Delhi, 6th March 2019 workshop
SUGAR AND HEALTH

SUGAR:
THE SCAPEGOAT OF NEW MILLENNIUM

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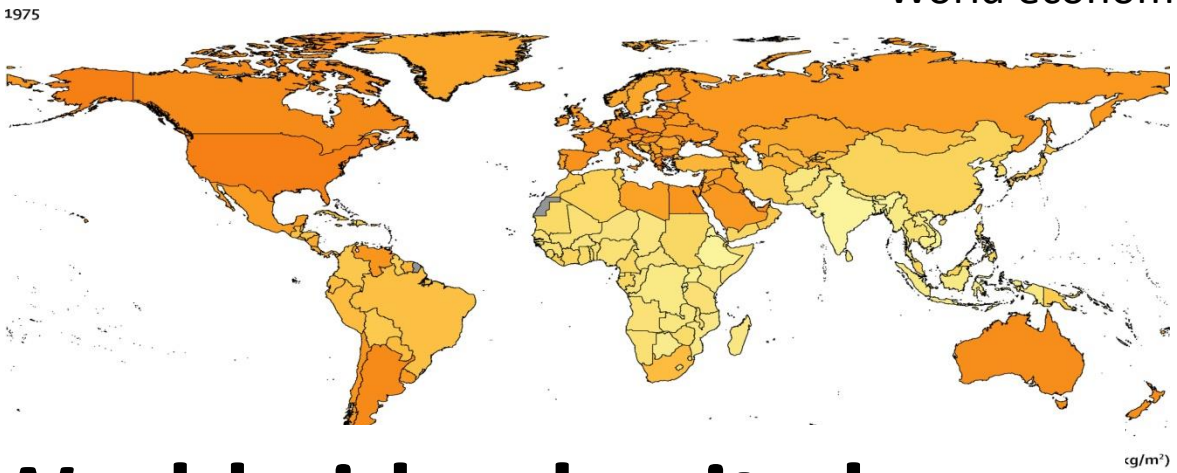
Worldwide overweight/obesity

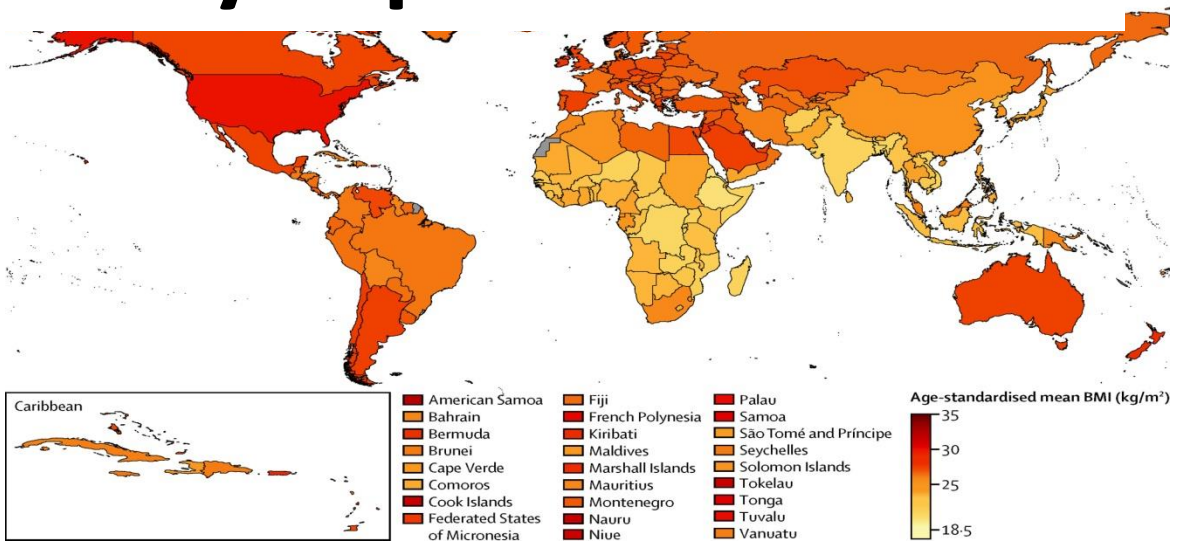
World economic forum data

1975



Worldwide obesity has nearly tripled since 1975

2014



Key facts
(WHO 2018)

