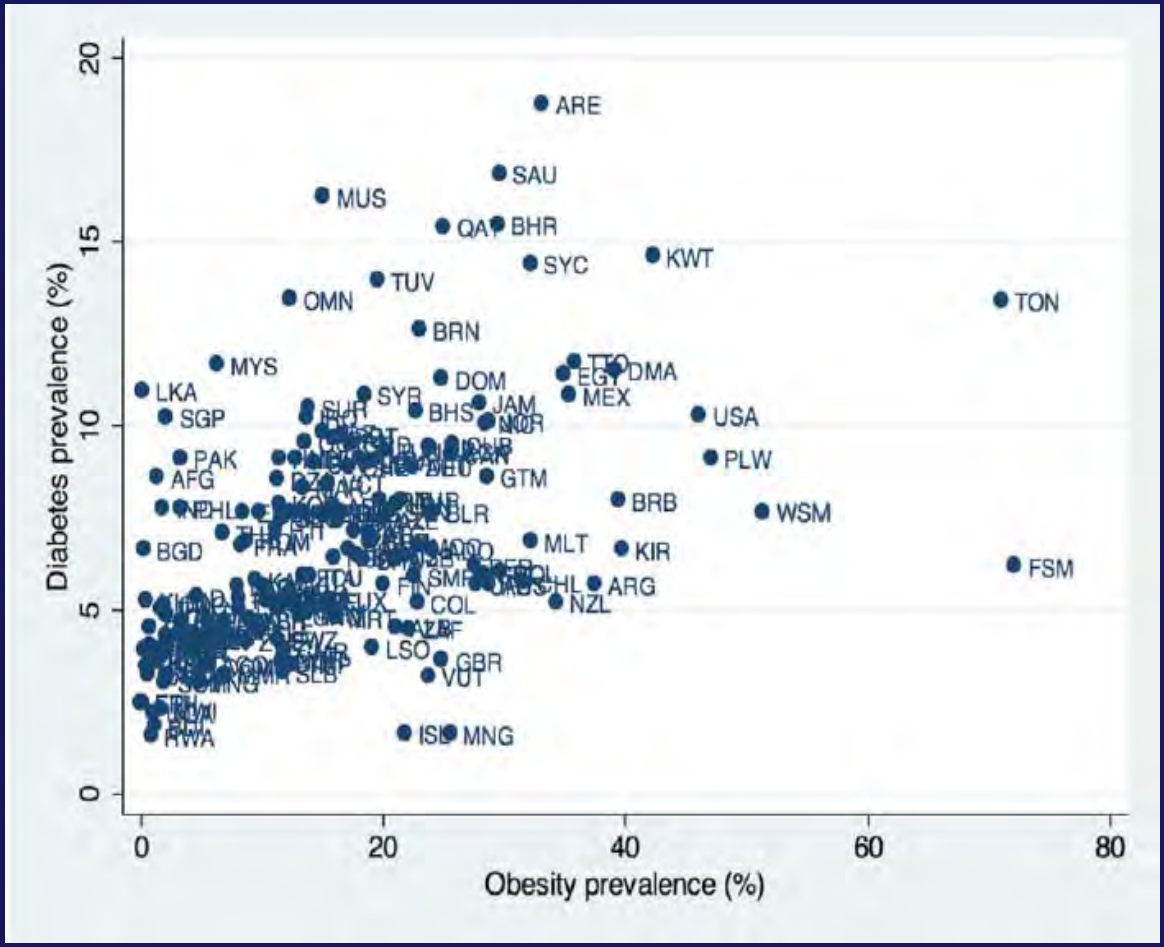
— Mechanisms

— Human Correlation

— Human Causation

Sugar and Diabetes: Confound by Obesity

Obesity is the problem (?)



Basu et al. PLoS One 8:e58783, 2013

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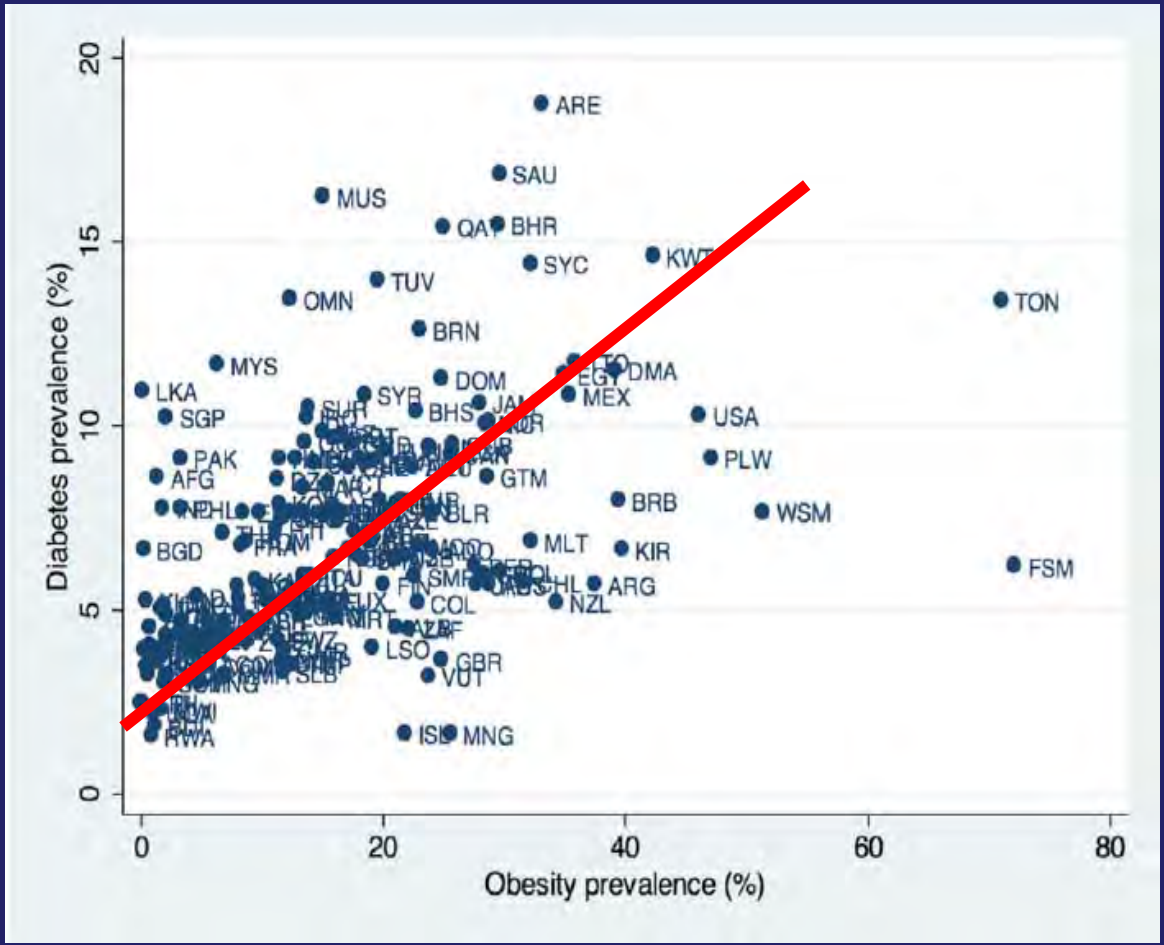

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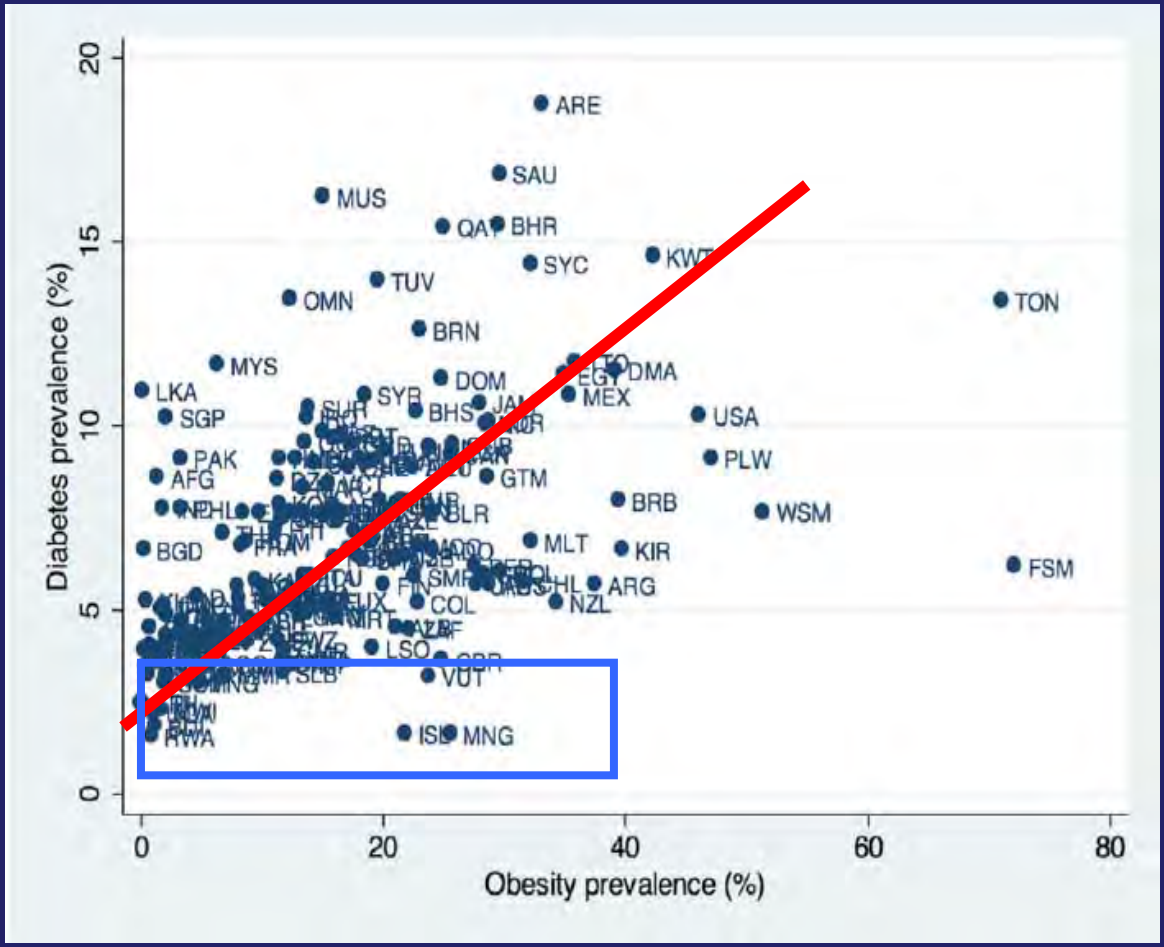

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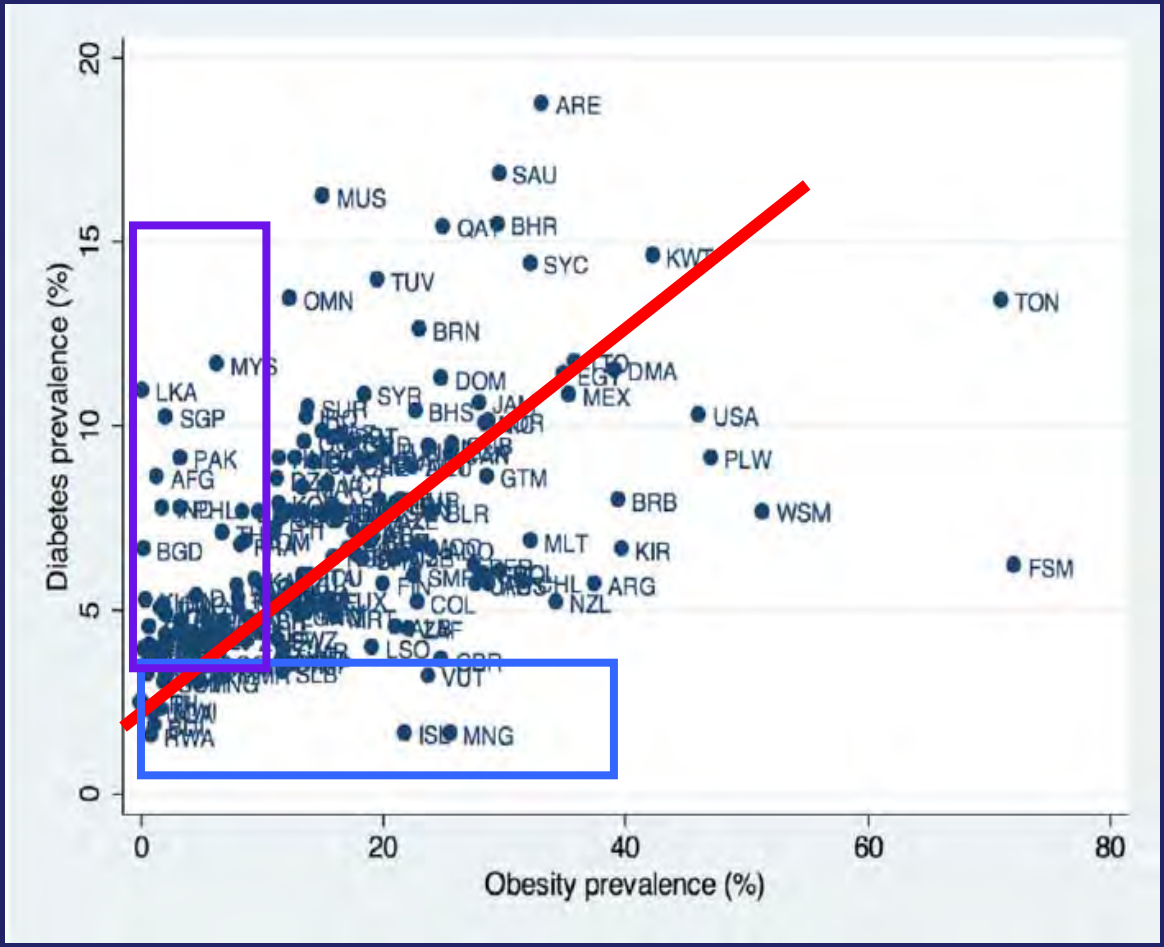
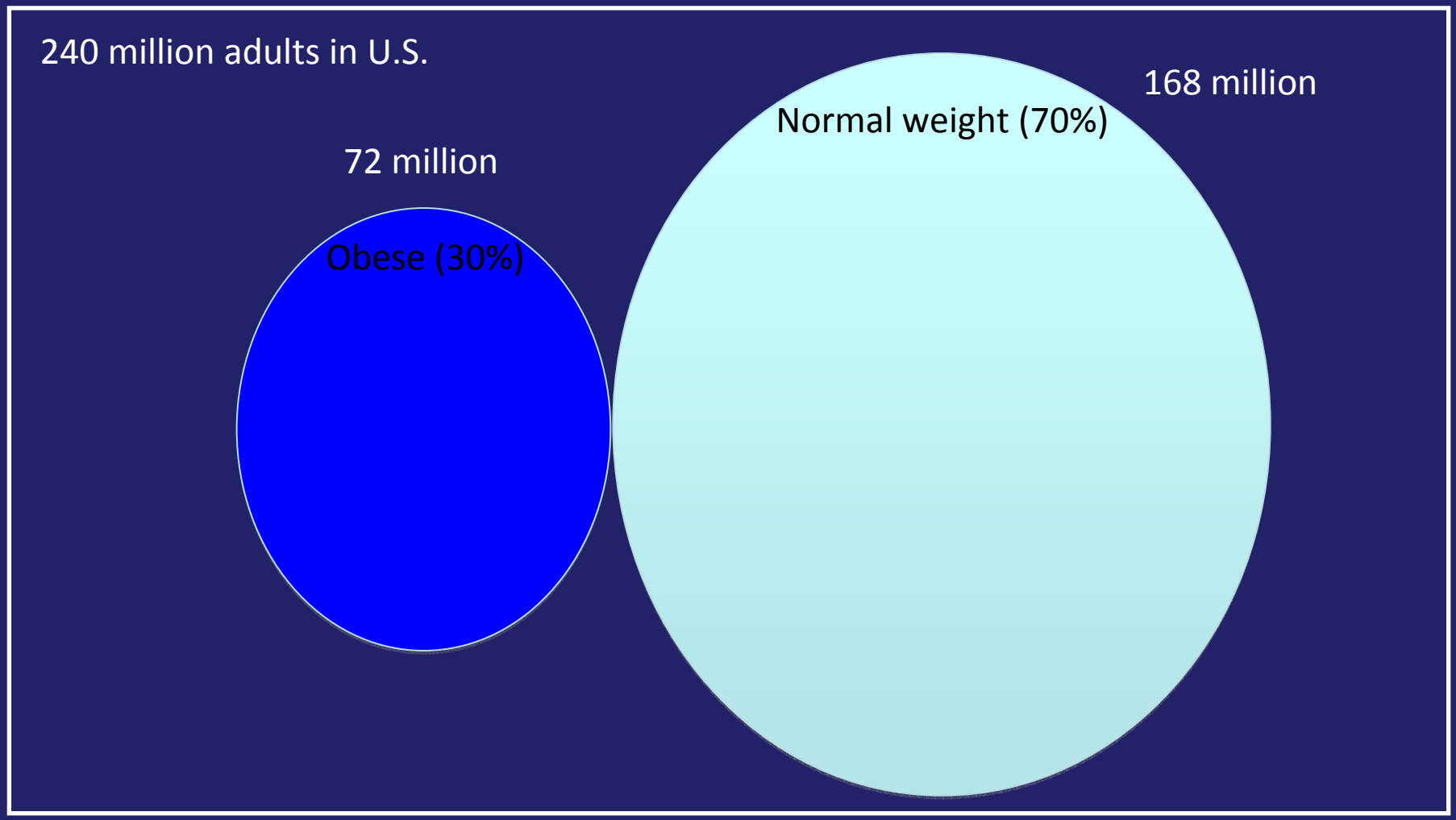


Basu et al. PLoS One 8:e58783, 2013

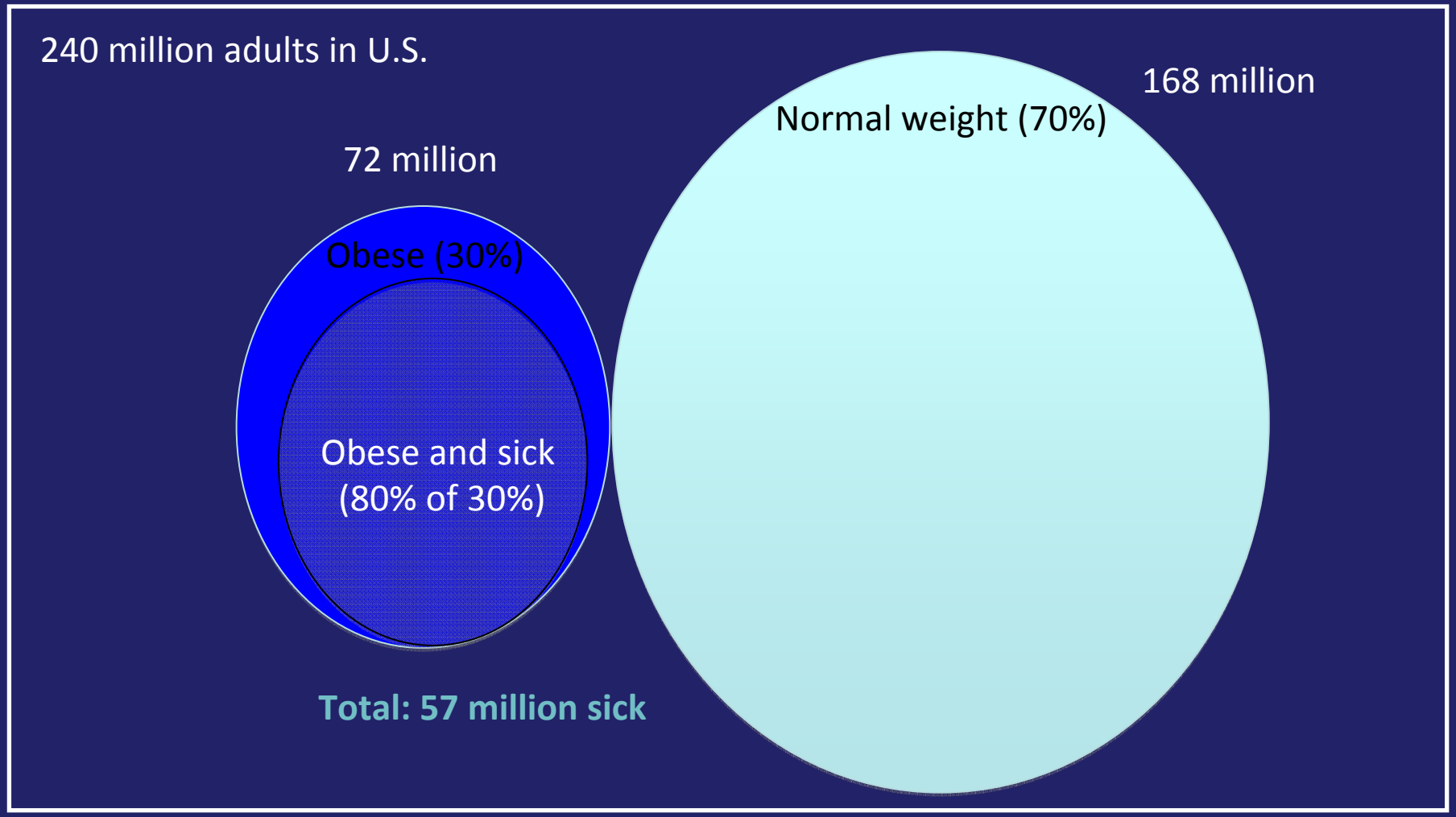
Diabetes is NOT a subset of obesity

- Obesity is increasing worldwide by 1% per year
- Diabetes is increasing worldwide by 4% per year

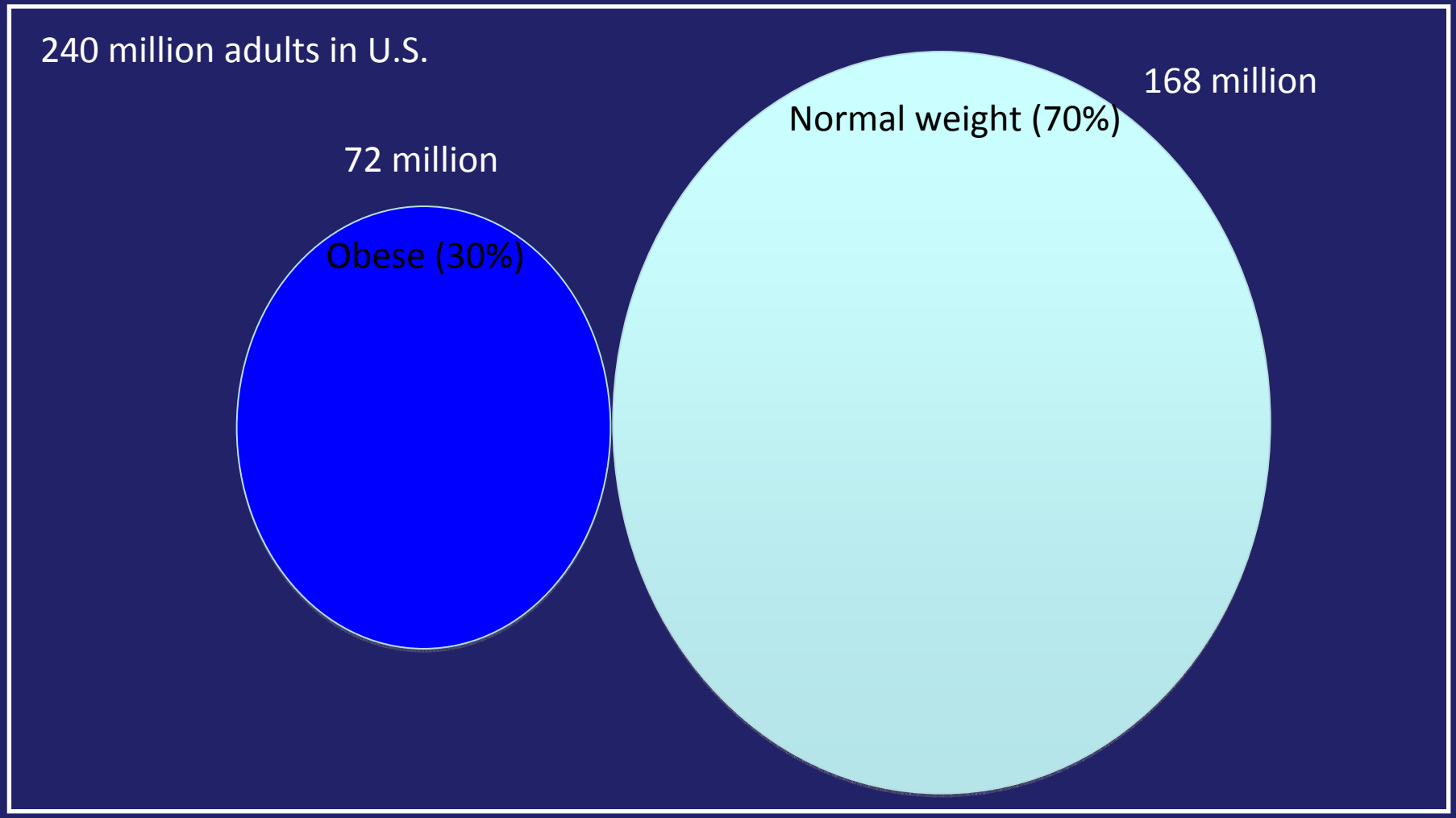
“Exclusive” view of obesity and metabolic dysfunction