Raised BMI is a major risk factor for noncommunicable diseases such as:

- **cardiovascular diseases (mainly heart disease and stroke), which were the leading cause of death in 2012;**
- **diabetes;**
- **musculoskeletal disorders (especially osteoarthritis – a highly disabling degenerative disease of the joints);**
- **some cancers (including endometrial, breast, ovarian, prostate, liver, gallbladder, kidney, and colon).**
- **The risk for these noncommunicable diseases increases, with increases in BMI.**

Key facts
(WHO 2018)

The fundamental cause of obesity and overweight is an energy imbalance between calories consumed and calories expended. Globally, there has been:

- **an increased intake of energy-dense foods that are high in fat**
- **an increase in physical inactivity due to the increasingly sedentary nature of many forms of work, changing modes of transportation, and increasing urbanization**
- **Changes in dietary and physical activity patterns are often the result of environmental and societal changes associated with development and lack of supportive policies in sectors such as health, agriculture, transport, urban planning, environment, food processing, distribution, marketing, and education.**

Sir Winston L. Spencer-Churchill (1874 – 1965)

James F. Fixx (1932 – 1984)

MICROBIOTA

- The number of bacteria present in the intestine exceeds 10 times the total of human cells.
- The bacterial genome in the intestine (microbioma) exceeds 150 times that of humans
- WE ARE A SUPERORGANISM

Unhealthy microbiota related diseases

Diseases associated with GI microbiota abnormalities.

Disease	Features	References
Rheumatoid arthritis	Chronic, inflammatory auto-immune disorder displaying reduced *Bifidiobacteria* and *Bacteroidetes-Porphyromonas-Prevotella* group, *Bacteroidetes fragilis* subgroup, and *Eubacterium rectale-Clostridium coccoides* group, and increased *Lactobacillus*.	Wu et al., 2016
Inflammatory bowel disease	Dysbiotic inflammatory response to intestinal microbes. Increased numbers of innate immunity cells (neutrophils, macrophages, dendritic cells, and natural killer T cells) and adaptive immunity cells (B and T lymphocytes), which enact immune tolerance or defense against the intestinal microbiota.	Abraham and Cho, 2009; Halfvarson et al., 2016
Irritable bowel syndrome	Enrichment of Firmicutes and reduction of Bacteroidetes.	Krogius-Kurikka et al., 2009; Rajilić-Stojanović et al., 2011; Jeffery et al., 2012; Kennedy et al., 2014
Ulcerative colitis	Reduction of Bifidobacteria. Inflammation confined to the mucosa of the colon.	Abraham and Cho, 2009; Duranti et al., 2016
Crohn's disease	Reduction of Firmicutes and Bacteroidetes. Transmural inflammation.	Eckburg and Relman, 2007; Abraham and Cho, 2009
Ileal Crohn's disease	A form of Crohn's disease typified by decreased Paneth cell α-defensins, weakened antibacterial activity of the ileal mucosa, leading to bacterial composition changes in the microbiota.	Wehkamp et al., 2005
Type 1 diabetes	Auto-immunity against pancreatic β-cells (normally producing insulin) in genetically predisposed individuals. Defective development or alterations of the microbiota may result in dysregulated immunity with autoimmune β-cells destruction, and/or increased leakiness of the gut epithelial barrier. Decreased microbiome diversity.	Atkinson and Eisenbarth, 2001; Bosi et al., 2006; Vaarala et al., 2008; Atkinson et al., 2013; Dunne et al., 2014
Asthma	The airway microbiome is affected by outbreaks of *Chlamydophila pneumoniae* during the development of bronchitis and pneumonia. The GI microbiota is affected by the environmental exposure of microbes, especially early in life, which in turn affects the maturation of immune function to protect against allergic sensitization.	Hahn et al., 1991; Huang and Boushey, 2015
Obesity	Shift in the proportion of Firmicutes and Bacteroidetes with a significant increase of the former, leading to obesity in conjunction with poor diet.	Consortium THMP, 2012; John and Mullin, 2016
Obesity and gastric bypass	Significantly fewer *Firmicutes* compared to obese and healthy patients. Increase in *Gammaproteobacteria* in post-gastric-bypass patients.	Zhang et al., 2009
Cancer (various)	Carcinogenesis may develop in response to epithelial injury and inflammation from infectious agents, genetic mechanism, or pathogens (e.g., *Helicobacter pylori, Salmonella enterica, Borrelia burgdorferi, Chlamydia psittaci*).	Virchow, 1863; Balkwill and Mantovani, 2001; Grivennikov et al., 2010; Moore and Chang, 2010; Trinchieri, 2012; Schwabe and Jobin, 2013
Typhoid fever	Caused by infection of *Salmonella* species (spp.) *S. enterica* serovar Typhi (*S.* Typhi).	Rabsch et al., 2001; Crump and Mintz, 2010; Graham, 2010; Ahmer and Gunn, 2011
Food poisoning and foodborne pathogens	Opportunistic pathogens (e.g., *Campylobacter, Salmonella, E. coli, Shigella, Cronobacter, Listeria, Cryptosporidium*, MRSA, etc.) disrupt the equilibrium of the microbiome leading to dysbiosis, loss of host bacterial diversity and multiple diseases.	Brown et al., 2012; Shim, 2013; Carriere et al., 2015; Josephs-Spaulding et al., 2016

Susannah Selber-Hnatiw Human Gut Microbiota: Toward an Ecology of Disease Front Microbiol 2017.

Unhealthy microbiota related diseases

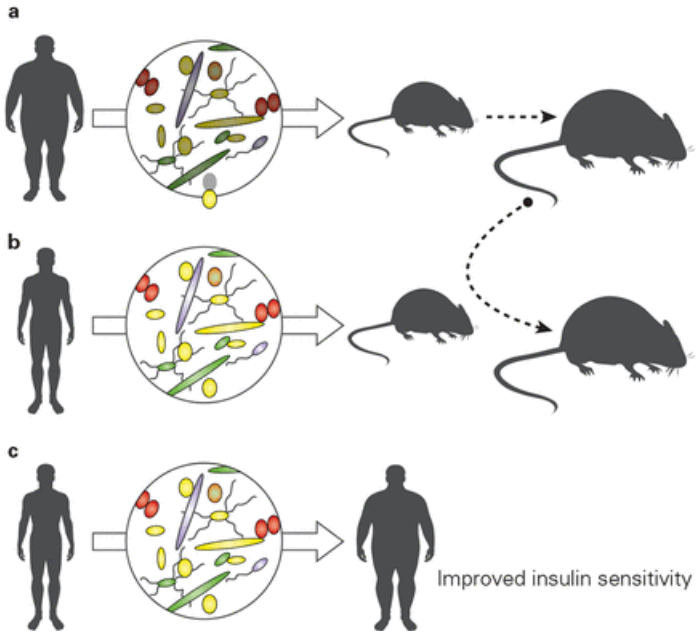
Diseases associated with GI microbiota abnormalities.