“Exclusive” view of obesity and metabolic dysfunction



“Inclusive” view of obesity and metabolic dysfunction



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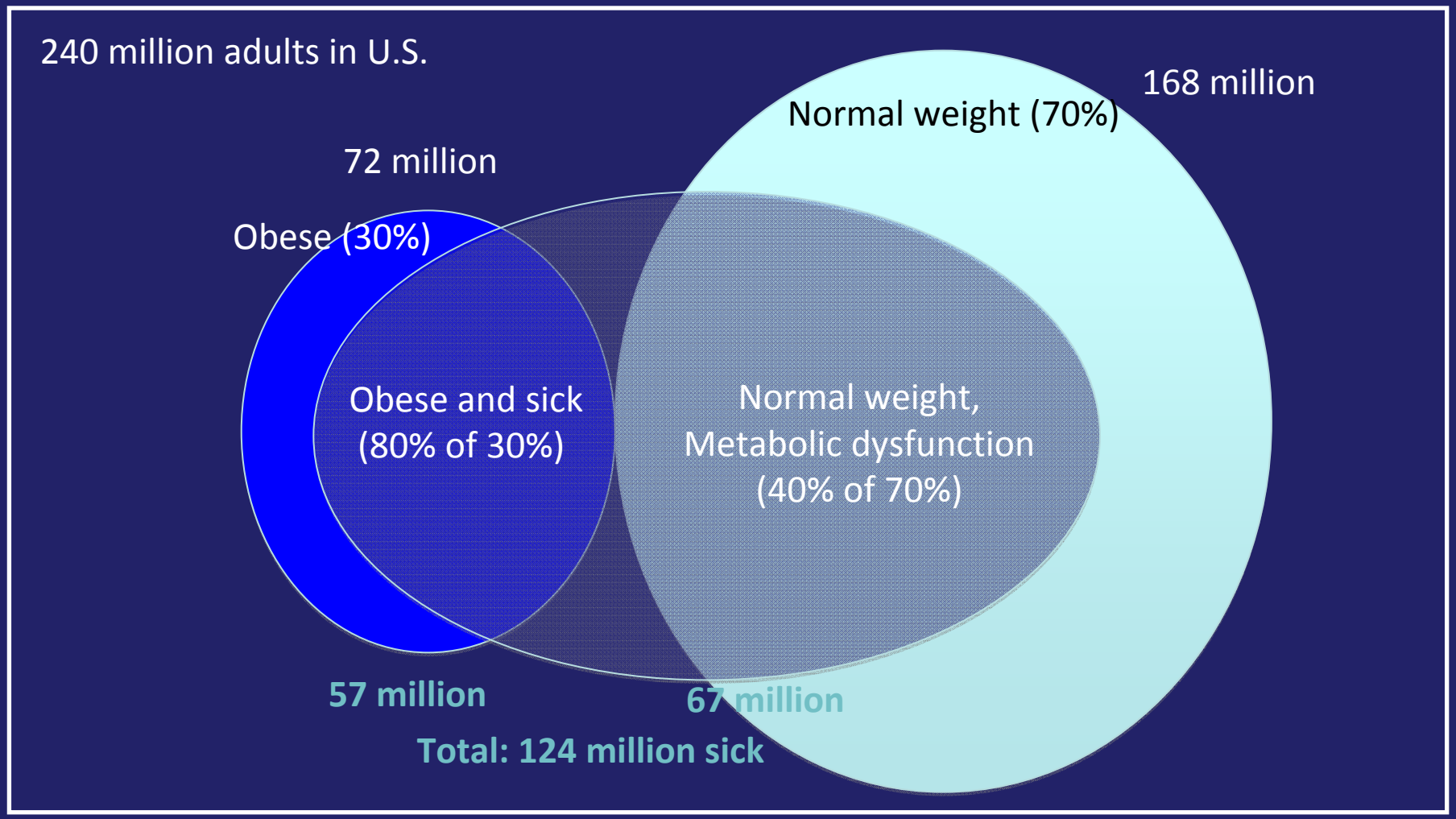
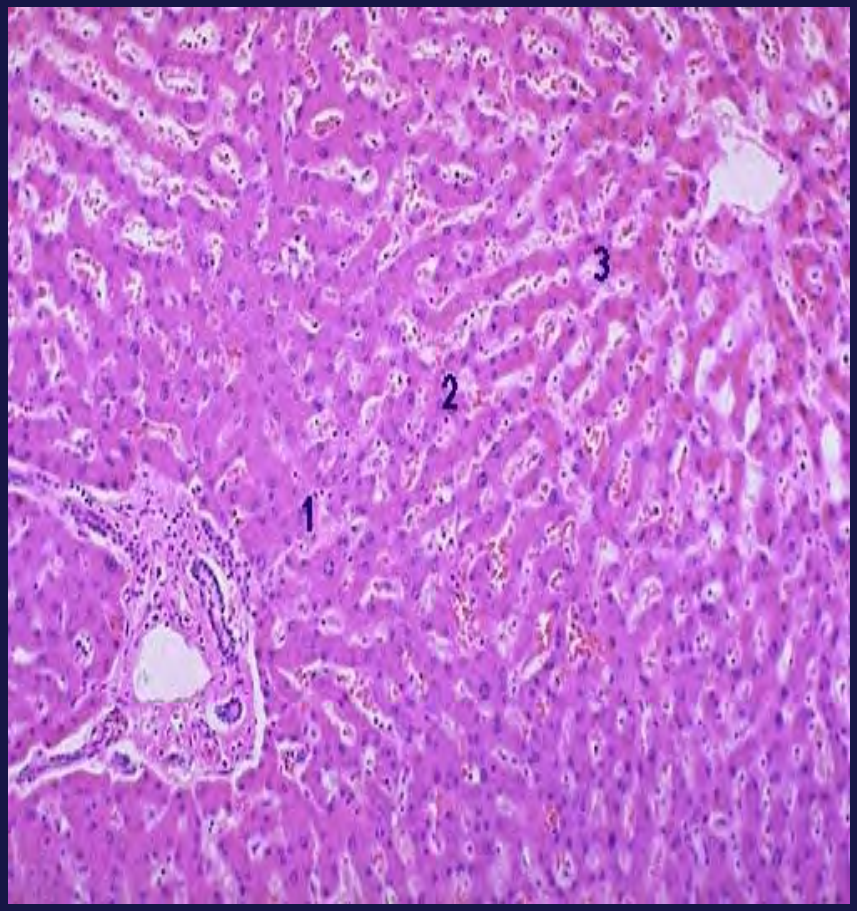
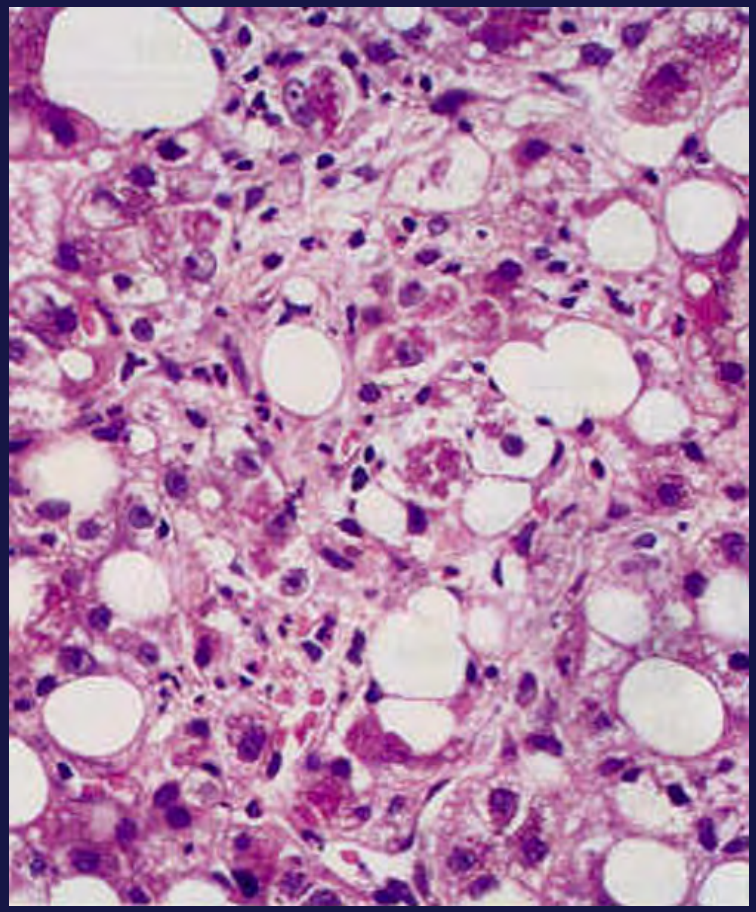


Sugar and Diabetes: Plausibility

Histology of (N)AFLD

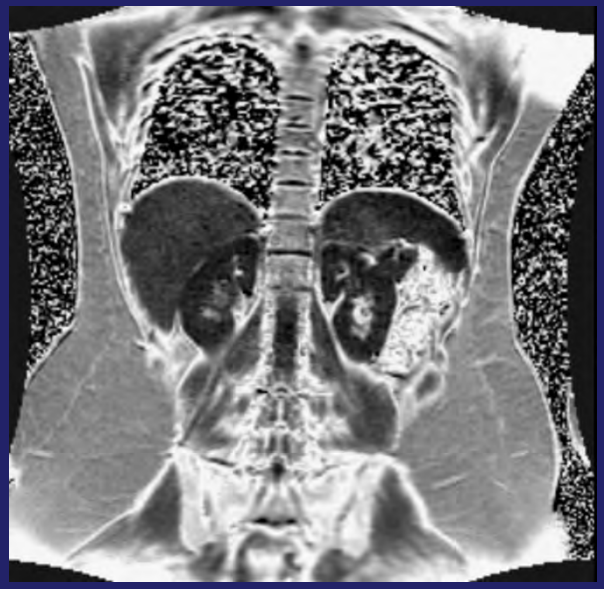


Normal



(N)AFLD

MRI Fat Fraction Maps



Obese

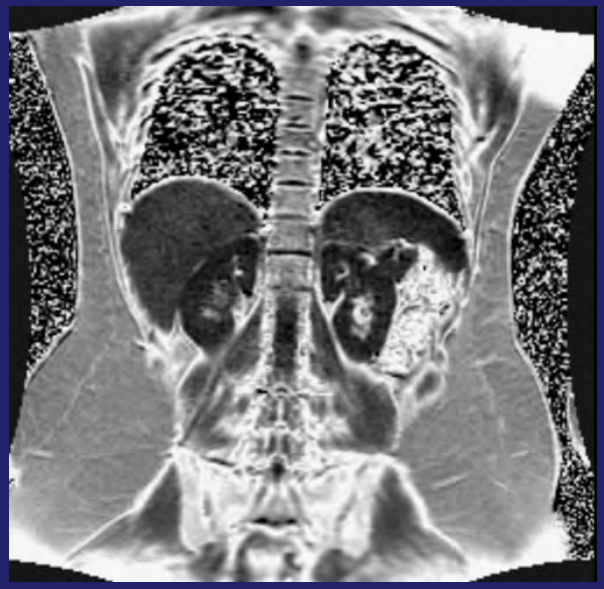
Low Liver Fat = 2.6%

MRI Fat Fraction Maps

Obese

Low Liver Fat = 2.6%

MRI Fat Fraction Maps



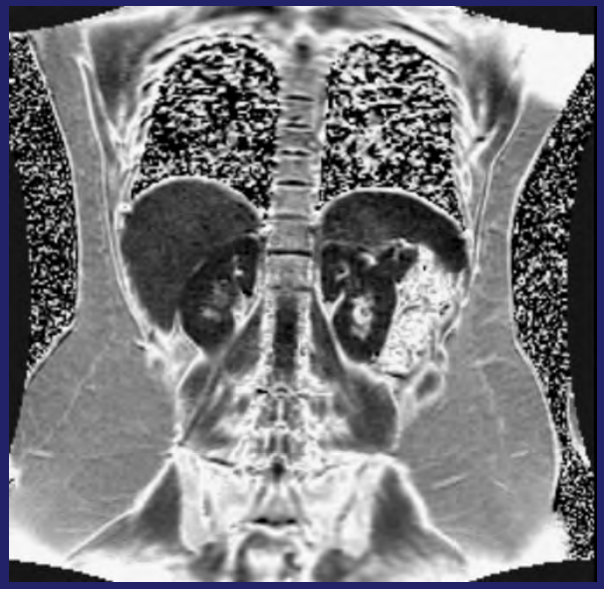
Obese

Low Liver Fat = 2.6%

Obese

High Liver Fat = 24%

MRI Fat Fraction Maps



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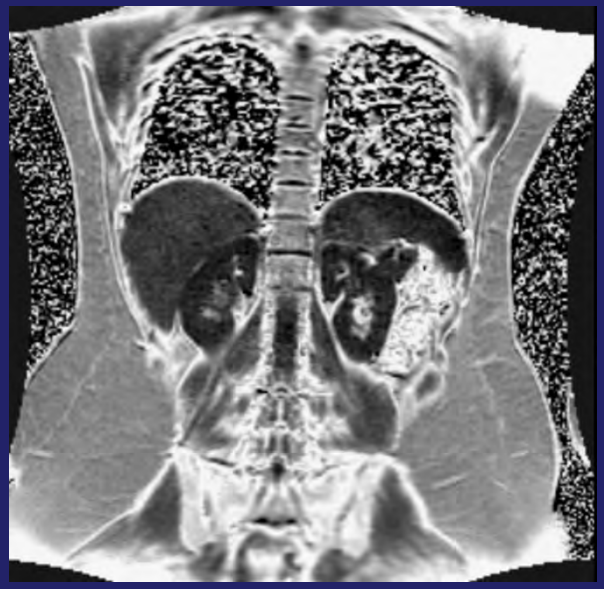


Obese

Low Liver Fat = 2.6%

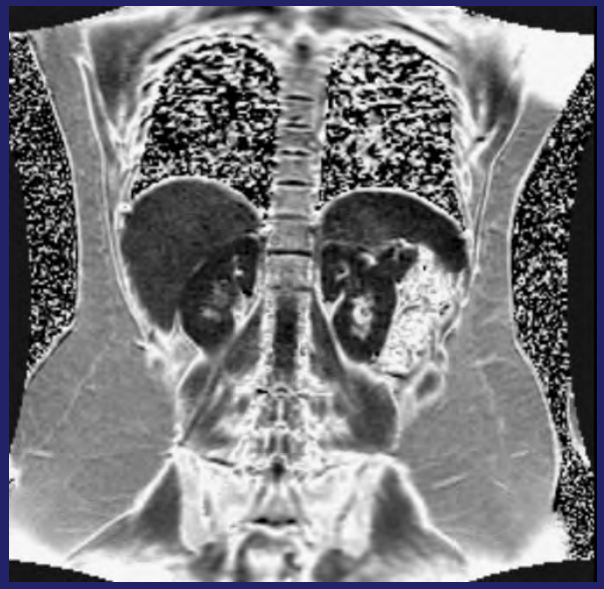
Obese

High Liver Fat = 24%

Thin

High Liver Fat = 23%

MRI Fat Fraction Maps



Obese

Low Liver Fat = 2.6%

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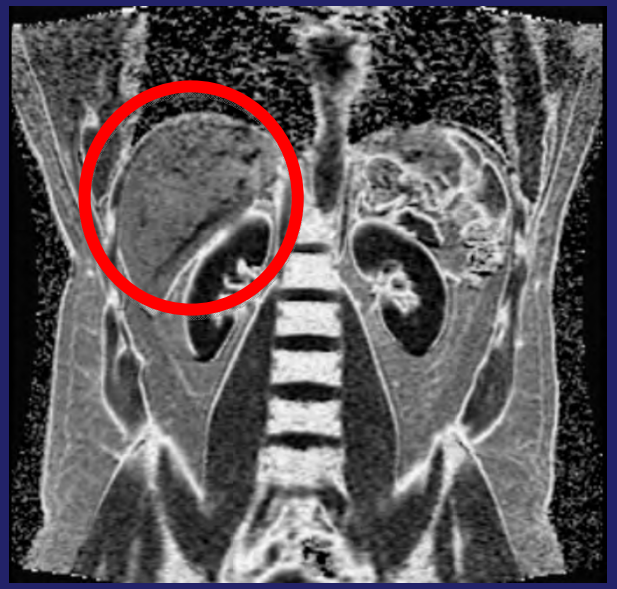
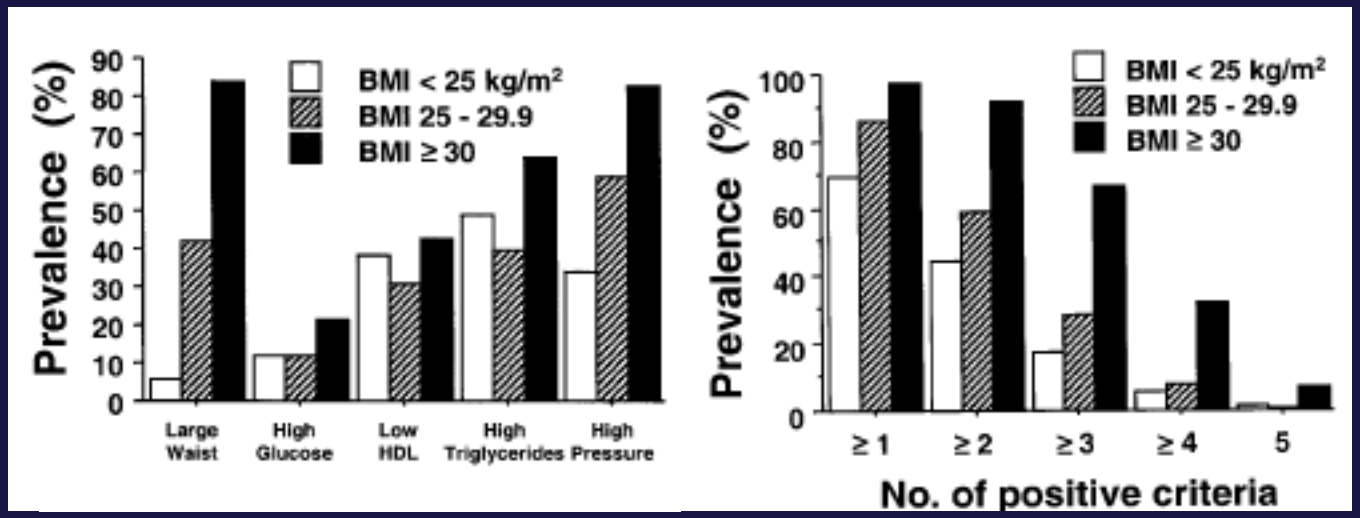


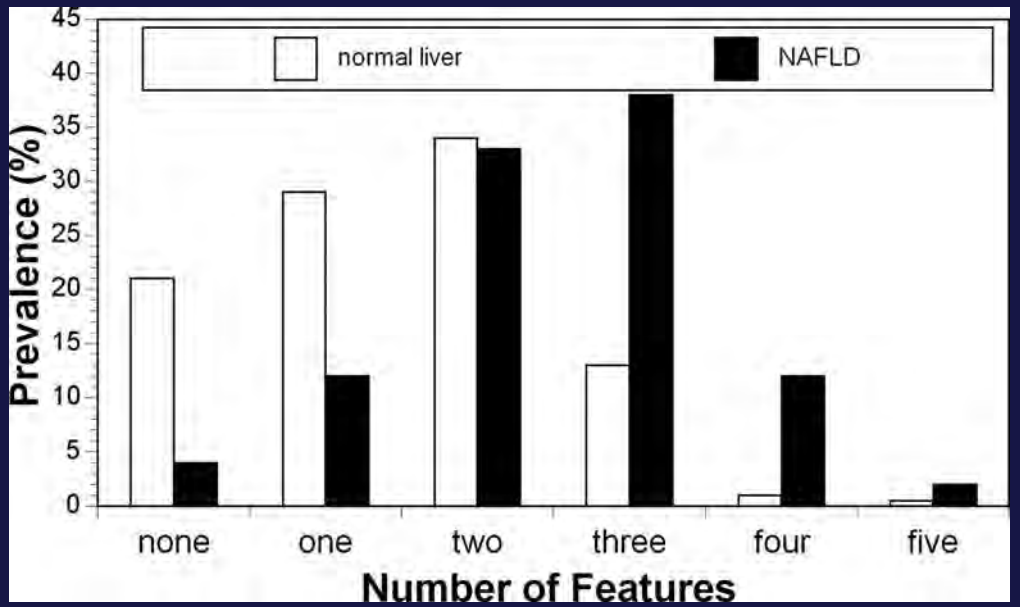
Thin

High Liver Fat = 23%

NAFLD and Metabolic Syndrome are congruent (if not the same)



Adults:
Marchesini et al. Hepatology 37:917, 2003



Children:
Schwimmer et al. Circulation 118:277, 2008

Epidemiology of NAFLD

Non-alcoholic fatty liver disease (NAFLD) has become epidemic

Steatosis:

- 45% Latinos
- 33% Caucasians
- 24% African Americans

NASH

- 5.5% of US Adults

Children:

- Steatosis in 13% of autopsy specimens ages 5-19
- 38% in obese autopsy specimens

Browning et al. Hepatology 40:1387, 2004; Schwimmer et al. Pediatrics 118:1388, 2006

NAFLD is a primary predictor of T2DM in Korean adults

TABLE 2. OR for T2DM at 5-yr follow-up

	T2DM -no./total no. (%)-		OR (95% confidence interval)		
	No fatty liver	Fatty liver	Unadjusted	Adjusted[a]	Adjusted[a] + baseline glucose
All	54/8120 (0.7%)	120/2971 (4%)	6.29 (4.55-8.69)	3.24 (2.19-4.78)	2.05 (1.35-3.12)
Insulin					
Quartile 1	13/2468 (0.5%)	8/307 (2.6%)	5.05 (2.08-12.29)	3.47 (1.23-9.79)	1.96 (0.63-6.13)
Quartile 2	16/2262 (0.7%)	6/511 (1.2%)	1.67 (0.65-4.28)	1.34 (0.46-3.87)	0.71 (0.22-2.26)
Quartile 3	11/2002 (0.6%)	22/768 (2.9%)	5.34 (2.58-11.06)	3.74 (1.59-8.84)	2.92 (1.12-7.62)
Quartile 4	14/1388 (1.0%)	84/1385 (6.1%)	6.34 (3.58-11.21)	3.31 (1.76-6.20)	2.42 (1.23-4.75)

[a] Adjusted for age, gender, BMI, alcohol (grams per day), education (+ 16 yr, + 16 yr), smoking (never or past, current), and exercise (+ 1 time/wk, + 1 time/wk).