Malnutrition	Decrease or absence of species that either efficiently process food categories or produce vitamins may lead to reduced nutrient absorption and inflammation. *Enterobacteriaceae* overgrowth may result in epithelial damage, diarrhea, and reduced nutrient absorption.	Mohan et al., 2006; Lupp et al., 2007; Round and Mazmanian, 2009; Kane et al., 2015
Clostridium difficile Infection	A nosocomial pathogen, CDI is associated with epithelial inflammation and necrosis of the colon, diarrhea, pseudomembranous colitis and toxic megacolon. Antibiotic exposure may increase risk of re-infection.	Heinlen and Ballard, 2010; Khanna et al., 2016
Peptic ulcer disease	*Helicobacter pylori* are H_2- receptor antagonists responsible for peptic ulcers, found in the stomach or duodenum. *H. pylori* was found to modify epithelial proliferation and apoptosis in gastric mucosa, reducing proliferation and increasing apoptosis *in vitro* in models of *H. pylori* infection.	Ding et al., 2008; Prabhu and Shivani, 2014
Chronic gastritis	*H. pylori* infection shown to increase epithelial cell turnover rate in gastric mucosa, with increased proliferation and apoptosis rates.	Wagner et al., 1997; Ahmed et al., 2000; Jang and Kim, 2000; Choi et al., 2003; Suzuki et al., 2004; Ernst et al., 2006; Ding et al., 2008
Gastric Mucosa-associated lymphoid tissue (MALT) tumors	Associated with *H. pylori* infections. Microbial virulence factors (*e.g.*, *CagA*, and *VacA*) activate inflammatory processes and cell proliferation.	Fox and Wang, 2007; Francescone et al., 2014; Wang et al., 2014
Multiple sclerosis	Increased *Methanobrevibacter* (phylum Euryarchaeota) and *Akkermansia* (phylum Verrucomicrobia) and decreased *Butyricimoas* (phylum Bacteroidetes).	Jangi et al., 2015
Depression	*Bifidobacterium infantis*, normally found in GI of neonatal infants and in administered probiotic drugs may have antidepressant effects in psychobiological systems.	Desbonnet et al., 2010; Dinan et al., 2013; Evrensel and Ceylan, 2015
Anxiety	Oral administration of subclinical doses of *Campylobacter jejuni* in murine models induced anxiety-like behavior without stimulating immunity. *Lactobacillus* and *Bifidiobacterium* may function as anxiolytic influence in a murine model.	Sudo, 2006; Lyte et al., 1998; Bravo et al., 2011; Messaoudi et al., 2011; Barrett et al., 2012; Evrensel and Ceylan, 2015; Akkasheh et al., 2016; Schnorr and Bachner, 2016
Non alcoholic fatty liver disease	Reduced levels of Bacteroidaceae, *Bacteroides* and *Oscillospira*	Chierico et al., 2016
Diarrheal illness	Enteric infection of the jejunum caused by *Cyclospora cayetanensis*, a foodborne and waterborne parasite. Results in diarrhea, including what is referred to as *traveler's diarrhea*.	Ortega and Sanchez, 2010
Giardiasis	Infection of the protozoan *Giardia lamblia* transmitted through the consumption of contaminated drinking water inducing abdominal cramps, gas, nausea, and weight loss. In a murine model, *Giardia* colonization and proliferation affected commensal bacteria with decreased *Firmicutes and Melainabacteria*, and increased *Proteobacteria*.	Maloney et al., 2015; Barash et al., 2017

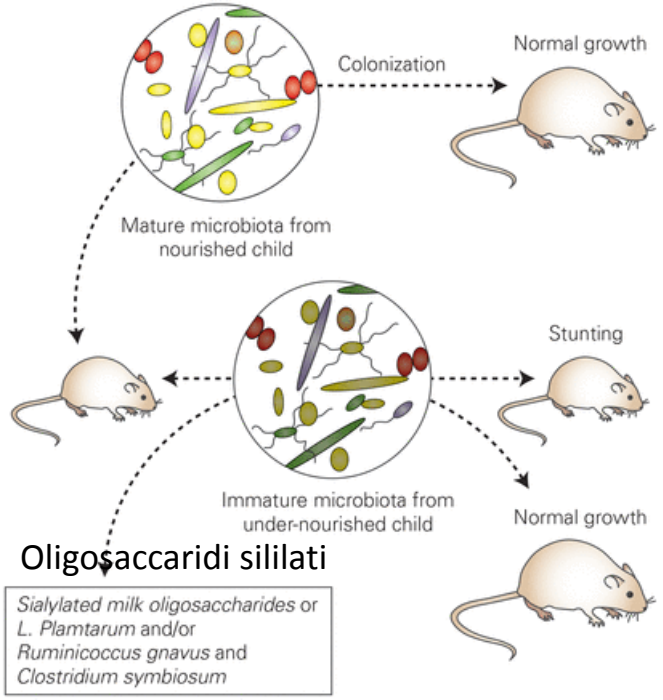
Susannah Selber-Hnatiw Human Gut Microbiota: Toward an Ecology of Disease Front Microbiol 2017.