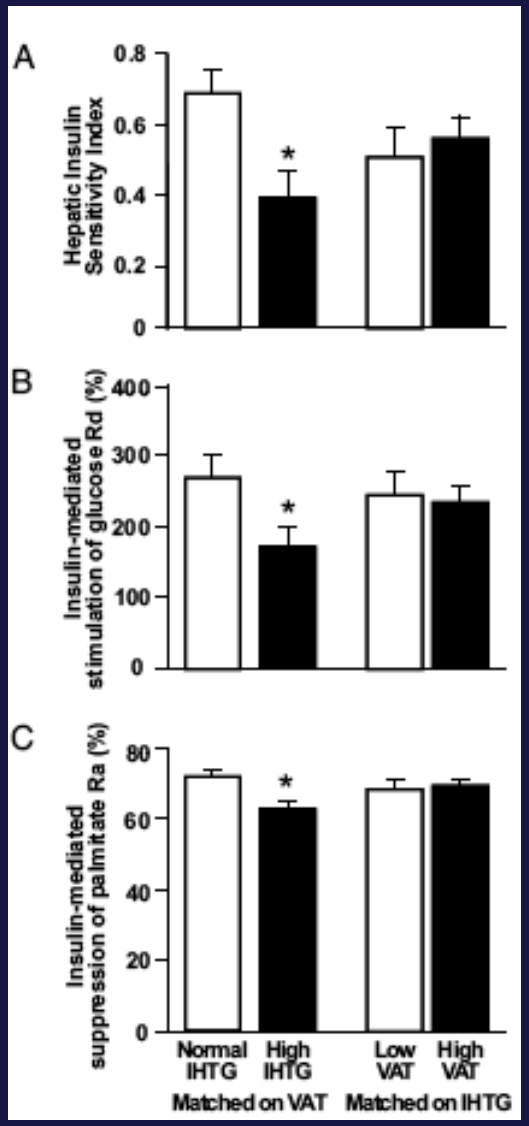
Sung and Kim, J Clin Endocrinol Metab 96:1093, 2011

Intrahepatic fat explains metabolic perturbation better than visceral fat

Hepatic Insulin Sensitivity Index

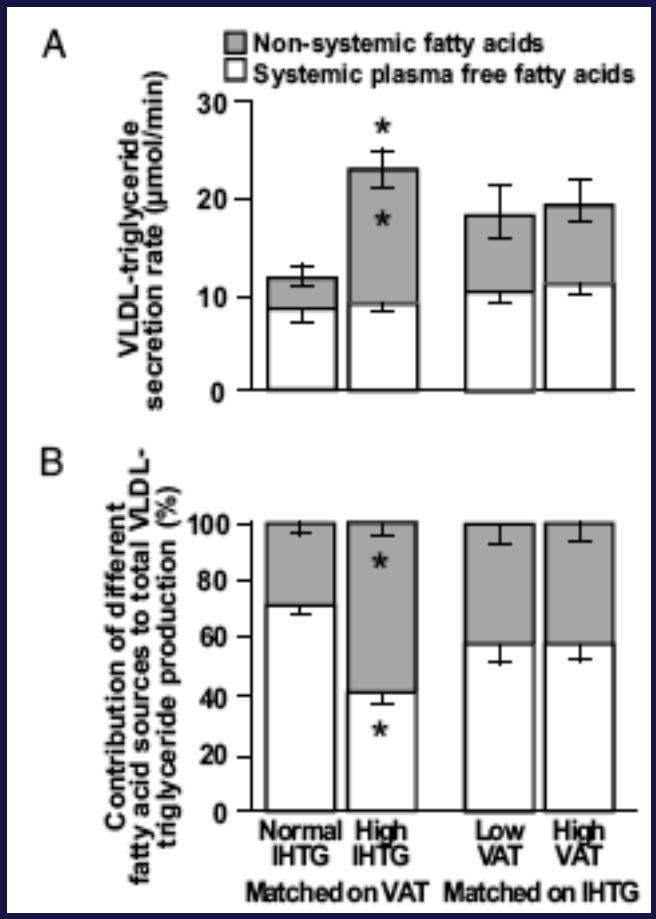
Insulin Stimulated Glucose Disposal Rate

Insulin Stimulated Palmitate Suppression Rate



VLDL Secretion Rate

Contribution Of Free Fatty Acids To VLDL



Fabbrini et al. Proc Natl Acad Sci 106:15430, 2009

Sugar and Diabetes: Mechanisms

The first problem: Fructose is not glucose

Common wisdom: A calorie is a calorie, and
"Sugar is just "empty calories"

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

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But:

- **Chronic fructose exposure promotes liver fat accumulation, which promotes Metabolic Syndrome**

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"Sugar is just "empty calories"

But:

- **Chronic fructose exposure promotes liver fat accumulation, which promotes Metabolic Syndrome**

- **Chronic fructose exposure increases protein glycation, which promotes cellular and structural aging**

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
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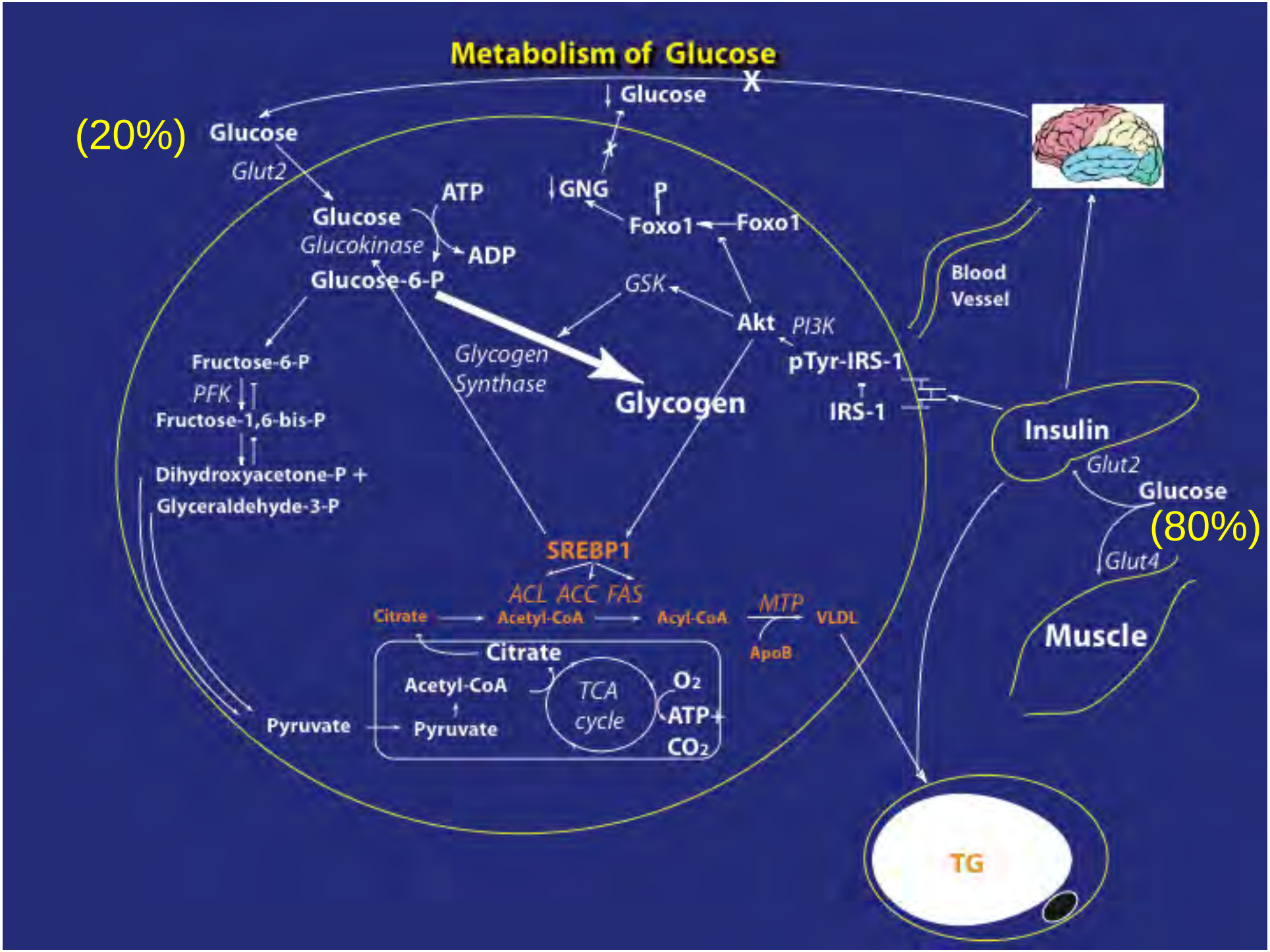

Metabolism of Glucose
(20%)
Glucose
Glut2
Glucose
Glucokinase
ATP
ADP
Glucose-6-P
Fructose-6-P
PFK
Fructose-1,6-bis-P
Dihydroxyacetone-P +
Glyceraldehyde-3-P
Pyruvate
Pyruvate
Acetyl-CoA
Citrate
TCA cycle
O2
ATP
CO2
Citrate
Acetyl-CoA
Acyl-CoA
ACL ACC FAS
SREBP1
MTP
ApoB
VLDL
TG
Glycogen Synthase
Glycogen
GSK
Akt
PI3K
pTyr-IRS-1
IRS-1
GNG
P
Foxo1
Foxo1
Glucose
X
Blood Vessel
Insulin
Glut2
Glucose
(80%)
Glut4
Muscle

Can you name an energy source that is:

Can you name an energy source that is:

Not necessary for life

Can you name an energy source that is:

Not necessary for life

There is no biochemical reaction in the body that requires it

Can you name an energy source that is:

- Not necessary for life
- There is no biochemical reaction in the body that requires it
- Is not nutrition

Can you name an energy source that is:

- Not necessary for life
- There is no biochemical reaction in the body that requires it
- Is not nutrition
- When consumed in excess it is toxic

Can you name an energy source that is:

- Not necessary for life
- There is no biochemical reaction in the body that requires it
- Is not nutrition
- When consumed in excess it is toxic
- We love anyway

Can you name an energy source that is:

Not necessary for life

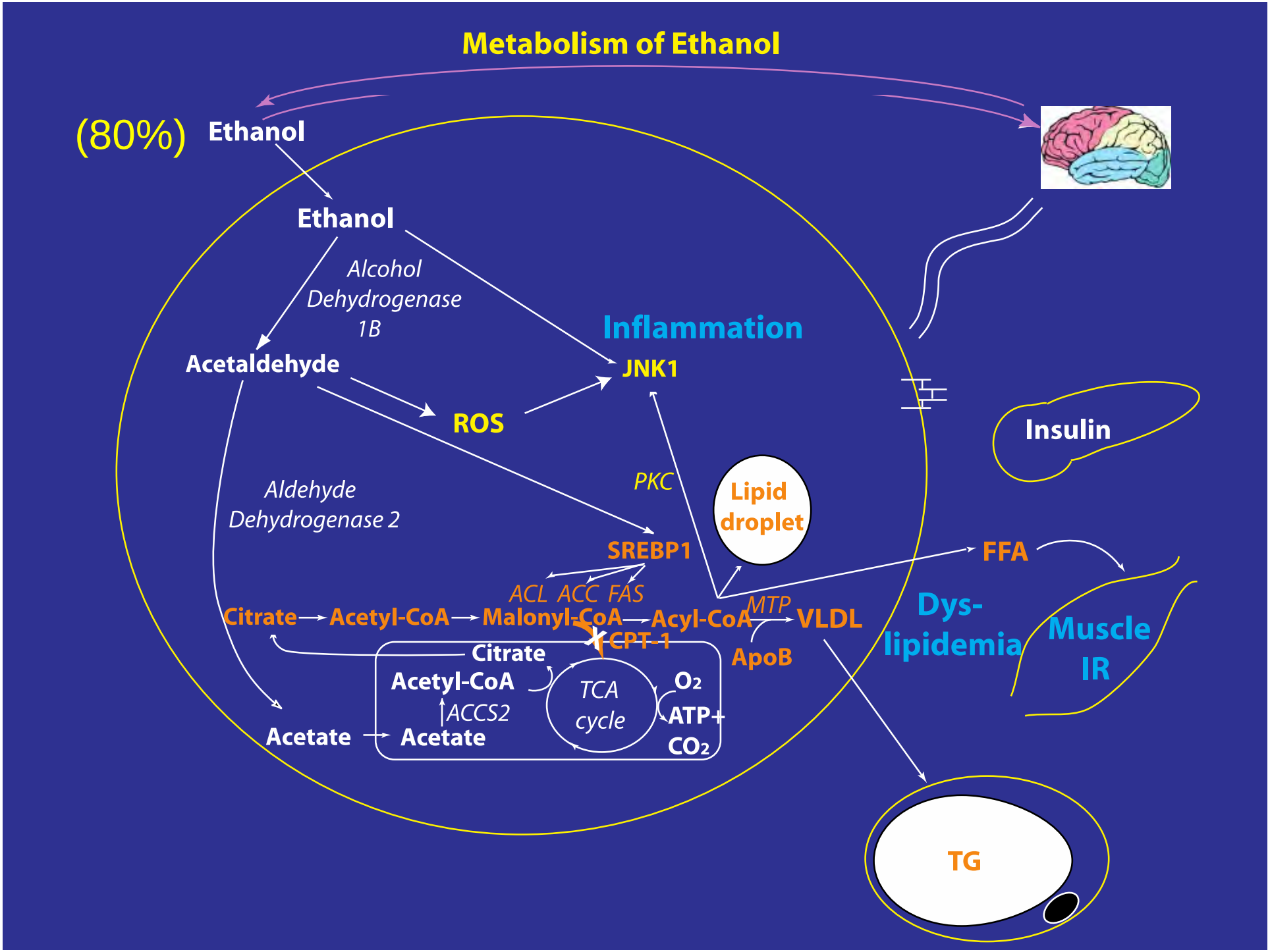
There is no biochemical reaction in the body that requires it

Is not nutrition

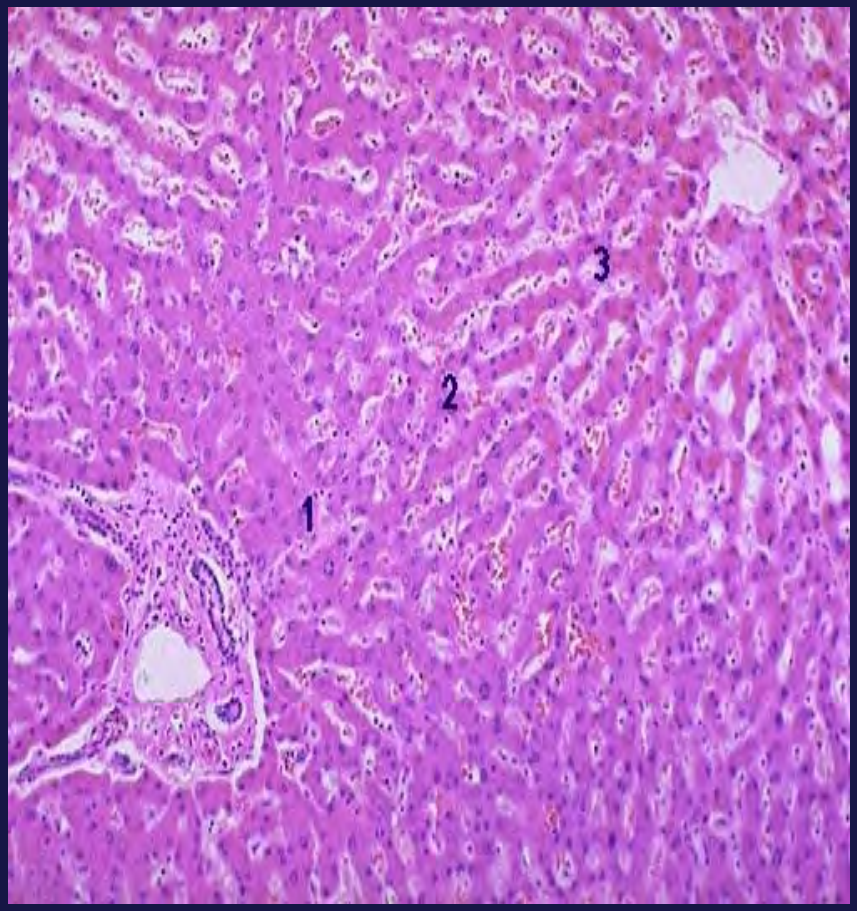
When consumed in excess it is toxic

We love anyway

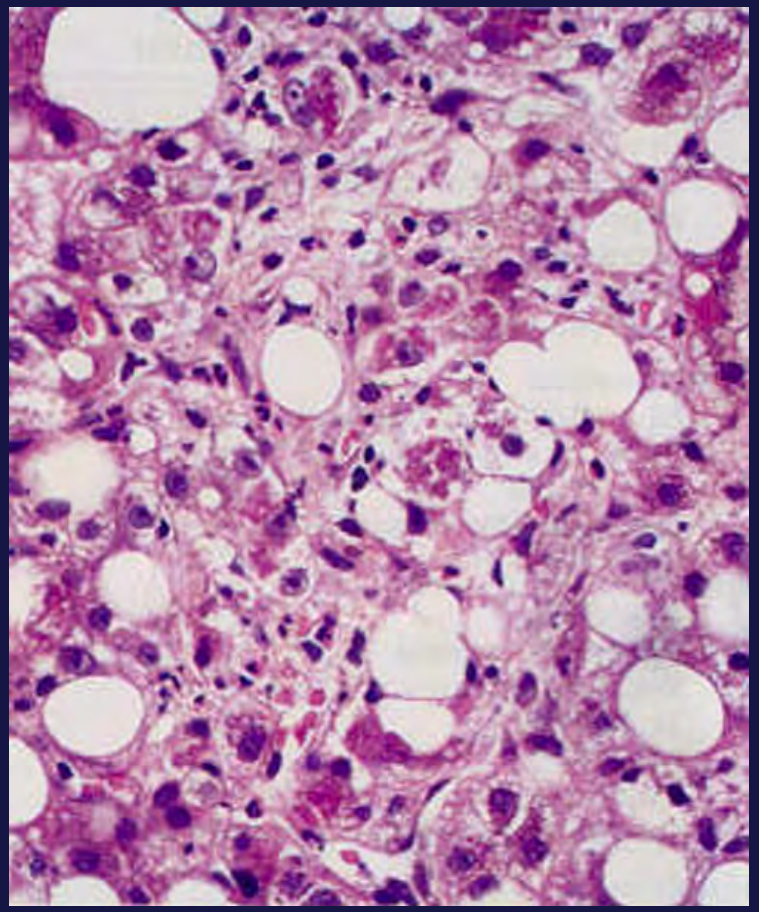
Answer: Ethanol


Metabolism of Ethanol
(80%)
Ethanol
Ethanol
Alcohol
Dehydrogenase
1B
Acetaldehyde
Inflammation
JNK1
ROS
Insulin
PKC
Aldehyde
Dehydrogenase 2
Lipid
droplet
SREBP1
FFA
ACL ACC FAS
Citrate
Acetyl-CoA
Malonyl-CoA
Acyl-CoA
MTP
VLDL
CPT-1
Dys-
lipidemia
Muscle
IR
Citrate
Acetyl-CoA
ACCS2
TCA
cycle
O2
ATP+
CO2
ApoB
Acetate
Acetate
TG

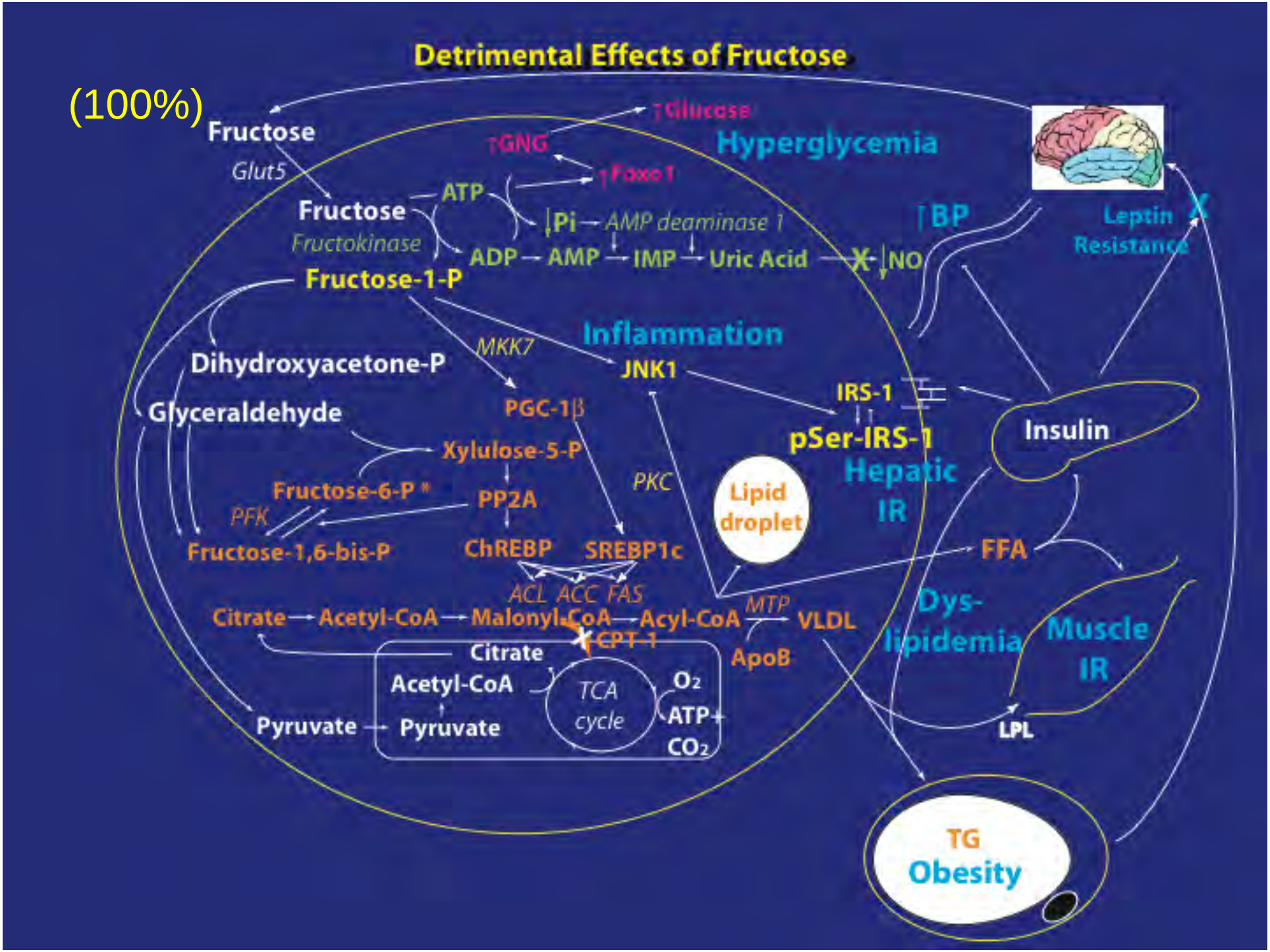
Histology of (N)AFLD



Normal



Alcohol?
Sugar?


(100%)
Detrimental Effects of Fructose
Fructose
Glut5
Fructose
Fructokinase
Fructose-1-P
ATP
ADP
↓Pi
AMP deaminase 1
AMP
IMP
Uric Acid
↑GNG
↑Glucose
↑Foxo1
Hyperglycemia
↑BP
↓NO
Leptin Resistance
Inflammation
MKK7
JNK1
Dihydroxyacetone-P
Glyceraldehyde
PGC-1β
Xylulose-5-P
Fructose-6-P
PFK
Fructose-1,6-bis-P
PP2A
PKC
IRS-1
pSer-IRS-1
Hepatic IR
Insulin
Lipid droplet
ChREBP
SREBP1c
ACL ACC FAS
Citrate
Acetyl-CoA
Malonyl-CoA
Acyl-CoA
CPT-1
MTP
VLDL
ApoB
FFA
Dys-lipidemia
Muscle IR
LPL
Citrate
Acetyl-CoA
Pyruvate
Pyruvate
TCA cycle
O2
ATP
CO2
TG
Obesity

Isocaloric fructose vs. complex carbohydrate increases intrahepatic lipid in adults

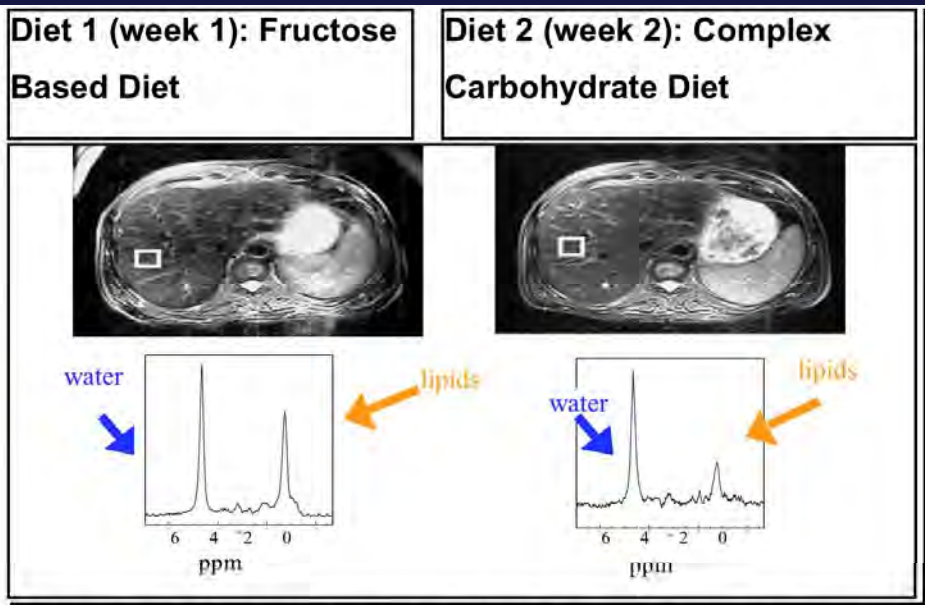


Figure 7: Decrease in lipids after one week on the complex-carbohydrate diet (right) after the fructose based diet (left) MRI (white box marks MRS location) & MR spectra for a subject showing: Complex-CHO based diet: lipids:water = 27% of the fructose-based diet (45).