Microbiota and obesity

Feeding the Microbiota: Transducer of Nutrient Signals for the Host Fergus Shanahan et al Gut. 2017



Microbiota in overnutrition. Schematic representation of the use of experimental 'humanised' mice (germ-free animals colonised with human microbiota from donors of various phenotypes, obese (A) and lean (B)), to demonstrate the potential impact of the microbiota on weight and metabolism of the host. Murine-to-murine transfers demonstrate the same effect and permit dietary manipulation. Human-to-human faecal microbial transplantation has also demonstrated the beneficial influence of a microbiota from a lean donor with improved insulin sensitivity in obese recipients (C). FMT, faecal microbial transplantation.



The undernourished microbiota. Colonisation of young mice with microbiota from healthy children leads to normal growth even if fed a nutrient deficient diet, whereas mice colonised by microbiota from a malnourished child exhibits stunting of growth. However, normal growth can be achieved by supplementation of the microbiota with specific microbial species at the time of initial colonisation or by dietary supplementation with sialylated milk oligosaccharides.

Microbiota and diet

European Review for Medical and Pharmacological Sciences 2016; 20: 4742-4749

The role of diet on gut microbiota composition

S. BIBBÒ[1,2], G. IANIRO[1], V. GIORGIO[3], F. SCALDAFERRI[1], L. MASUCCI[4], A. GASBARRINI[1], G. CAMMAROTA[1]

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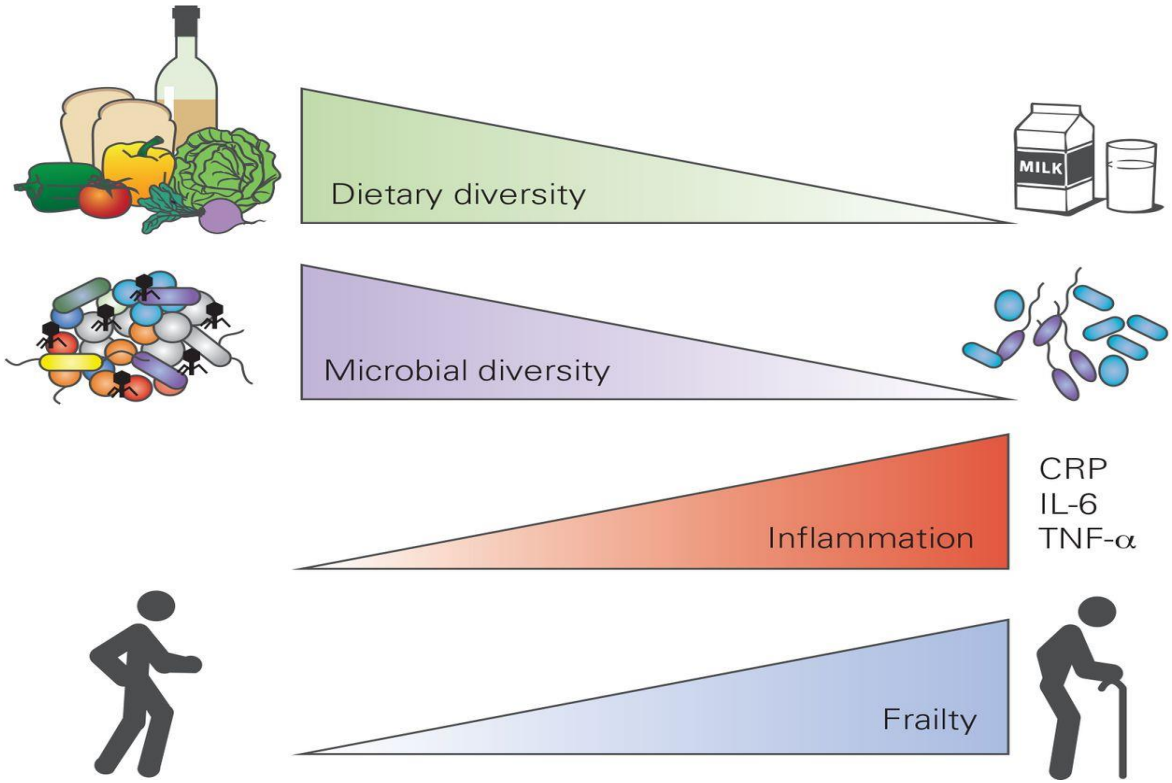
Microbiota and diet