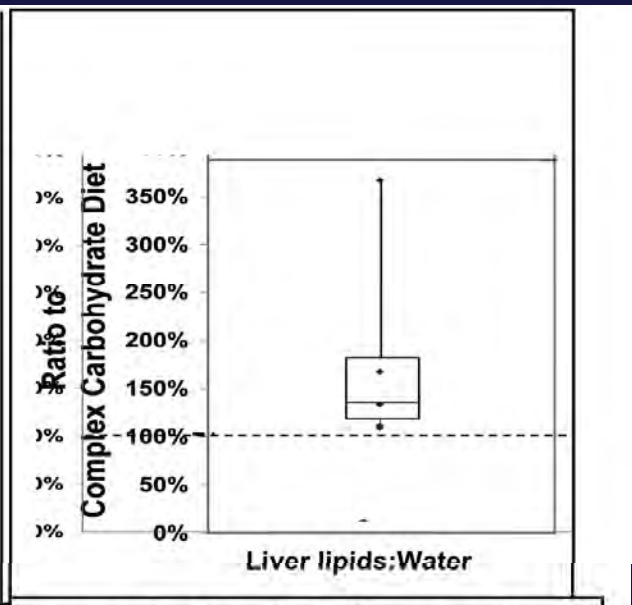


Figure 8: Hepatic lipid: water for all subjects after a 7-day fructose based diet [% of values after a 7-day complex-carbohydrate diet]. The box indicates the median, first and third quartiles, with the bars indicating the extreme values (45).

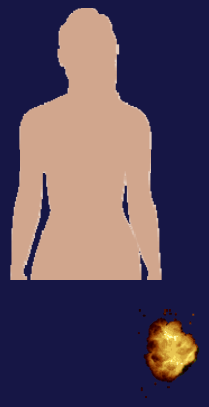
Noworlowski et al. Proc Int Soc Mag Res Med 2699, 2009

The second problem

The common link

The browning reaction or Maillard reaction or non-enzymatic glycation

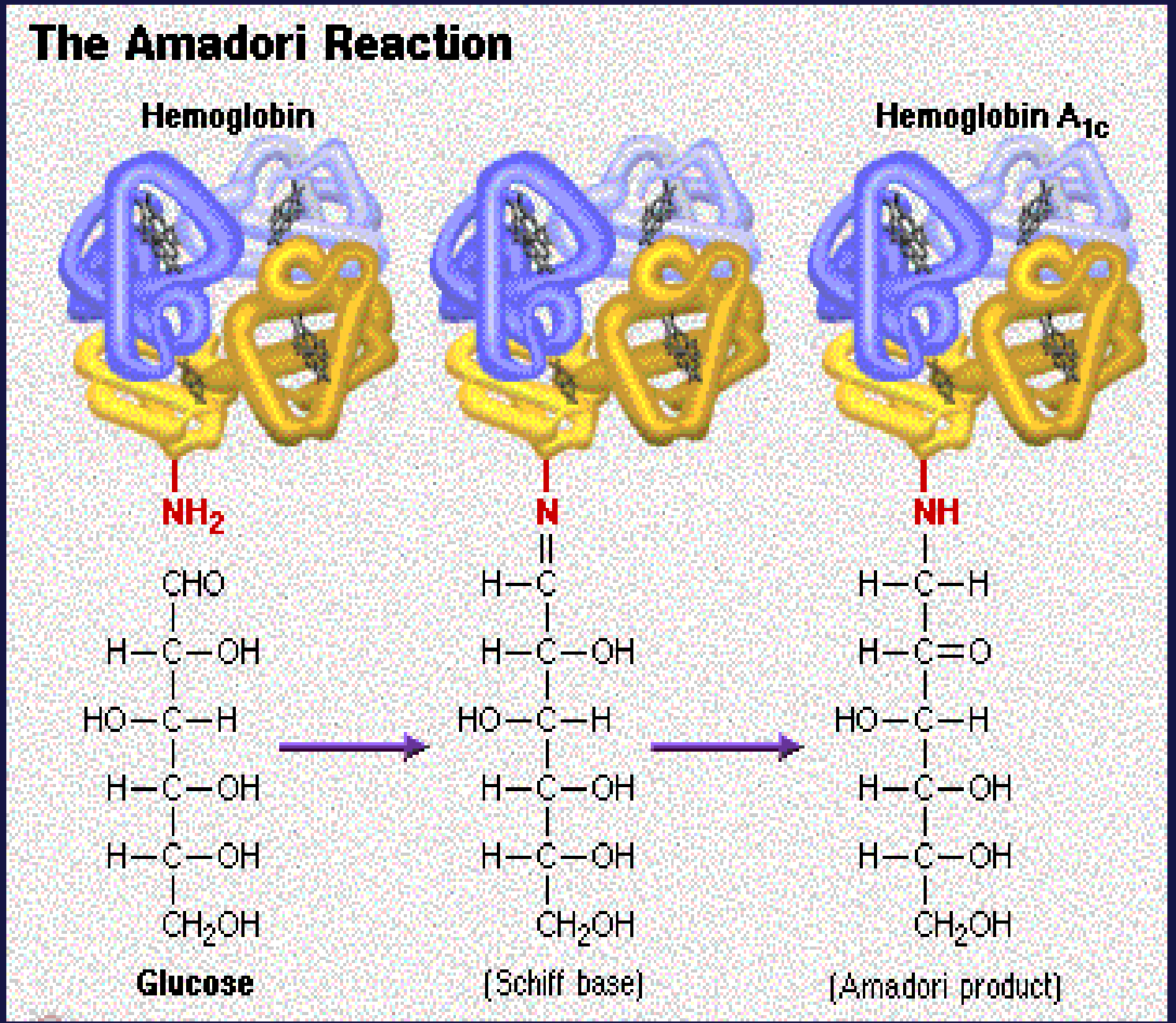
Instead of roasting 1 hour at 375 degrees we slow cook at 98.6 degrees for 75 years



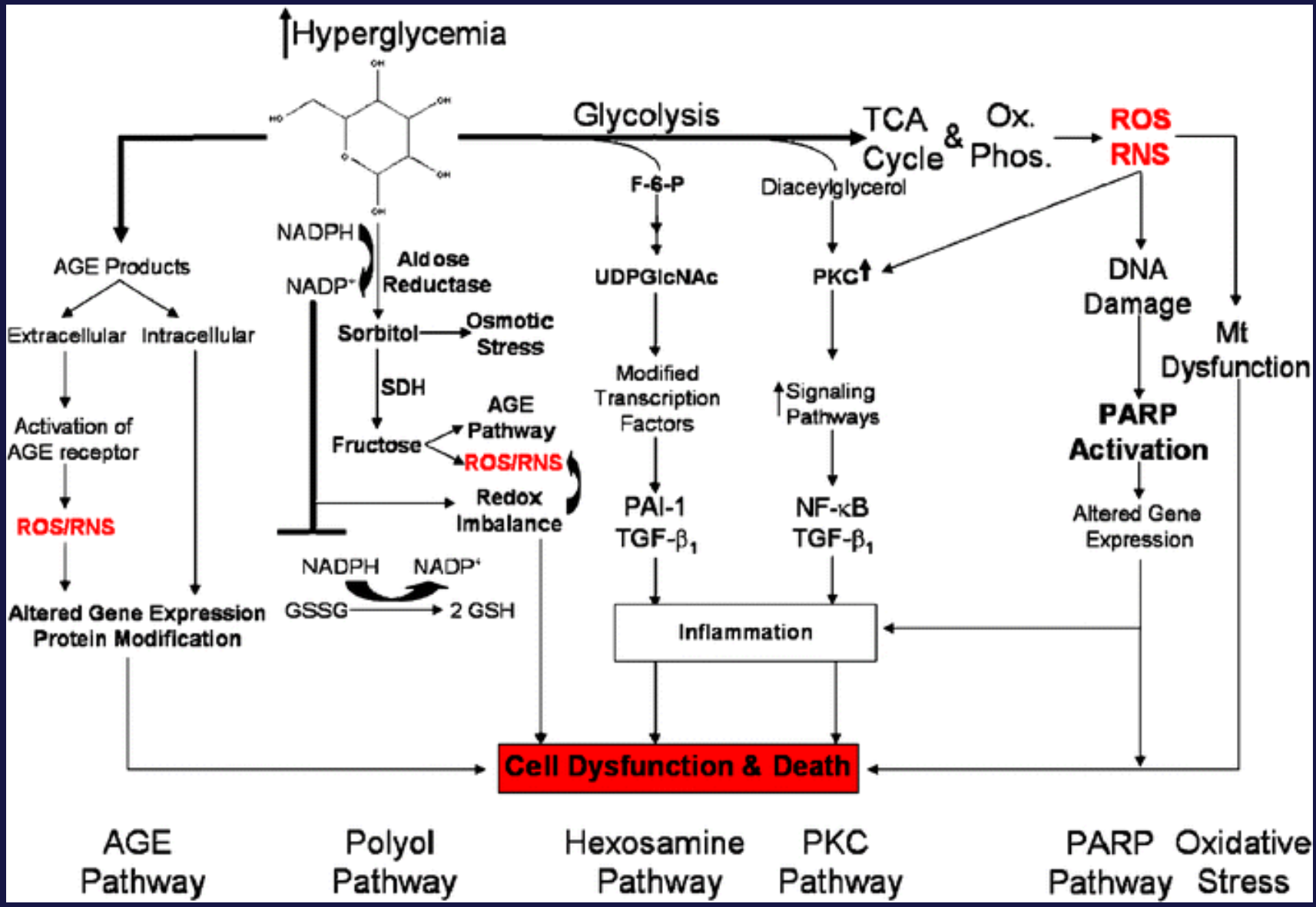
Aging and costal cartilage

Courtesy Dr Baynes

The Amadori Reaction

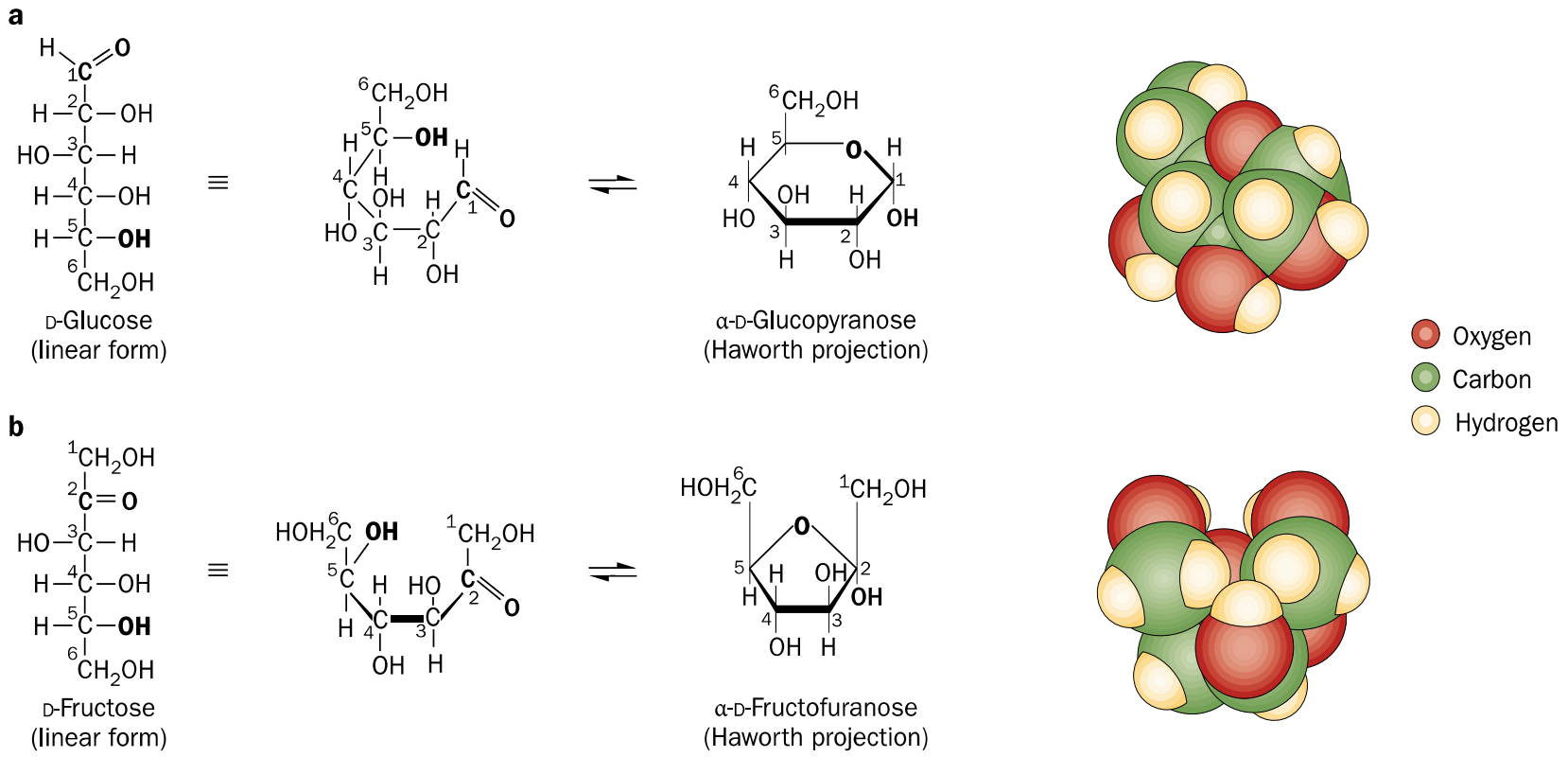


Generation of reactive oxygen species by carbohydrate



Figueroa-Romero et al. Rev Endo Metab Dis 9:301, 2008

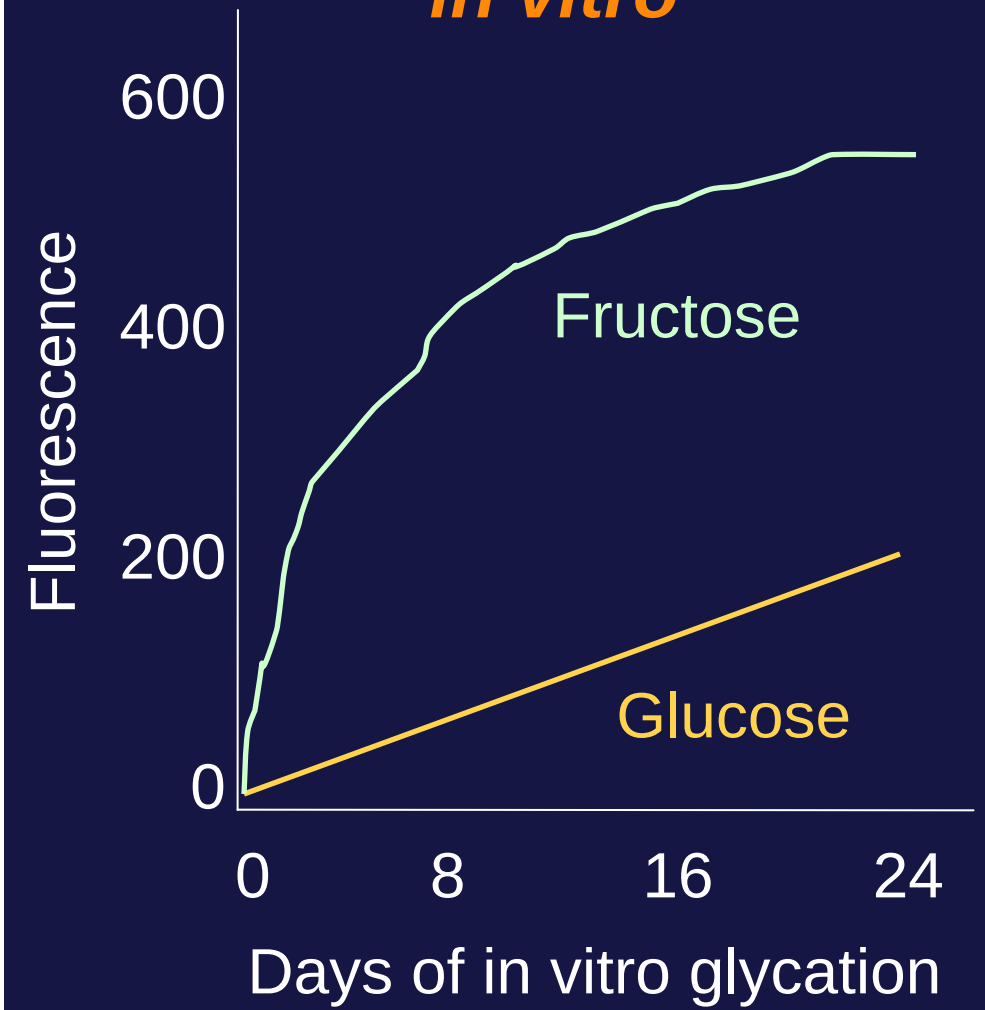
The furan ring of fructose is more unstable, so at equilibrium, fructose exists in the linear form



Lim et al. Nat Rev Gastro Hepatol 7:251, 2010

Non-enzymatic glycation: fructose >> glucose

Fructose and glycation *in vitro*



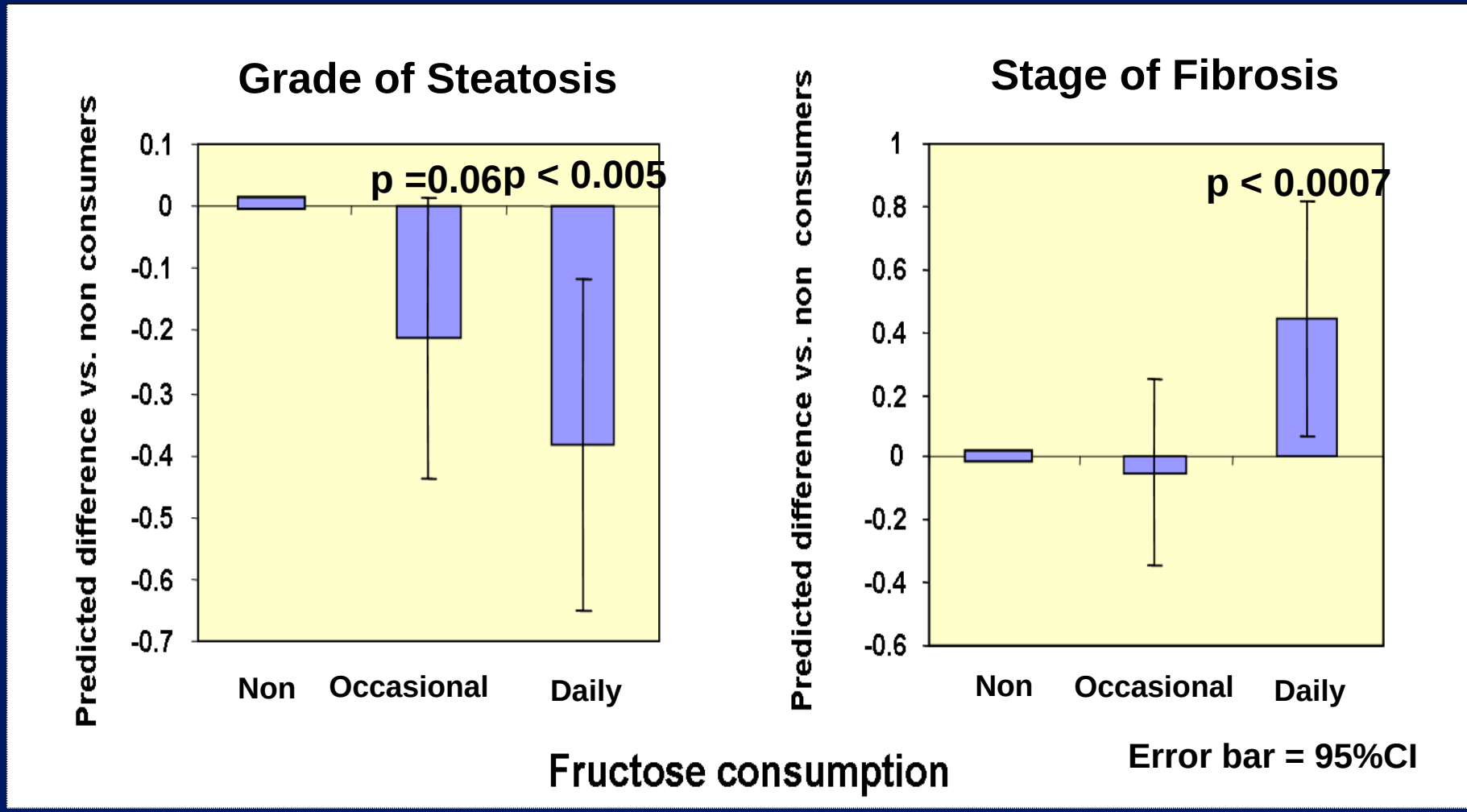
Ahmed and Furth, Clin Chem 38:1301, 1992

Rates of reactivity

	Rate (/mM/hr)	Carbonyl %
Glucose	0.6	0.002
Galactose	2.8	0.02
Fructose	4.5	0.7

Bunn and Higgins, Science 213:222, 1981

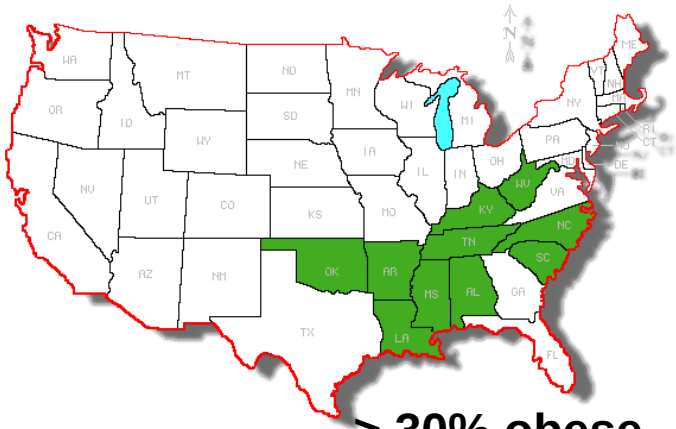
Association of fructose consumption with severity of steatosis and fibrosis



Abdelmalek et al. Hepatology 51:1961, 2010

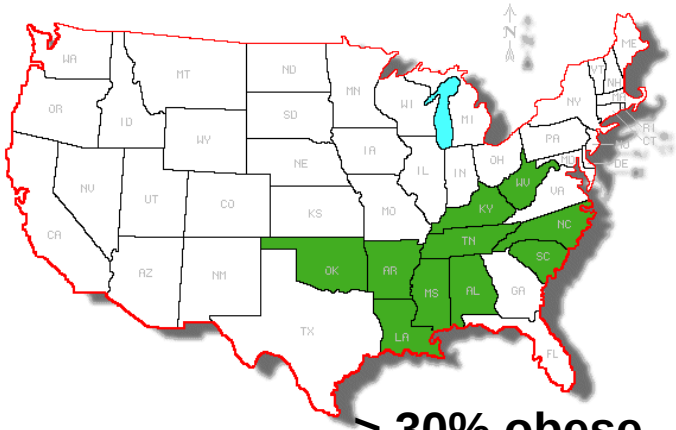
Sugar and Diabetes: Human Correlation

10 Most Obese States



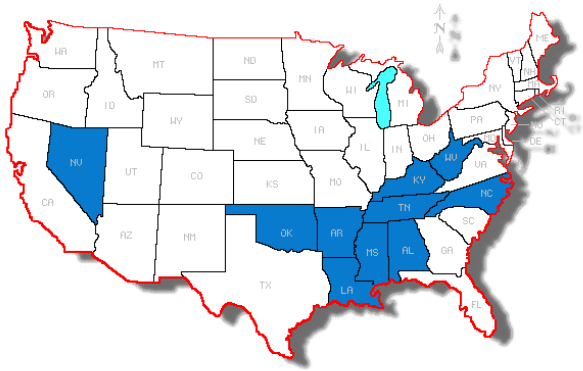
> 30% obese

10 Most Obese States



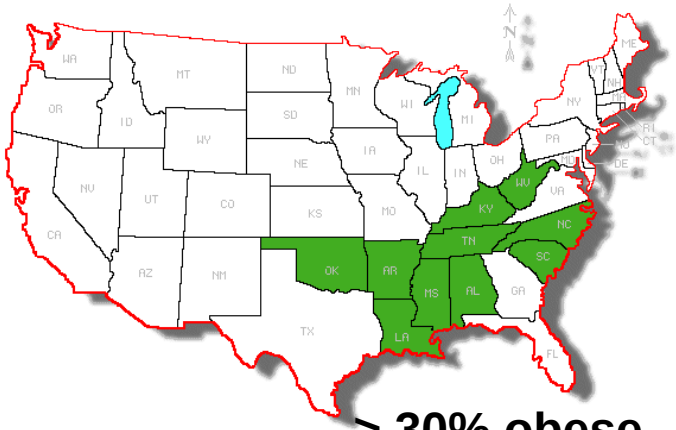
> 30% obese

10 Laziest States



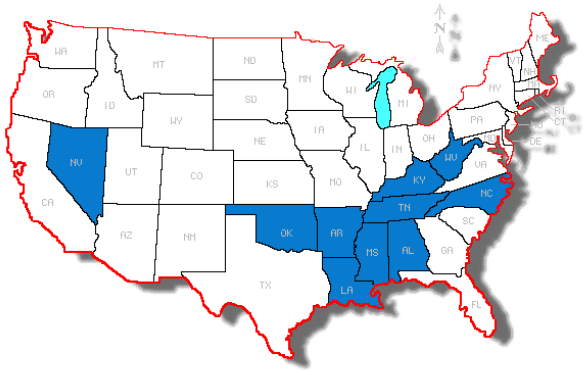
< 63% active

10 Most Obese States



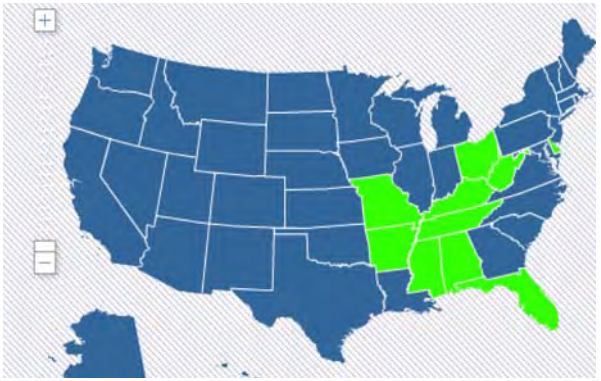
> 30% obese

10 Laziest States

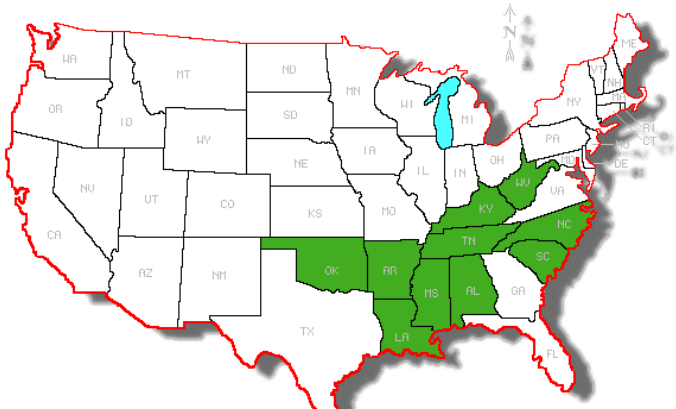


< 63% active

10 Most Unhappy States

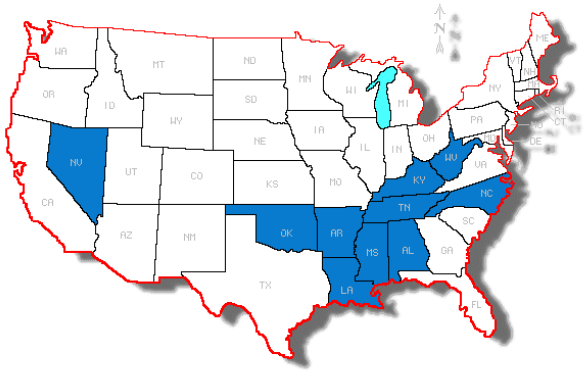


10 Most Obese States



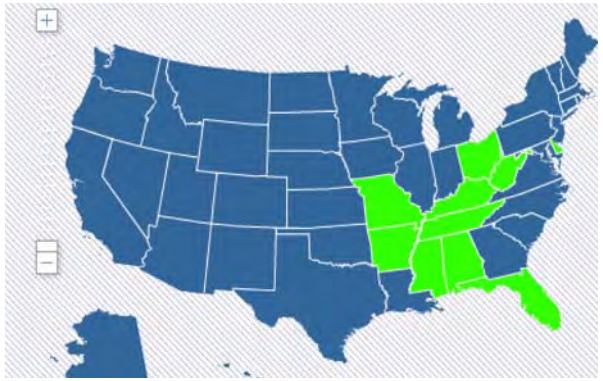
> 30% obese

10 Laziest States

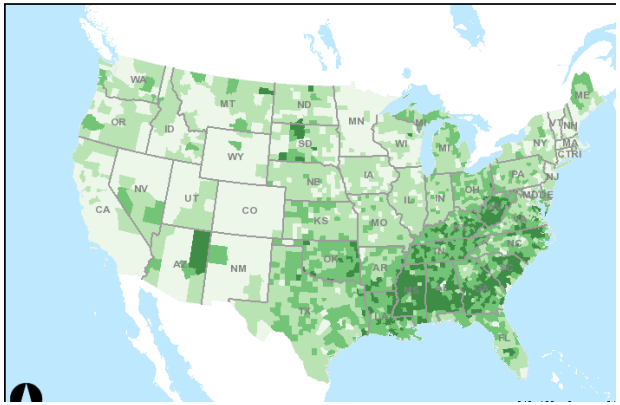


< 63% active

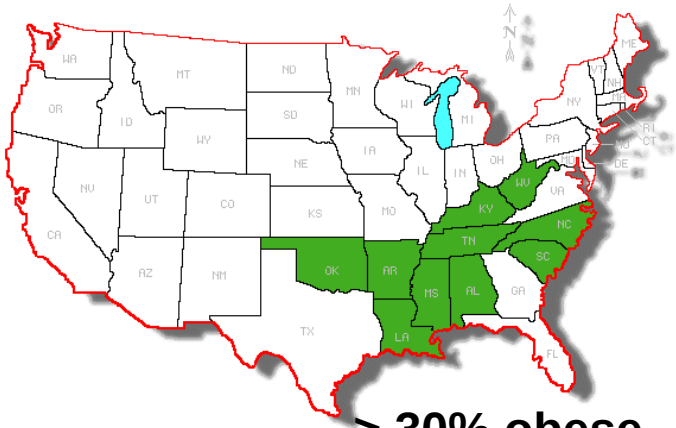
10 Most Unhappy States



Adult Diabetes Rate

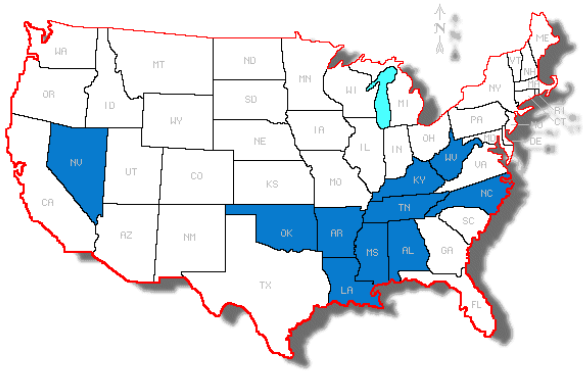


10 Most Obese States



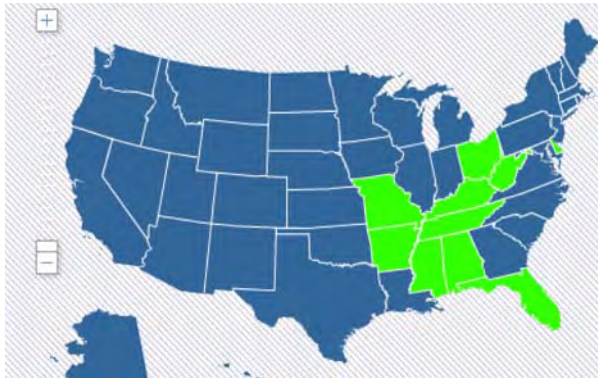
> 30% obese

10 Laziest States

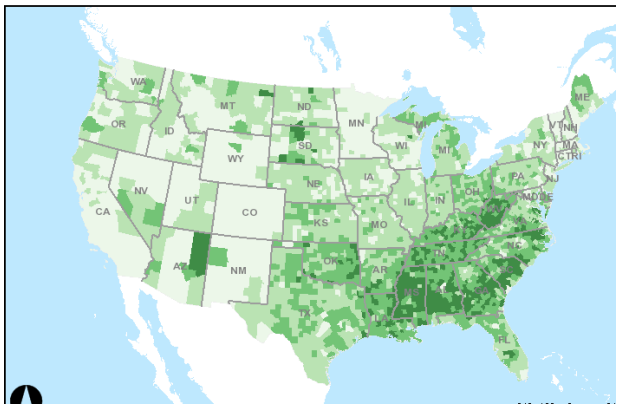


< 63% active

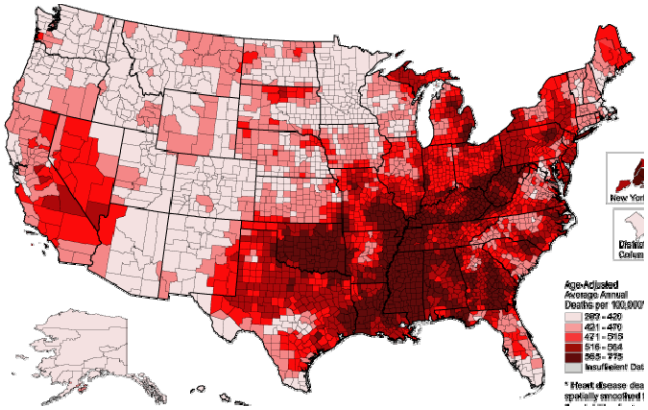
10 Most Unhappy States



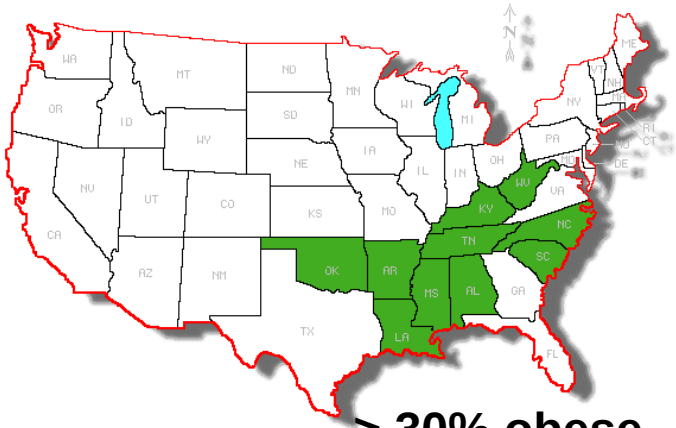
Adult Diabetes Rate



Adult Heart Disease Rate

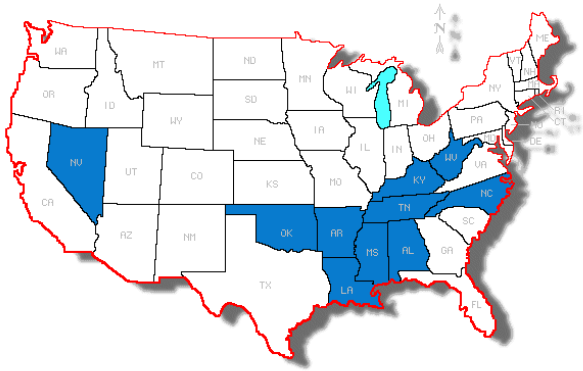


10 Most Obese States



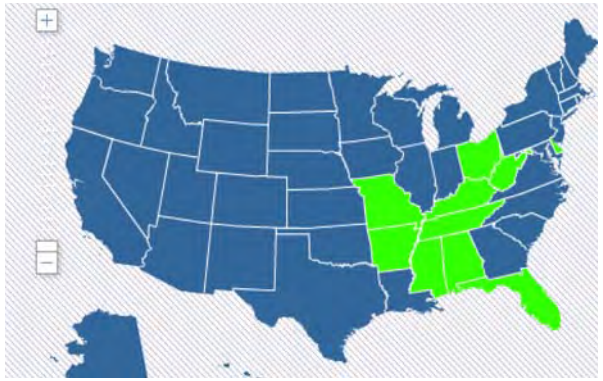
> 30% obese

10 Laziest States

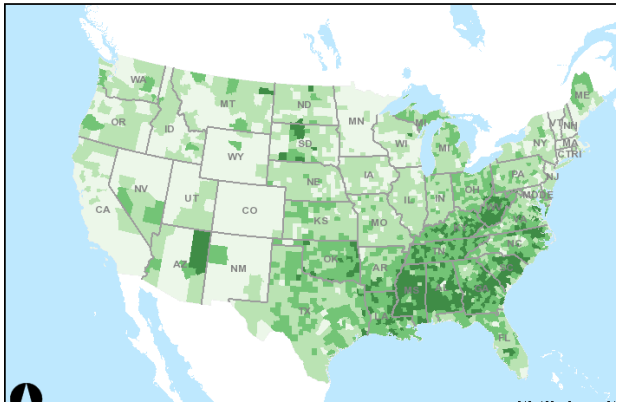


< 63% active

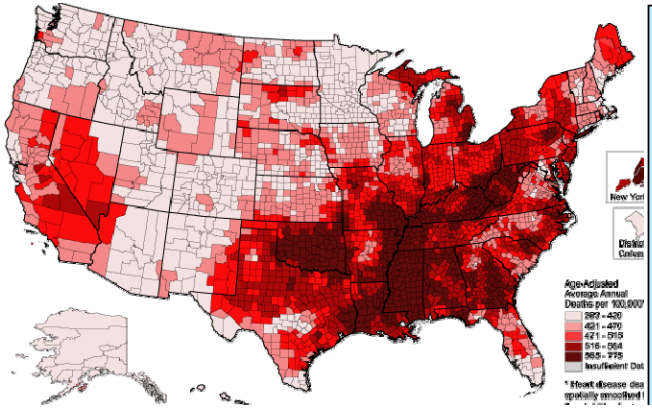
10 Most Unhappy States



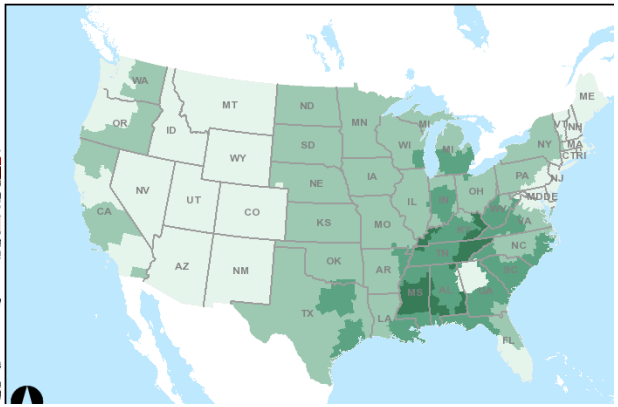
Adult Diabetes Rate



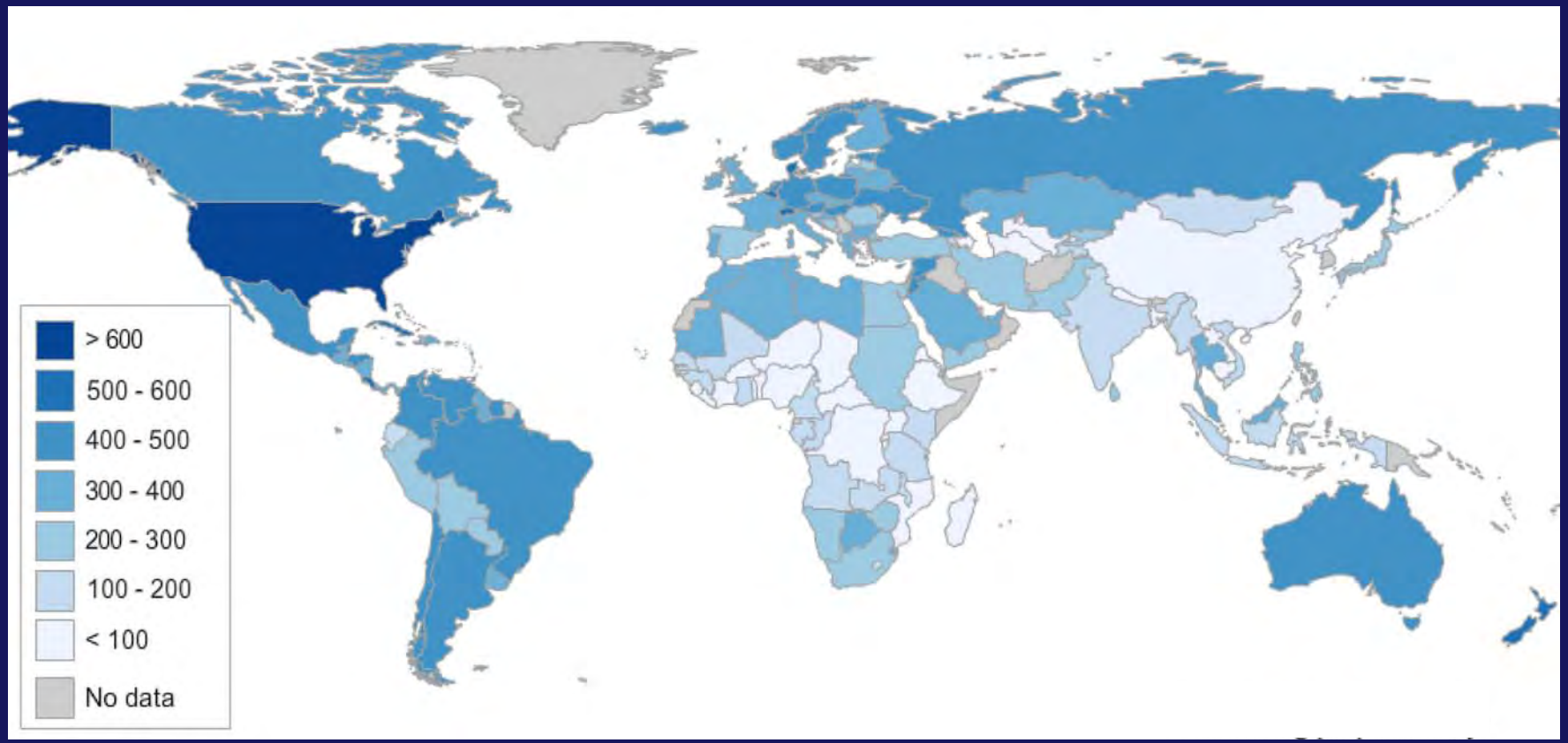
Adult Heart Disease Rate



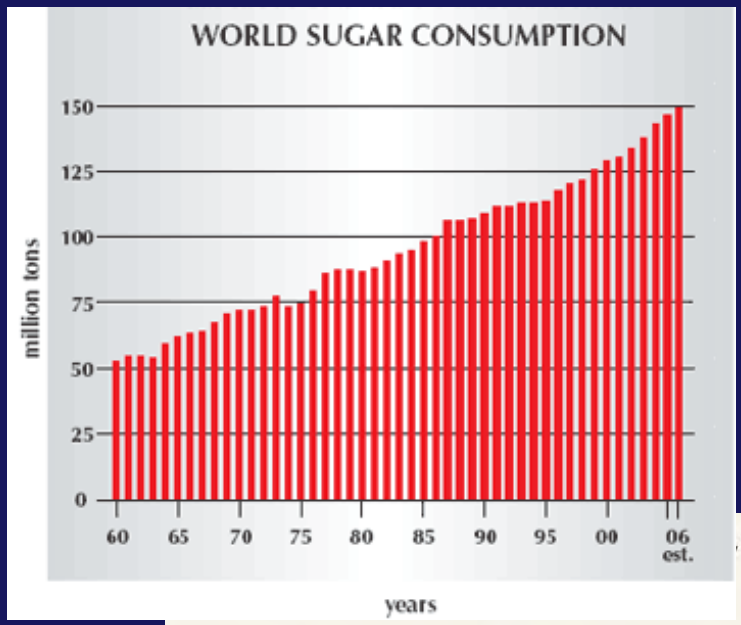
Soda Per Capita

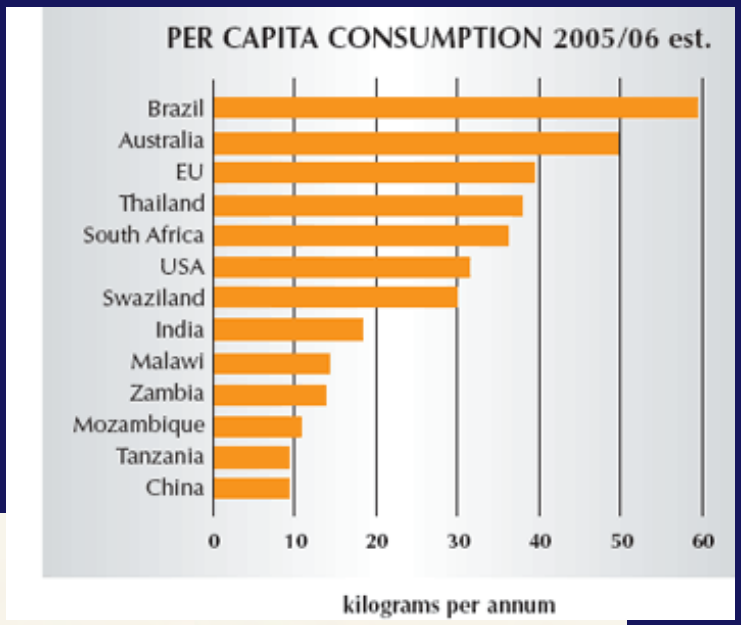


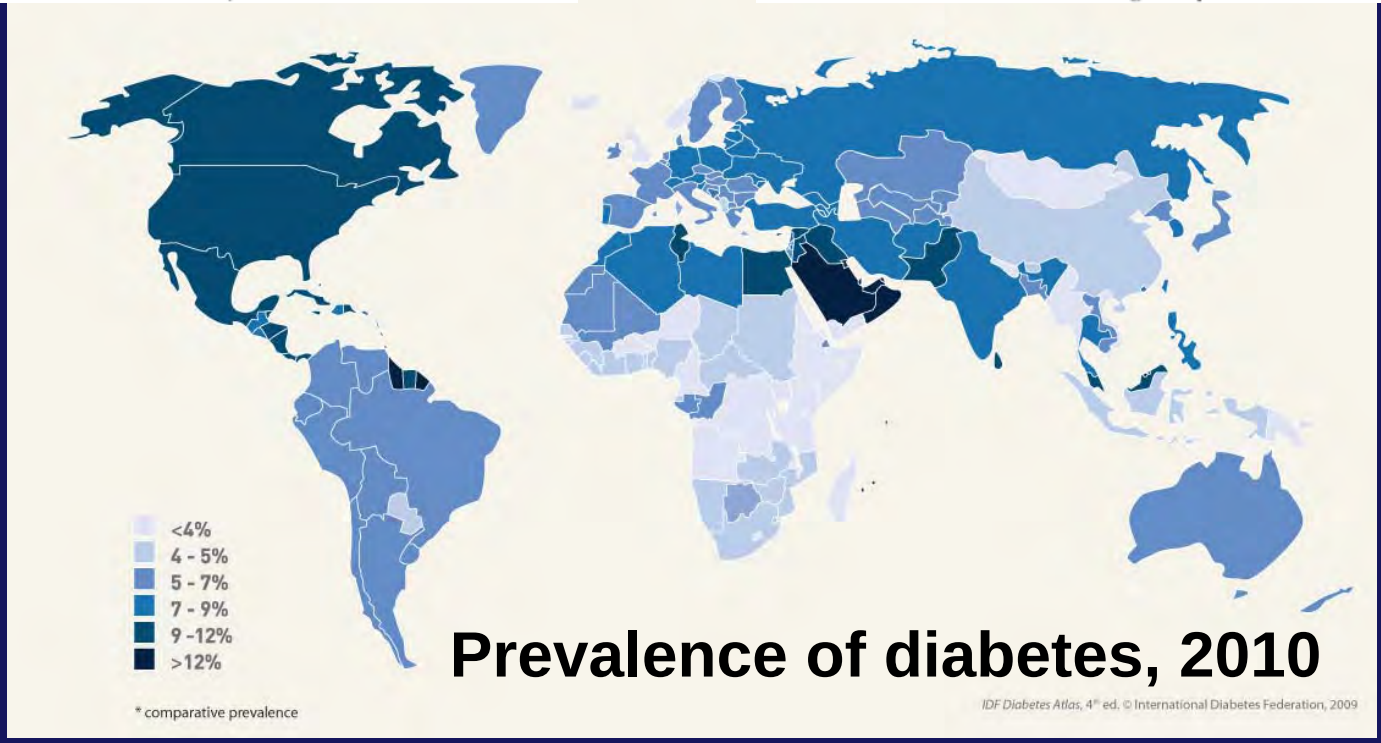
Global consumption of sugar/sugarcrops
Calories per day, 2007