Feeding the Microbiota: Transducer of Nutrient Signals for the Host Fergus Shanahan et al Gut. 2017



Diversity as staple, not simply spice of life. Diversity of dietary intake correlates with microbial diversity in the gut which is inversely linked with inflammatory tone and risk of frailty in the elderly. Thus, a monotonous or restricted diet (although with adequate calories and essential nutrients) which is often liquidised and convenient in the case of the elderly is linked with loss of microbial diversity, and perhaps more importantly, with loss of key microbial functions and a risk of a gain in pathobionts, including susceptibility to overgrowth of *Clostridium difficile*. CRP, C-reactive protein; IL-6, interleukin-6; TNF-α, tumour necrosis factor-α.

Obesity Epidemic

CHO restriction is one of the most frequent nutritional behaviors

Spread of high-protein diets aimed to weight-loss.

Widespread belief that western diets are excessively sugar-rich.

"Carbophobia" a term indicating the phylosophy of all low-carb diets (Atkins, Zone, Dukan, paleolitic)

Lose weight or lose fat

Not only “weight loss” but mainly decrease fat mass preservating free fat mass.

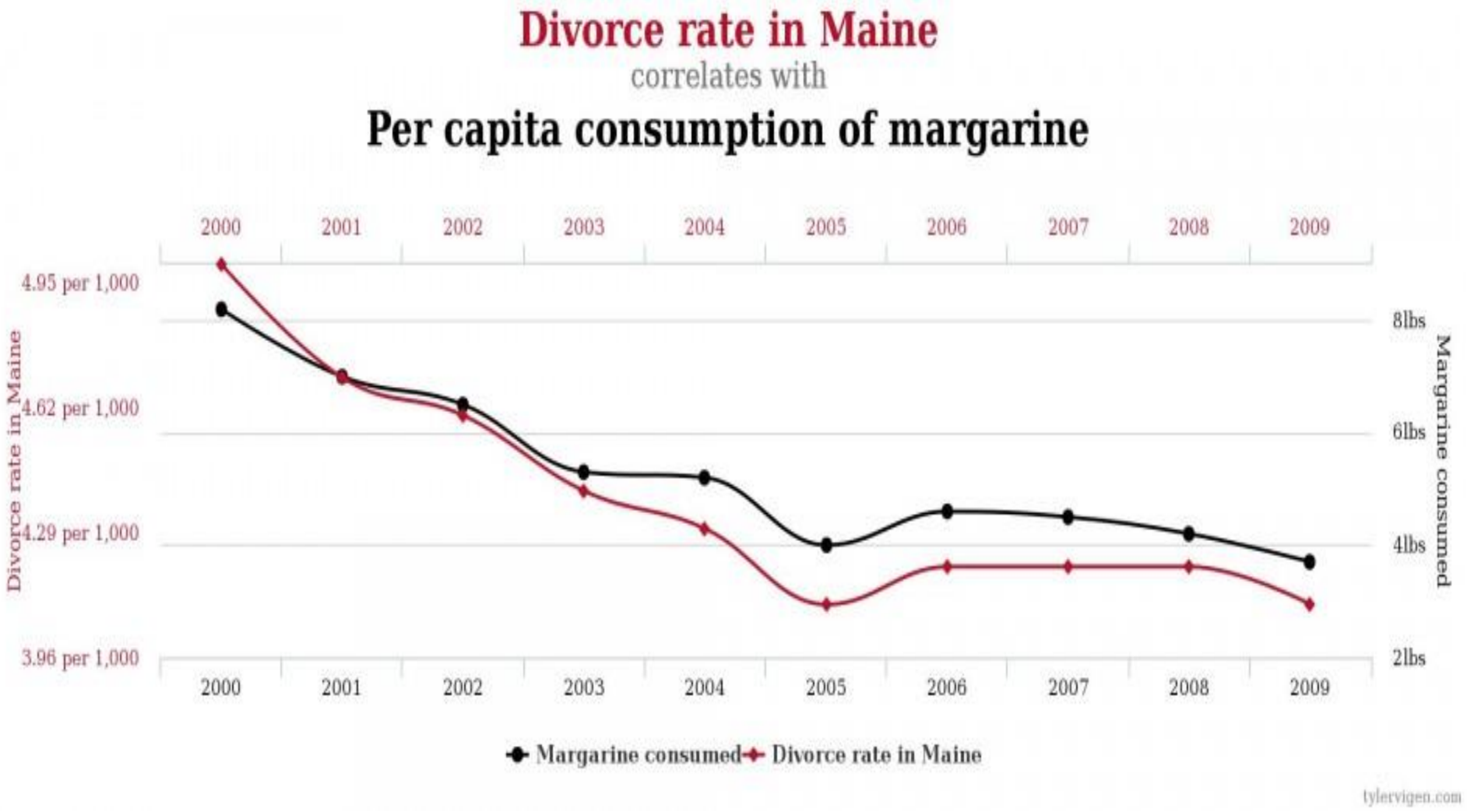
Brain feeds almost exclusively with glucose

Low carb diet= free fat mass loss to synthesize glucose from proteins (neoglucogenesis)