Data from Food and Agriculture Organization, World Health Organization, 2007


WORLD SUGAR CONSUMPTION
million tons
150
125
100
75
50
25
0
60
65
70
75
80
85
90
95
00
06 est.
years


PER CAPITA CONSUMPTION 2005/06 est.
Brazil
Australia
EU
Thailand
South Africa
USA
Swaziland
India
Malawi
Zambia
Mozambique
Tanzania
China
0
10
20
30
40
50
60
kilograms per annum


<4%
4 - 5%
5 - 7%
7 - 9%
9 -12%
>12%
* comparative prevalence
Prevalence of diabetes, 2010
IDF Diabetes Atlas, 4th ed. © International Diabetes Federation, 2009

SSB's and BMI-adjusted risk of diabetes in EPIC-Interact (Europe)

Table 2 HRs (and 95% CIs) for type 2 diabetes according to type and amount of sweet beverage consumption in the EPIC-InterAct study

Variable and model	<1 glass[a]/ month HR	1–4 glasses[a]/ month HR[b] (95% CI)	>1–6 glasses[a]/ week HR[b] (95% CI)	≥1 glass[a]/ day HR[b] (95% CI)	*p* for trend
Juices and nectars (median intake, g/day)	(0.0)	(17.1)	(100.0)	(338.3)	
No. cases	5,837	1,702	3,425	720	
Crude model	1.00 (ref)	0.88 (0.80, 0.98)	0.89 (0.83, 0.94)	0.97 (0.85, 1.11)	0.64
Adjusted model	1.00 (ref)	0.91 (0.80, 1.02)	0.96 (0.88, 1.04)	1.00 (0.87, 1.15)	0.63
Adjusted model+EI	1.00 (ref)	0.91 (0.81, 1.02)	0.96 (0.88, 1.04)	0.99 (0.86, 1.14)	0.84
Adjusted model+EI+BMI	1.00 (ref)	0.97 (0.86, 1.10)	1.04 (0.96, 1.13)	1.06 (0.90, 1.25)	0.21
Total soft drinks[c] (median intake, g/day)	(0.0)	(20.0)	(95.1)	(413.1)	
No. cases	5,794	1,604	2,987	1,299	
Crude model	1.00 (ref)	1.21 (1.07, 1.36)	1.30 (1.18, 1.43)	1.78 (1.55, 2.04)	<0.0001
Adjusted model	1.00 (ref)	1.21 (1.07, 1.37)	1.26 (1.13, 1.42)	1.58 (1.35, 1.84)	<0.0001
Adjusted model+EI	1.00 (ref)	1.21 (1.07, 1.37)	1.27 (1.12, 1.43)	1.59 (1.35, 1.88)	<0.0001
Adjusted model+EI+BMI	1.00 (ref)	1.17 (0.97, 1.42)	1.11 (0.98, 1.26)	1.21 (1.05, 1.41)	0.0005
Sugar-sweetened soft drinks[d] (median intake, g/day)	(0.0)	(19.3)	(94.3)	(425.7)	
No. cases	3,948	964	1,599	605	
Crude model	1.00 (ref)	1.14 (0.97, 1.35)	1.16 (1.05, 1.28)	1.68 (1.40, 2.02)	<0.0001
Adjusted model	1.00 (ref)	1.13 (0.97, 1.31)	1.04 (0.94, 1.15)	1.39 (1.16, 1.67)	<0.0001
Adjusted model+EI	1.00 (ref)	1.12 (0.96, 1.31)	1.04 (0.94, 1.15)	1.39 (1.15, 1.69)	0.001
Adjusted model+EI+BMI	1.00 (ref)	1.19 (0.91, 1.56)	1.07 (0.94, 1.21)	1.29 (1.02, 1.63)	0.013
Artificially sweetened soft drinks[e] (median intake, g/day)	(0.0)	(18.3)	(89.0)	(500.0)	
No. cases	5,242	689	894	291	
Crude model	1.00 (ref)	1.09 (0.97, 1.23)	1.52 (1.36, 1.69)	1.84 (1.52, 2.23)	<0.0001
Adjusted model	1.00 (ref)	1.10 (0.93, 1.29)	1.46 (1.29, 1.65)	1.93 (1.47, 2.54)	<0.0001
Adjusted model+EI	1.00 (ref)	1.08 (0.93, 1.26)	1.46 (1.29, 1.65)	1.88 (1.44, 2.45)	<0.0001
Adjusted model+EI+BMI	1.00 (ref)	1.05 (0.81, 1.35)	1.18 (1.03, 1.35)	1.13 (0.85, 1.52)	0.24

Romaguera-Bosch et al. Diabetologia 56:1520, 2013

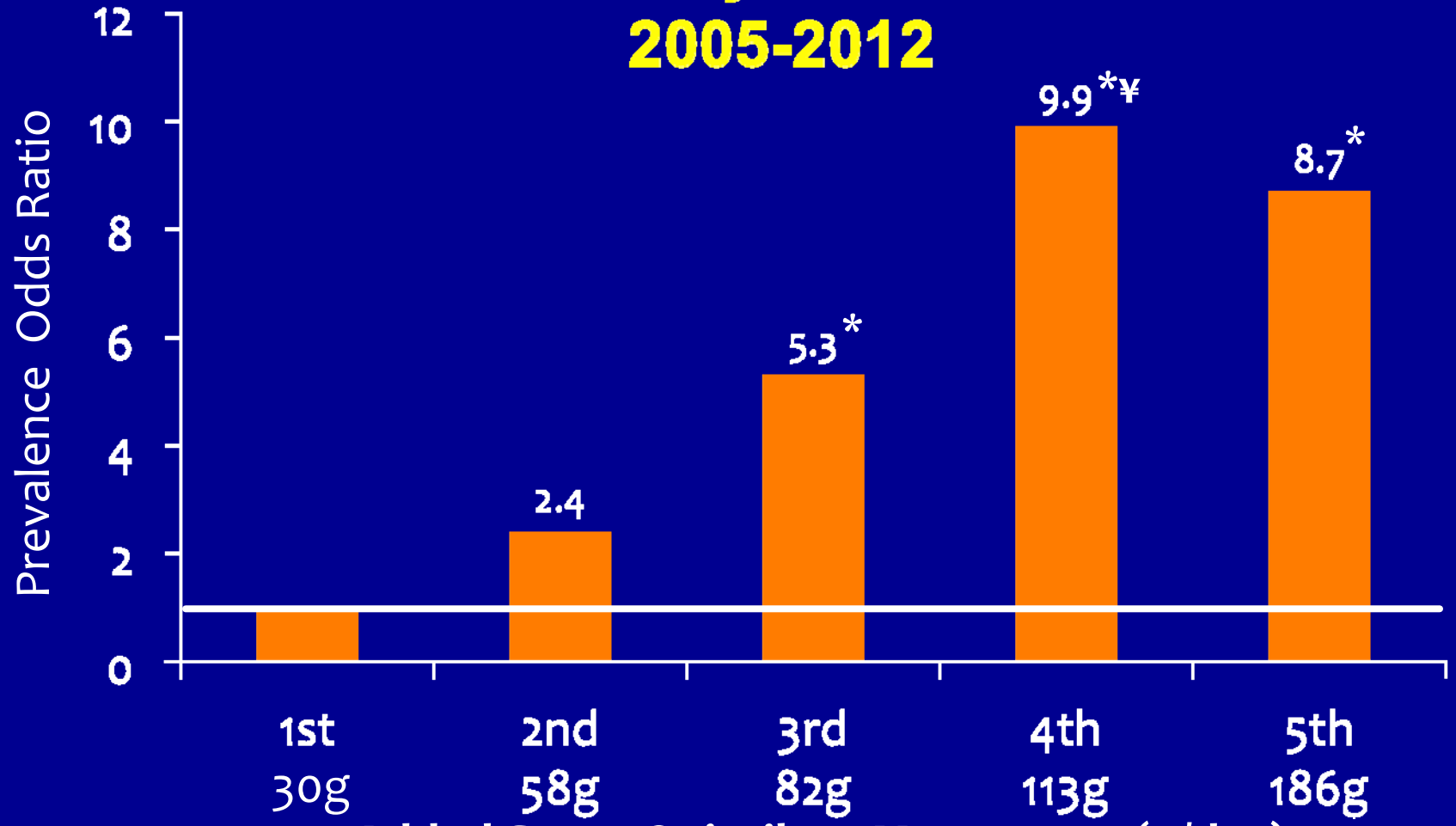
Associations between consumption of sugar sweetened beverages and fruit juice and incident type 2 diabetes: meta-analysis of prospective cohort studies

Sugar Sweetened Beverages (n=17)	Not Adjusted for Adiposity: Relative Risk	I^2		Adjusted for Adiposity: Relative Risk	I^2
Meta-analysis, crude:	1.25 (1.14 to 1.37)	89		—	—
+multivariable adjusted	1.18 (1.09 to 1.28)	89		1.13 (1.06 to 1.21)	79
+calibration for information bias	1.43 (1.20 to 1.70)	86		1.28 (1.12 to 1.46)	73
+calibration for publication bias	1.42 (1.19 to 1.69)	85		1.27 (1.10 to 1.46)	73

Fruit Juices (n=13)	Not Adjusted for Adiposity: Relative Risk	I^2		Adjusted for Adiposity: Relative Risk	I^2
Meta-analysis, crude:	0.97 (0.90 to 1.06)	79		—	
+multivariable adjusted	1.05 (0.99 to 1.11)	58		1.07 (1.01 to 1.14)	51
+calibration for information bias	1.06 (0.98 to 1.14)	49		1.10 (1.01 to 1.20)	29
+calibration for publication bias	Not detected	—		Not detected	—

Imamura et al. BMJ dx.doi.org/10.1136/bmj.h3576 (epub 21 July 2015)

Adjusted[1] Prevalence Odds Ratio for Metabolic Syndrome, NHANES 2005-2012



[1] Adjusted for age, BMI z-score, energy intake, and physical activity

* Statistically significant compared to 1st quintile ($p < 0.05$)

¥ Statistically significant compared to 2nd quintile (Wald test, $p < 0.05$)

Rodriguez et al., Public Health Nutr 2016

Sugar and Diabetes: Human Causation

An international econometric analysis of diet and diabetes

Food and Agriculture Organization (FAO); FAOSTAT

Food Supply data in kcal/capita/day calculation:

Food Supply= Σ Supply Elements - Σ Utilization Elements =

(Production + Import Quantity + Stock Variation – Export Quantity) - (Feed + Seed + Processing + Waste).

Only industrial waste factored in.

Extracted Food Supply data for 2000 and 2007:

Total Calories	Roots & Tubers, Pulses, Nuts, Vegetables
Fruits-Excluding Wine	Meat
Oils	Cereals
Sugar, Sugarcrops & Sweeteners	

International Diabetes Federation (IDF)

2000 (1st ed) and 2010 (3rd ed)

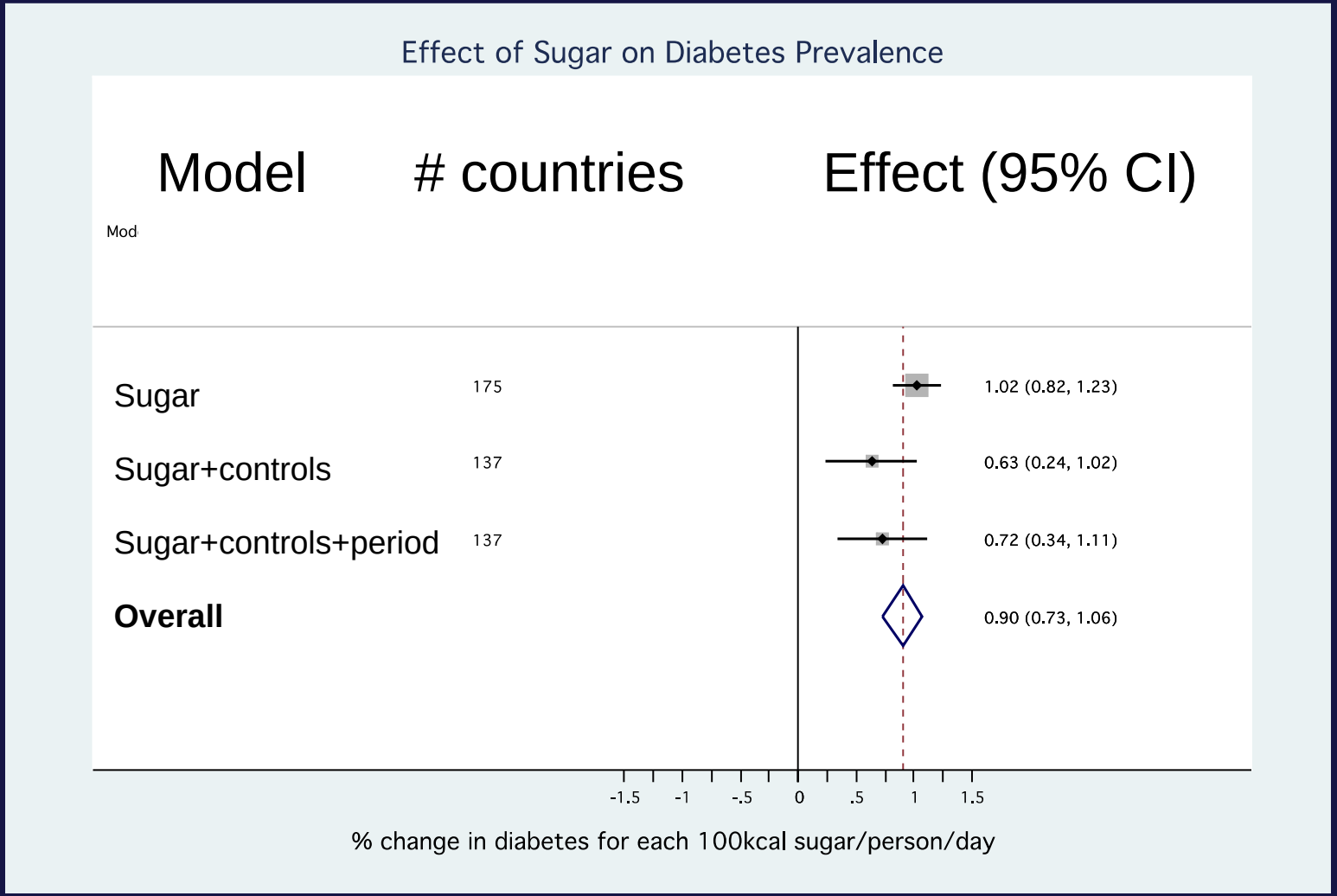
The World Bank World Development Indicators Database

GDP expressed in purchasing power parity in 2005 US dollars for comparability among countries

Basu et al. PLoS One, e57873, 2013

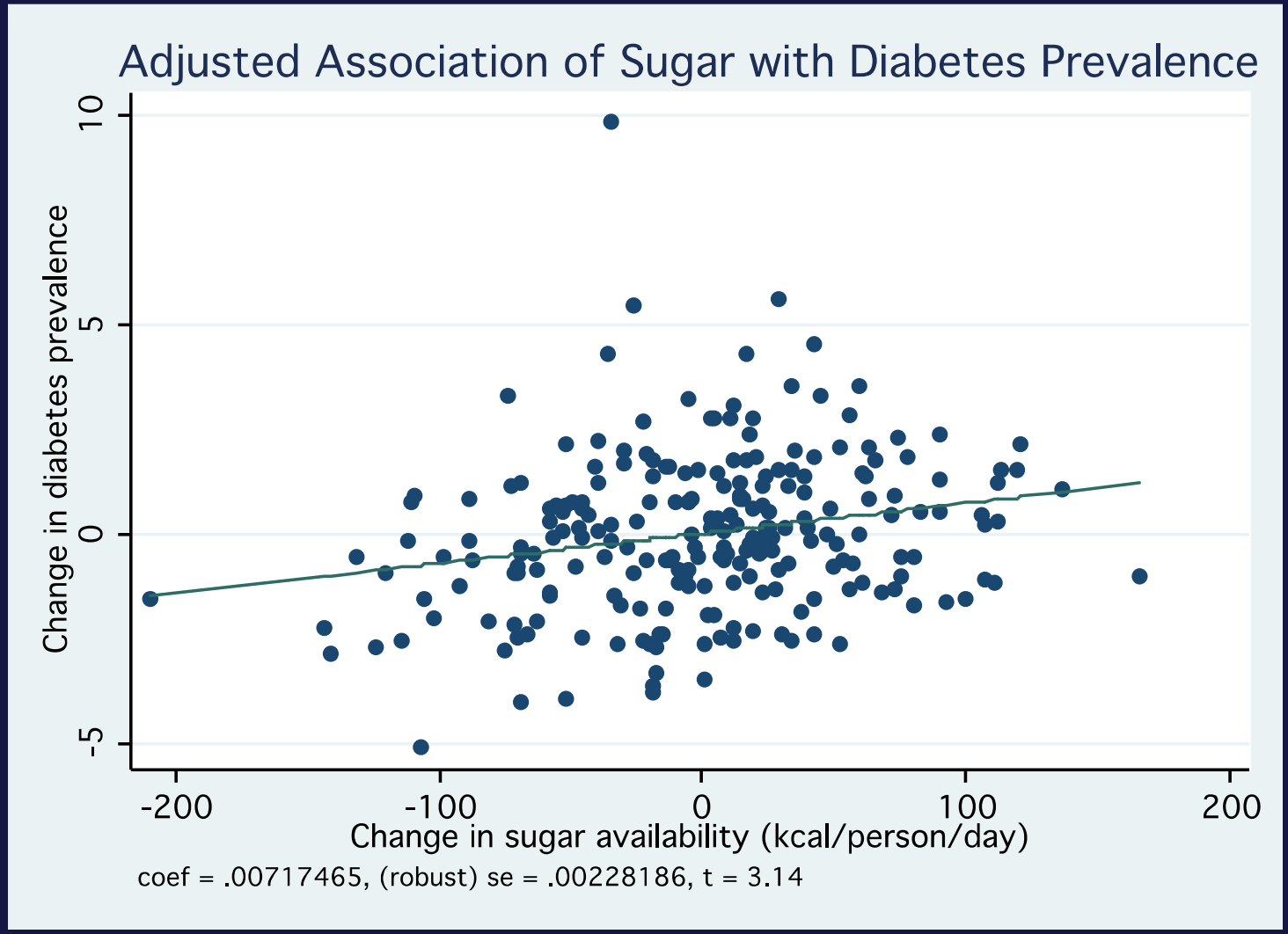
An international econometric analysis of diet and diabetes

Diabetes prevalence rose from 5.5% to 7.0% for 175 countries 2000-2010



Basu et al. PLoS One, e57873, 2013

An international econometric analysis of diet and diabetes



Basu et al. PLoS One, e57873, 2013

An international econometric analysis of diet and diabetes

Only changes in sugar availability predicted changes in diabetes prevalence

Every extra 150 calories increased diabetes prevalence by 0.1%

But if those 150 calories were a can of soda, diabetes prevalence increased 11-fold, by 1.1% (95% CI 0.03 — 1.71%, p <0.001)

This study meets the Bradford Hill criteria for Causal Medical Inference:

—dose —duration —directionality —precedence

We estimate that 25% of diabetes worldwide is explained by sugar

Basu et al. PLoS One, e57873, 2013

Interventional Proof

Original Article
PEDIATRIC OBESITY

Obesity

Isocaloric Fructose Restriction and Metabolic Improvement in Children with Obesity and Metabolic Syndrome

Robert H. Lustig[1], *Kathleen Mulligan*[2,3], *Susan M. Noworolski*[4], *Viva W. Tai*[2], *Michael J. Wen*[2], *Ayca Erkin-Cakmak*[1], *Alejandro Gugliucci*[3], and *Jean-Marc Schwarz*[5]

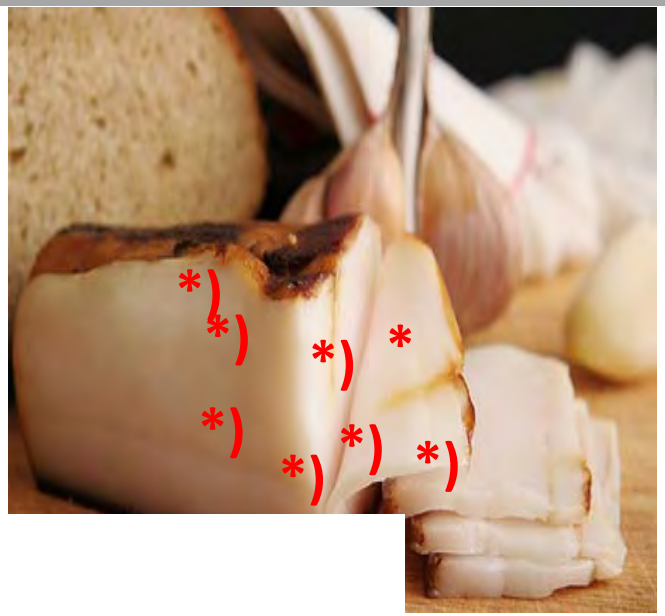
Lustig et al. Obesity 24:453, 2016

Strategy

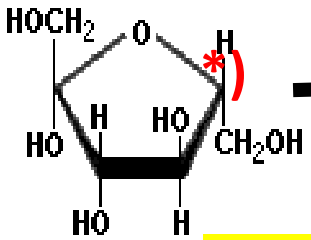
- Isocaloric fructose restriction x 9 days in children who are habitual sugar consumers
- No change in weight
- Substitute complex carbs for sugar
- Maintain baseline macronutrient composition of the the diet
- Study in PCRC at Day 0 and Day 10
- Assess changes in organ fat, *de novo* lipogenesis, and metabolic health

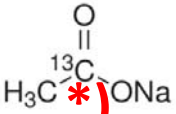
DNL is the Conversion of Dietary Carbohydrates into Lipids

Into



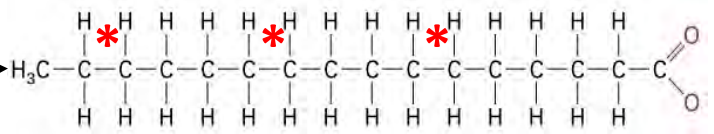
Fructose





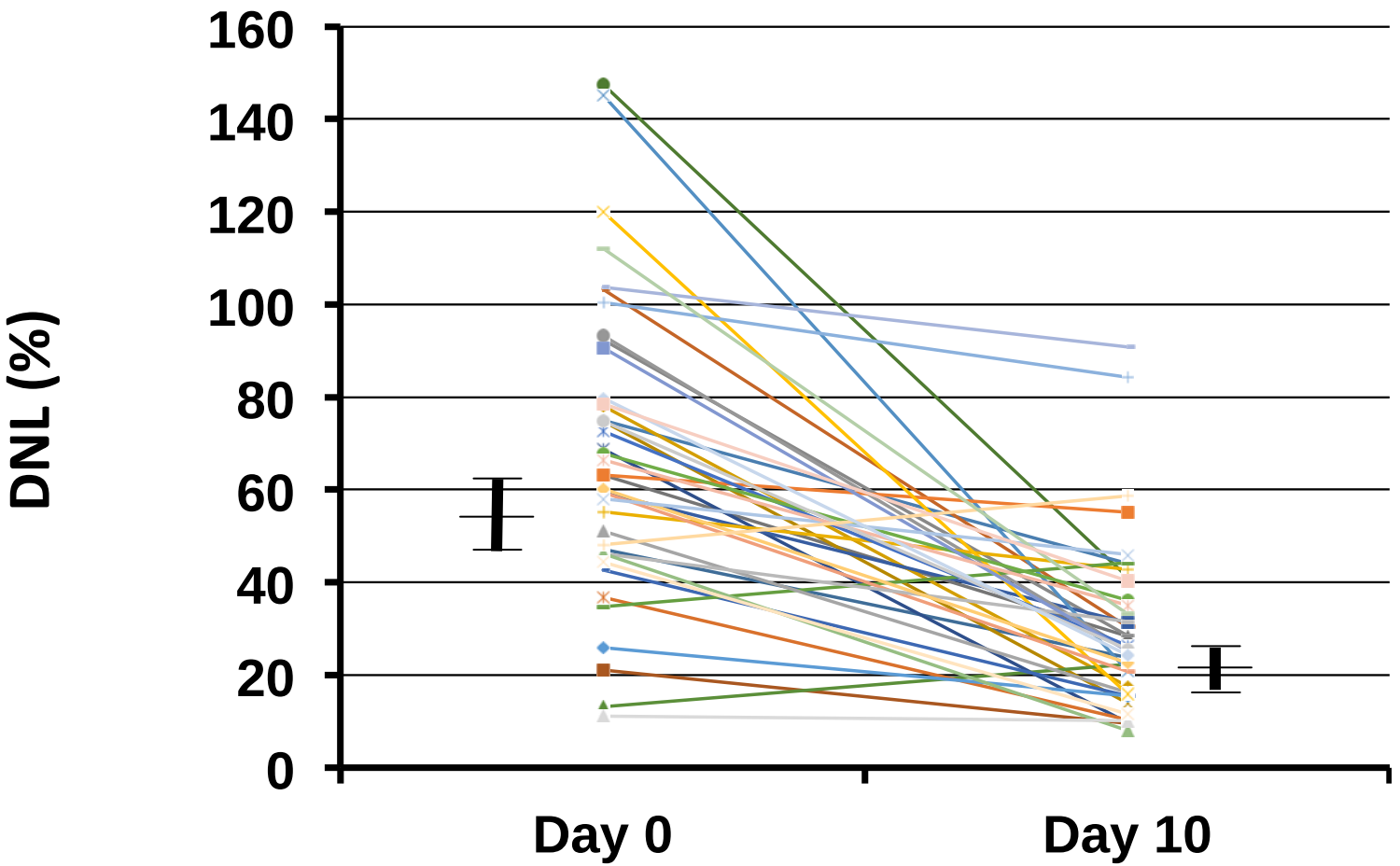
Acetate

Palmitate

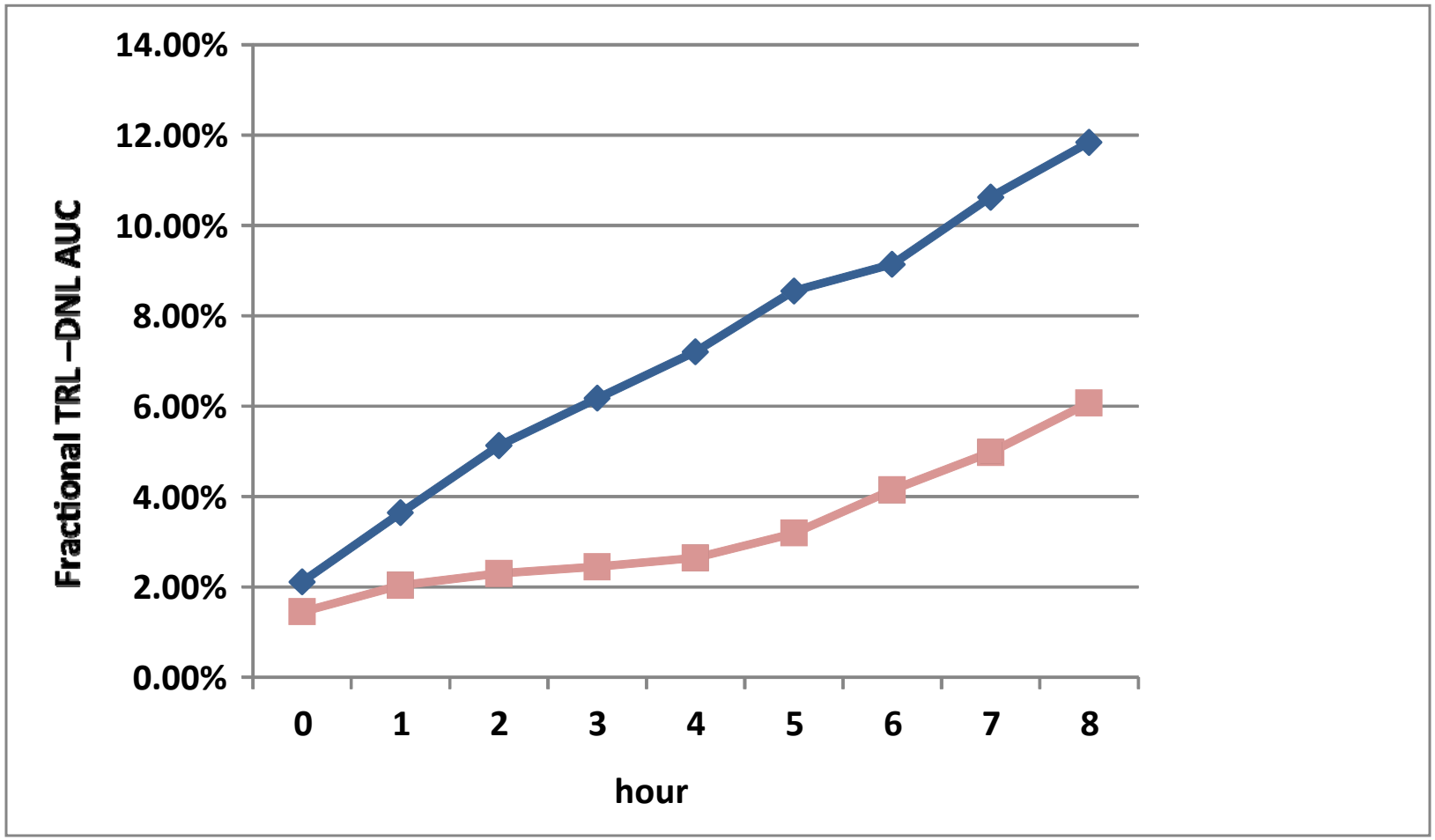


New Tracer Method using MIDA: Hellerstein and Neese, AJP 1999

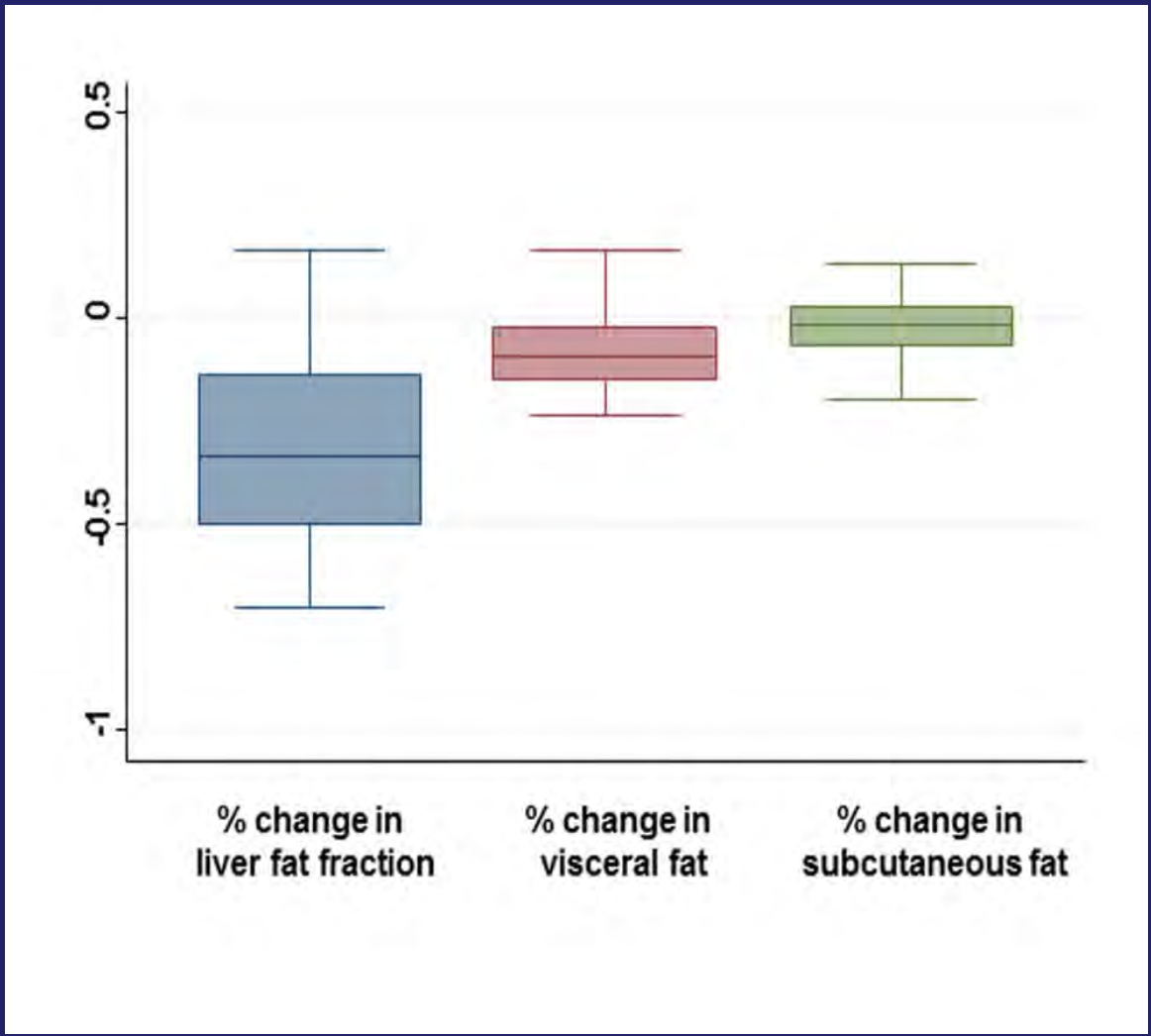
DNL AUC Pre and Post Fructose Restriction



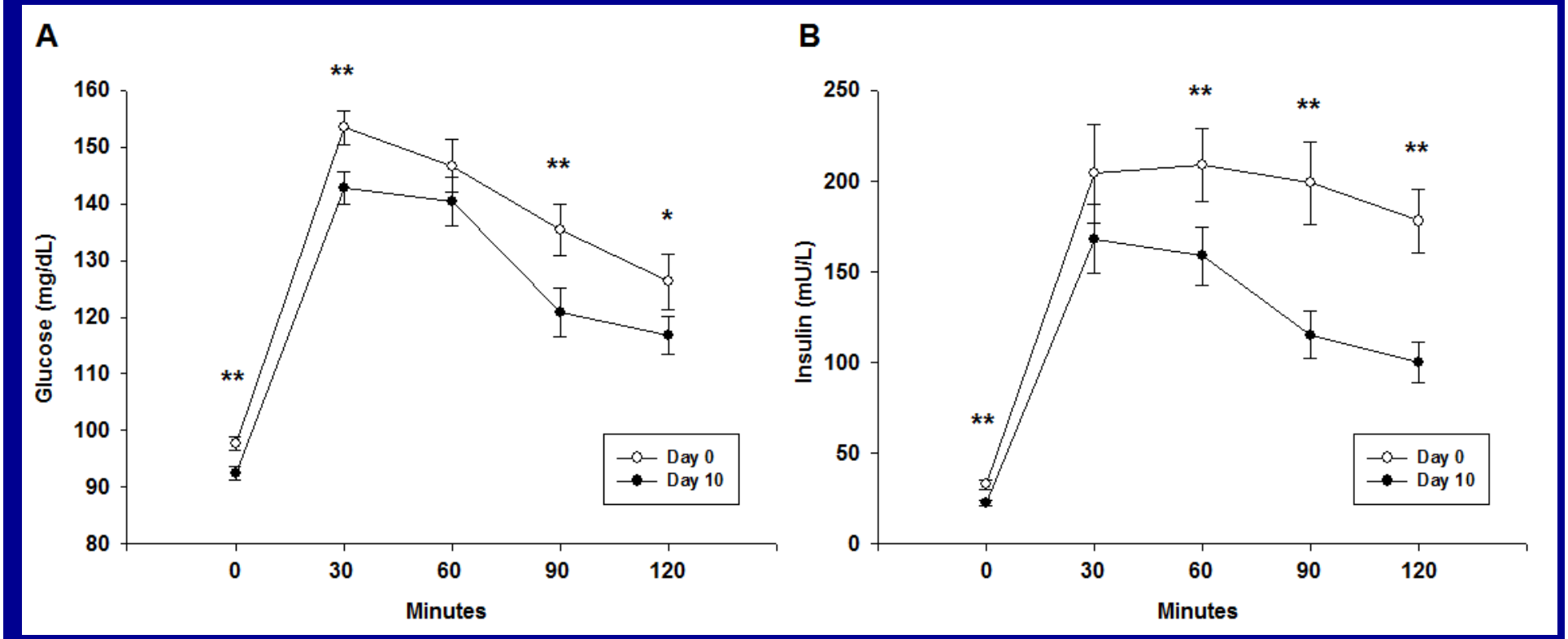
Triglyceride-rich Lipoprotein DNL AUC (n=17)



Changes in liver, visceral, and subcutaneous fat (n = 37)



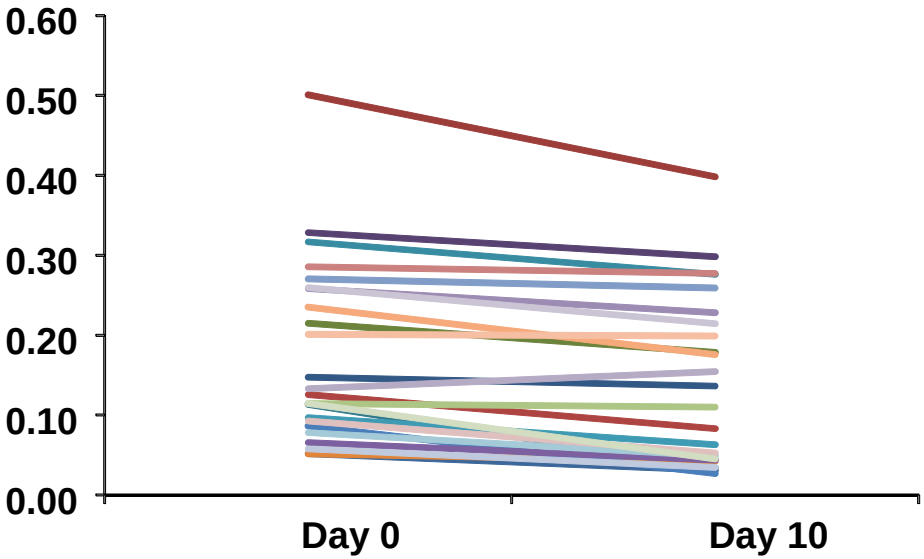
Oral glucose tolerance test before and after isocaloric fructose restriction



Lustig et al. Obesity (in press)

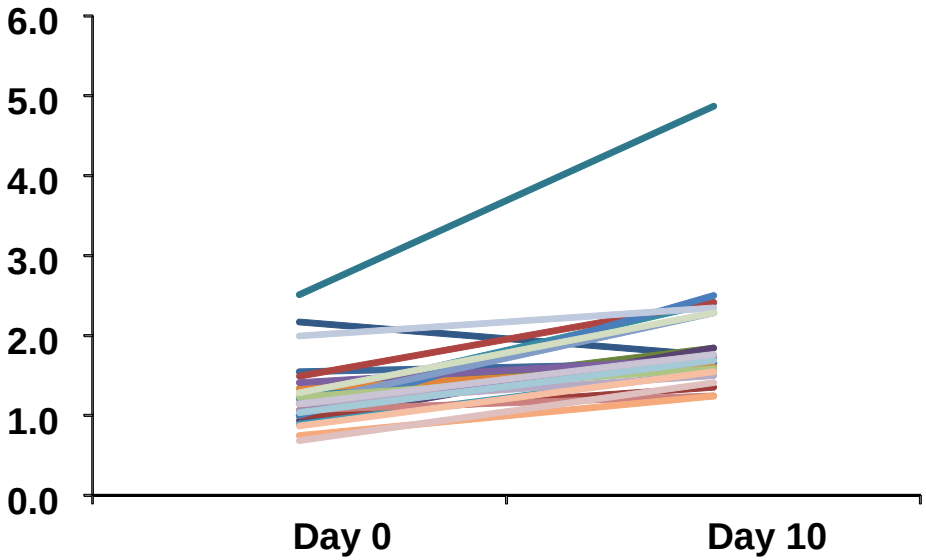
For pediatric subjects with hepatic steatosis (n = 25)

Change in Liver Fat and Insulin Sensitivity



Liver Fat

-30% (-50, -20); p <0.001



Composite Insulin Sensitivity Index

+0.65 (0.41, 0.88); p <0.001

Adjusted for change in weight

Lustig et al. Obesity Society, Nov. 4, 2015

Correlation between Insulin Sensitivity & Liver Fat vs Visceral Fat

Spearman R	Day 0		Day 10		Change in fat (Absolute)	
	Liver Fat Fraction	Visceral Fat	Liver Fat Fraction	Visceral Fat	Liver Fat Fraction	Visceral Fat
Insulin Sensitivity (CISI) DAY 0	- 0.36 §	- 0.57*				
Insulin Sensitivity (CISI) DAY 10			- 0.28	- 0.34 §		
Change in Insulin sensitivity (ΔCISI)					**- 0.54***	0.06

* $p < 0.05$

§ $0.05 < p < 0.1$

Lustig et al. Obesity Society, Nov. 4, 2015

What the data say

- Prospective correlational data demonstrate **associations** between added sugar and heart disease and diabetes, **exclusive of calories or obesity**

- Econometric data show **causal medical inference** for added sugar and diabetes, **exclusive of calories or obesity**

- Interventional **isocaloric glucose for fructose exchange** shows improvements in fatty liver disease, insulin resistance and metabolic health in children in 10 days, and insulin resistance is driven by liver fat

Recognition at the American Heart Association

AHA Scientific Statement

Dietary Sugars Intake and Cardiovascular Health

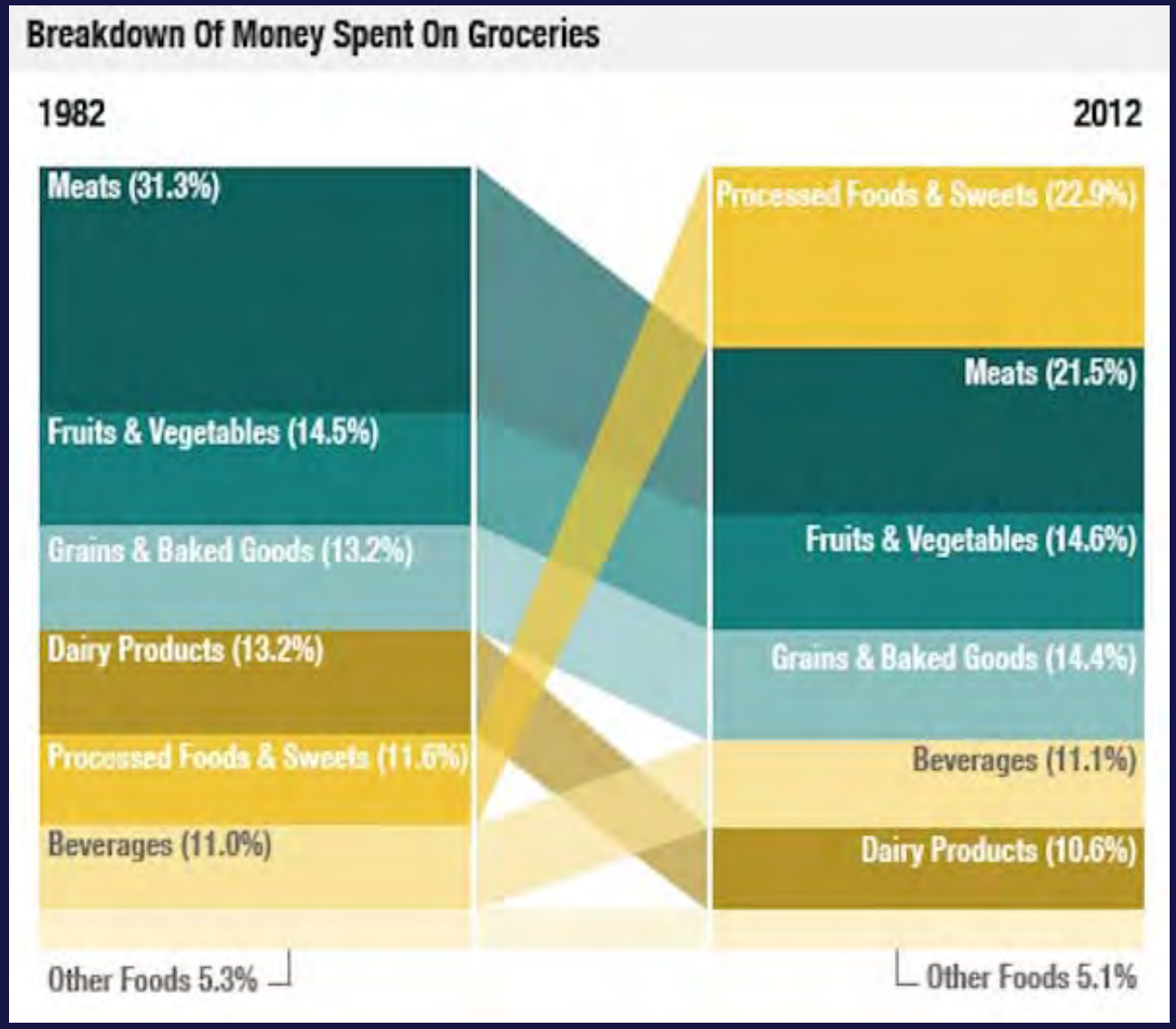
A Scientific Statement From the American Heart Association

Rachel K. Johnson, PhD, MPH, RD, Chair; Lawrence J. Appel, MD, MPH, FAHA; Michael Brands, PhD, FAHA; Barbara V. Howard, PhD, FAHA; Michael Lefevre, PhD, FAHA; Robert H. Lustig, MD; Frank Sacks, MD, FAHA; Lyn M. Steffen, PhD, MPH, RD, FAHA; Judith Wylie-Rosett, EdD, RD; on behalf of the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity, and Metabolism and the Council on Epidemiology and Prevention

Recommends reduction in sugar intake from 22 tsp/day to 9 tsp/day (males) and 6 tsp/day (females)

Circulation 120:1011, 2009

How our food dollars have been reallocated

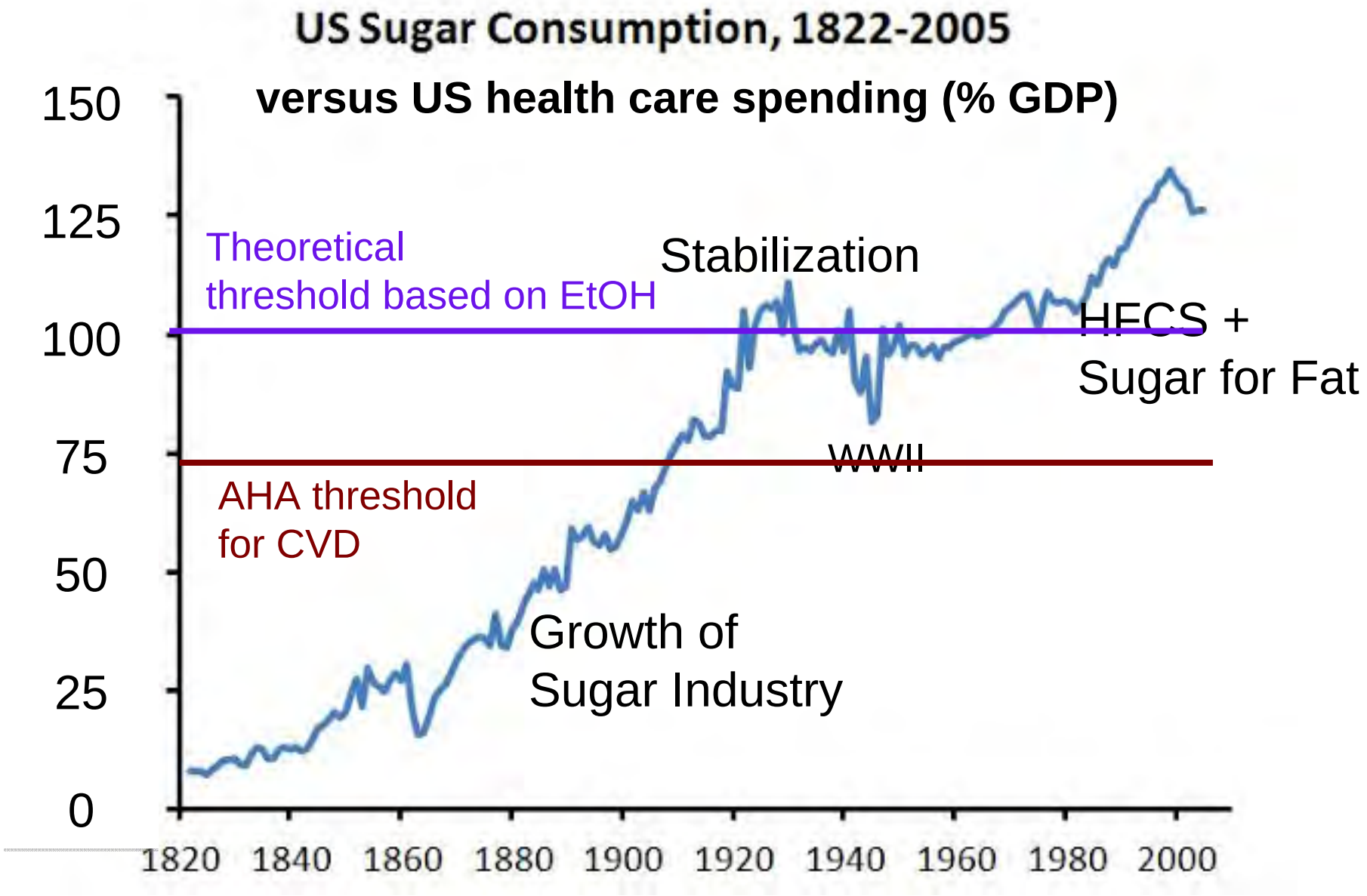


Philpott, Mother Jones 2012 (from Bureau of Labor Statistics)

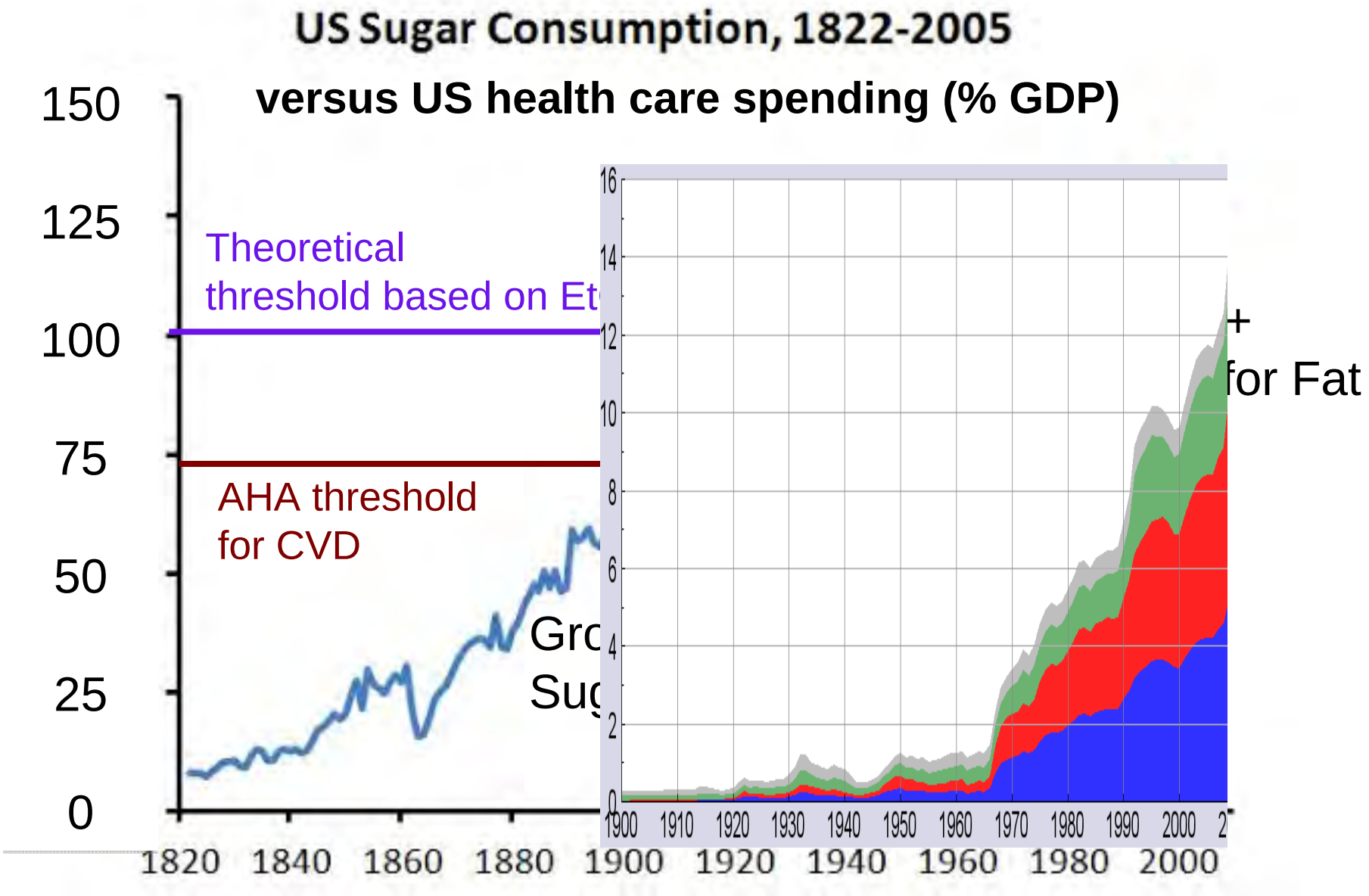
An inconvenient truth

The “medical” model isn’t the medical model;

It’s the “sugar” model



U.S. Commerce Service 1822-1910, combined with Economic Research Service, USDA 1910-2010
http://ushealthcarespending.gov



U.S. Commerce Service 1822-1910, combined with Economic Research Service, USDA 1910-2010
http://ushealthcarespending.gov

DIARIO FUERZA DEL ESTADO DE MÉXICO

ESPECIALISTAS PROPONEN UN IMPUESTO MÁS ALTO A REFRESCOS

octubre 16, 2013

La organización civil el Poder del Consumidor y especialistas recomendaron imponer un gravamen de dos pesos a las bebidas azucaradas, a fin de disminuir su consumo y evitar daños en la salud como el sobrepeso y la diabetes.


YES ON D
Berkeley vs. Big Soda

Health | Wed Nov 11, 2015 7:08pm EST

Related: HEALT

Diabetes experts tell G20 to tax sugar to save lives and money

BY BEN HIRSCHLER

Diabetes experts called on world leaders on Thursday to use sugar taxes to fight obesity, arguing such a move would save lives and slash healthcare budgets.

Ahead of a meeting of G20 leaders this weekend, the International Diabetes Federation (IDF) wants the dual epidemics of obesity and diabetes to be placed on the global agenda alongside major geopolitical and financial issues.

Budget 2016

George Osborne unveils sugar tax in eighth budget as growth forecast falls

- Proceeds of levy on soft drinks to fund school sports
- Cuts to business rates, capital gains and corporation tax
- Income tax personal allowance increased
- Growth forecast down from 2.4% to 2%

Katie Allen, Anushka Asthana and Rowena Mason

Wednesday 16 March 2016 10.58 EDT

Osborne announces soft drinks sugar tax and tax-free personal allowance – budget highlights video

George Osborne has unveiled a new tax on sugary drinks, such as Coca-Cola, Red Bull and Irn Bru, pledging to use the takings to provide more sports funding for schools.

Policy Efforts in Northern California: SSB Distributor Tax

Join the Movement:

Endorse

Contribute

Share on Social Media

Oakland:

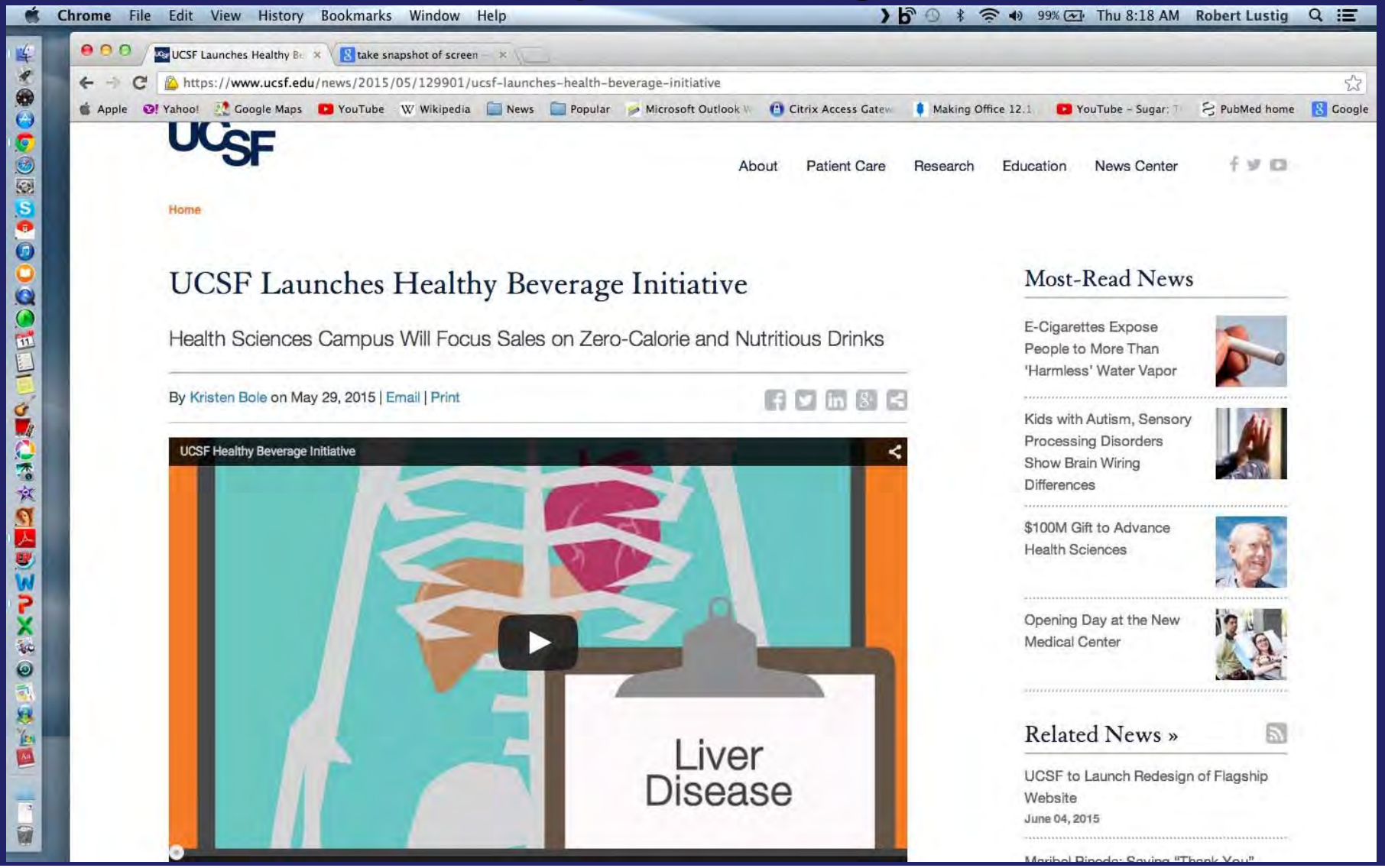
www.yes4healthyoaklandchildren.com

San Francisco

www.sfunitedtoreducediabetes.com

Proposal #1

UCSF Healthy Beverage Initiative



Proposal #2

Type 2 Diabetes should be renamed:

PROCESSED FOOD DISEASE

Proposal #3

Rollback the subsidies for processed food:

CORN
WHEAT
SOY
SUGAR

Proposal #4

REAL FOOD APPROVED

Proposal #5

Remove Sugar from the FDA "Generally Recognized as Safe" (GRAS) List

Conclusions

- The dentists knew about fructose/sugar toxicity long before the doctors did, but fluoride allowed for “selective amnesia”
- Although dietary fat can induce NAFLD, fat ingestion does does not explain the current epidemic of NAFLD/NASH; but fructose does
- A calorie is NOT a calorie, and fructose is NOT glucose
- Fructose is “alcohol without the buzz”; it is a dose-dependent chronic hepatotoxin ; NASH and ASH share the same pathogenesis
- Evolution doesn’t lie: the overlap between tooth and ilver decay inform us about the changes in our environment, and what to do about them
- But understanding the science often doesn’t translate into policy

BACON 'WANTED TO BE LIKE SCARFACE'

Surrey Six witness says Red Scorpions leader sought power and control NEWS A6

CANUCKS SUCCUMB IN WILD ONE TO ISLES

SPORTS A28-30

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Outdoor

FED UP

A FILM BY STEPHANIE SOECHTIG

sundance

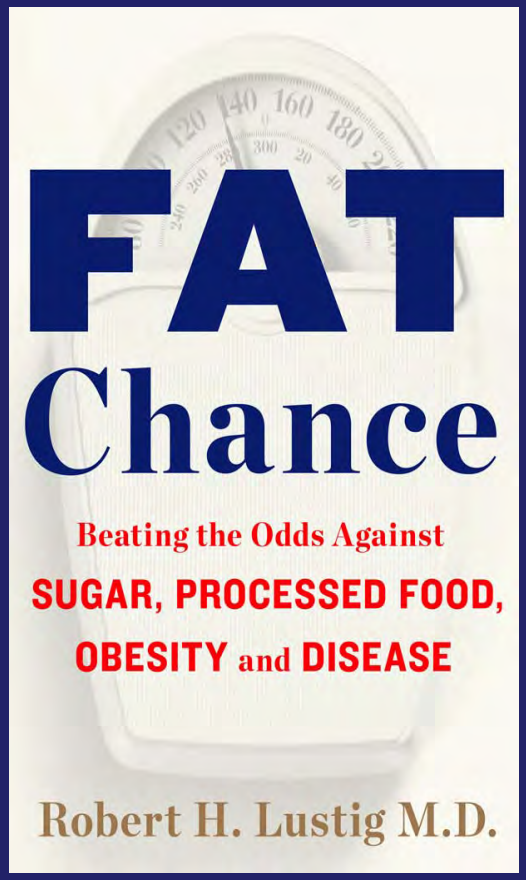
On NETFLIX

ABOUT PRESSKIT FILMMAKERS CONTACT

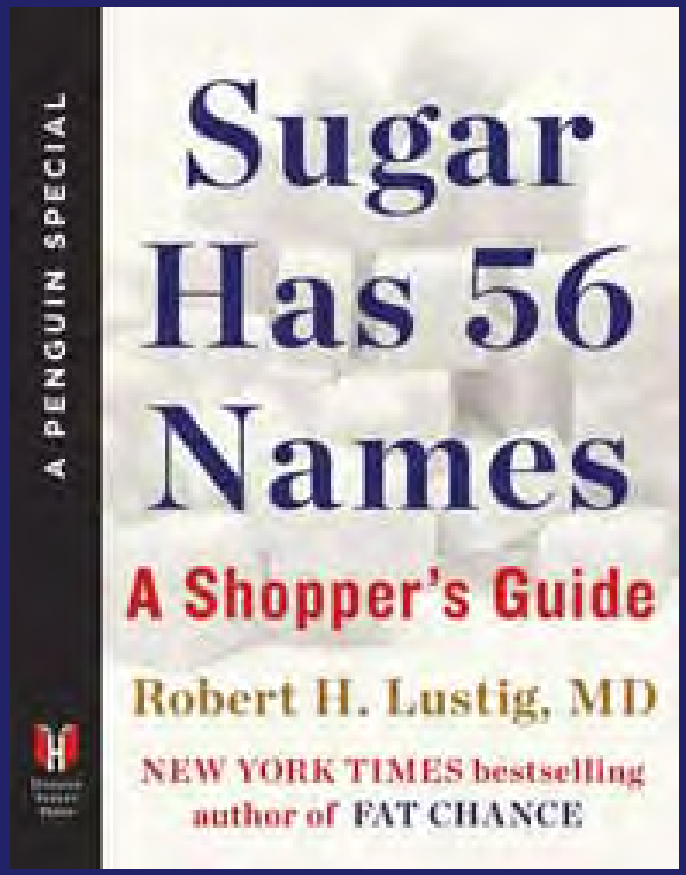
Fed Up blows the lid off everything we thought we knew about food and weight loss, revealing a 30-year campaign by the food industry, aided by the U.S. government, to mislead and confuse the American public, resulting in one of the largest health epidemics in history.

Canadian Documentary On NETFLIX

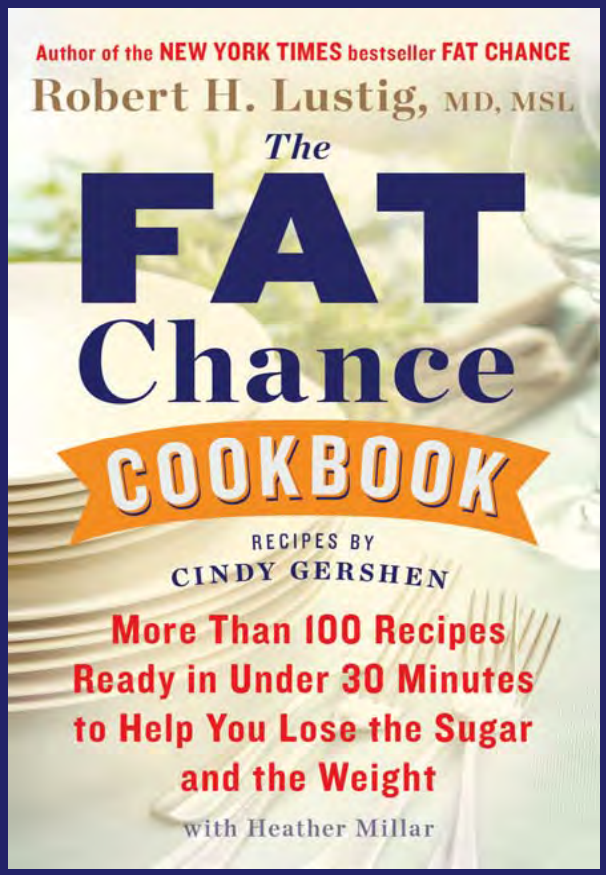
Further reading



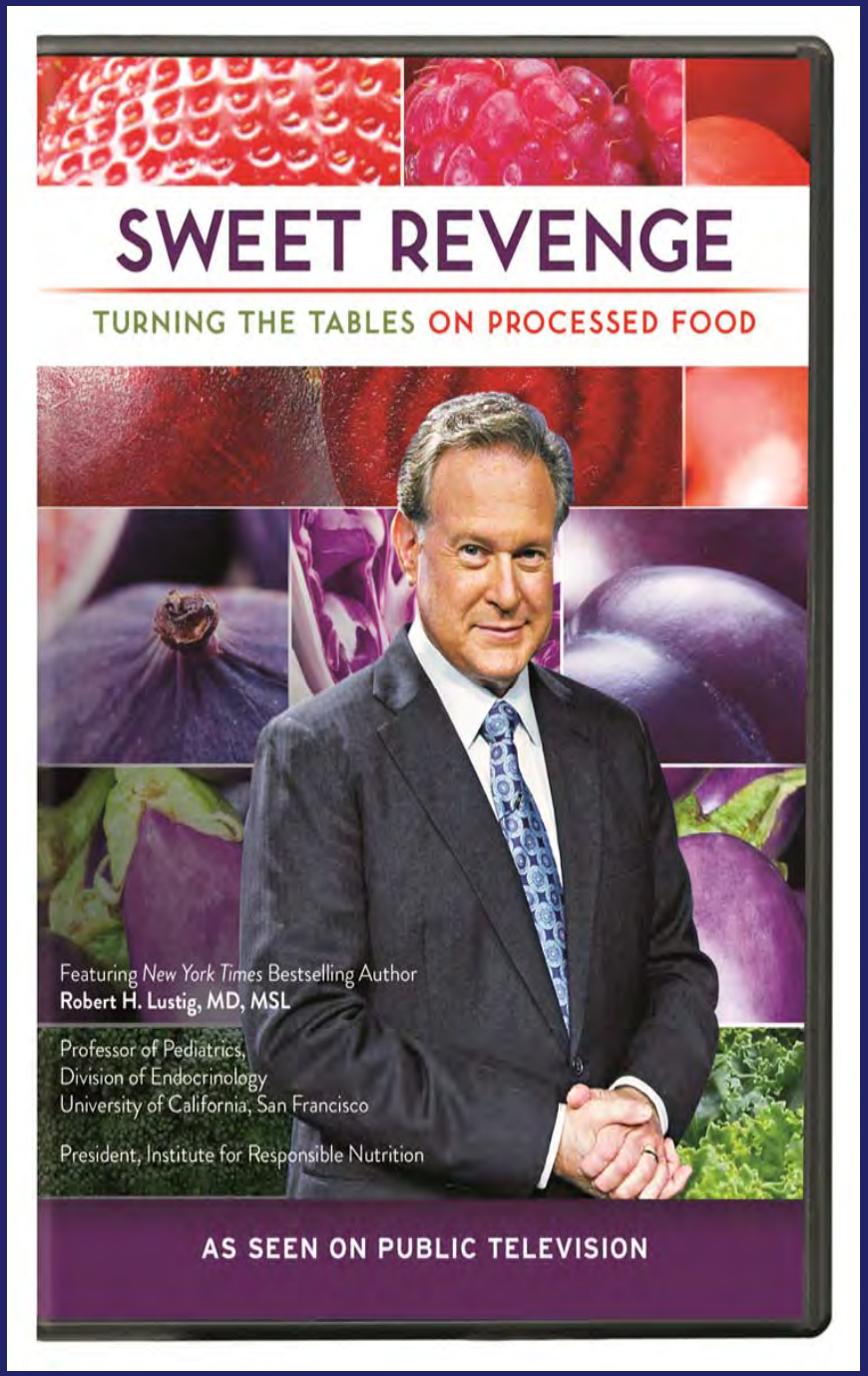
Hudson Street Press
(Penguin USA)



E-book Hudson Street Press
Sept 3, 2013



Hudson Street Press
Jan 1, 2014



Public Television Special, USA
Now also in Spanish
“Dulce Venganza”

We have started a non-profit to provide

medical, nutritional and legal analysis and consultation

to promote personal and public health vs. Big Food

responsiblefoods.org

Collaborators

UCSF Weight Assessment for Teen and Child Health (WATCH)

Andrea Garber, Ph.D., R.D.
Patrika Tsai, M.D., M.P.H.
Stephanie Nguyen, M.D. M.A.S.
Emily Perito, M.D.
Jung Sub Lim, M.D., Ph.D.

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Martin Renner, Ph.D.