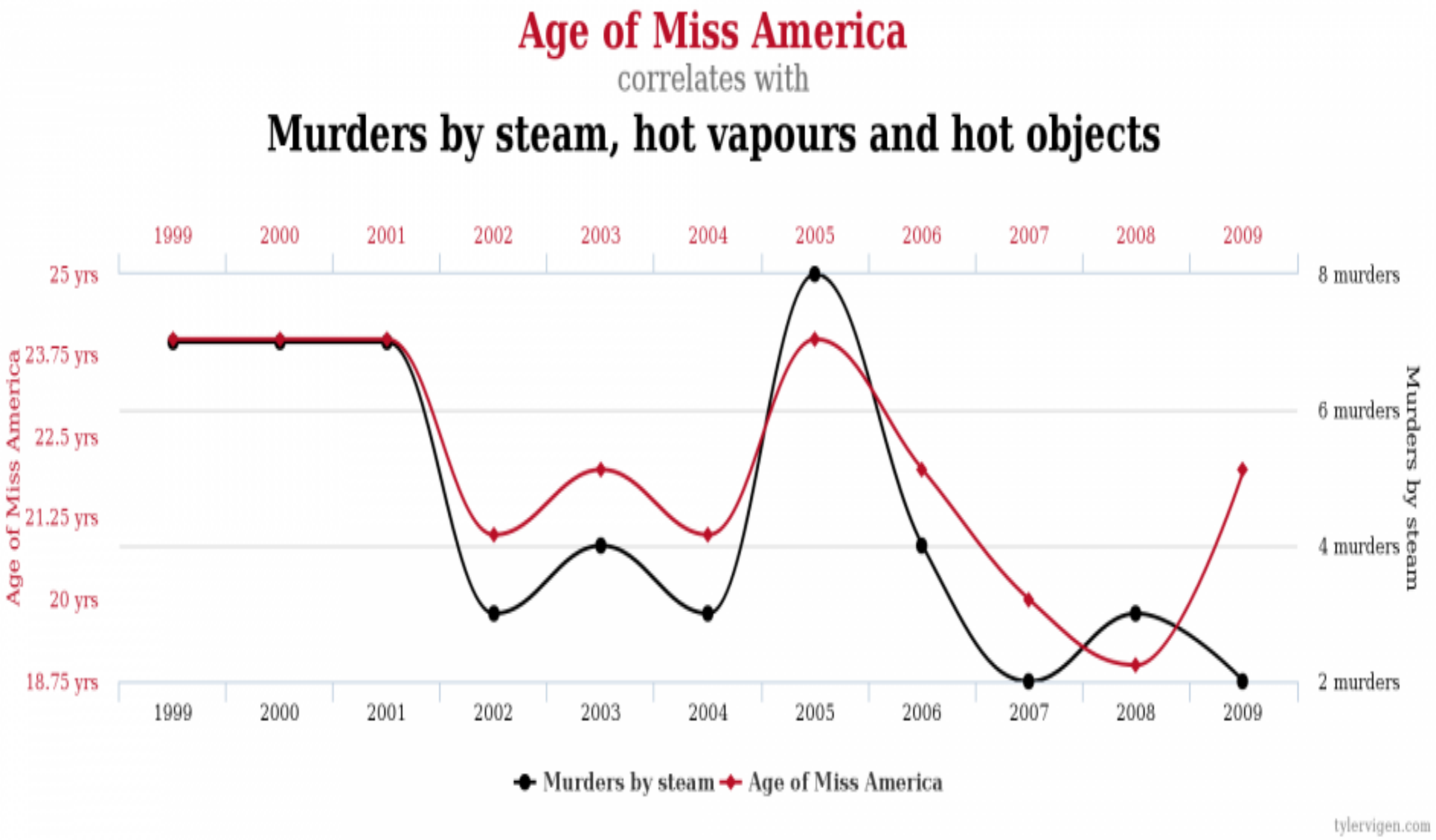
Role of each macronutrient is not easily relievable by another.

Risk of “normal weight” obesity

Information vs Disinformation



Vigen T. Spurious correlations 2015



Vigen T. Spurious correlations 2015

Sugar demonization

Solid evidences?

Guideline: Sugars intake for adults and children.
Geneva: World Health Organization; 2015.

Table 6. GRADE evidence profile for the effect of decreasing intake of free sugars to below 5% of total energy intake

Authors: Paula Moynihan and Sarah Kelly
Question: What is the effect of decreasing intake of free sugars to below 5% of total energy intake (<10 kg/person/year)?
Setting: General population

Quality assessment							No. of participants (sugars intake <10 kg/person/year)	Effect[a]	Quality
No. of studies	Design	Risk of bias	Inconsistency	Indirectness	Imprecision	Other considerations			
Dental caries (measured with standard indices)									
3	Ecological studies[b]	Serious risk of bias[c]	No serious inconsistency[d]	No serious indirectness[e]	No serious imprecision	None	18447[f]	Development of dental caries decreased in all 3 studies when intake of sugars fell from <15 kg/person/year to <10 kg/person/year[g] Correlation between intake of sugars and dental caries was in the range of r=0.6 – r=0.8	⊕⊖⊖⊖ VERY LOW[h]

r, correlation coefficient

[a] Meta-analysis was not possible due to the variability of the outcome measures.
[b] The three studies were national studies of dental caries levels compared with per capita sugar availability data.
[c] With ecological studies it is not possible to link exposures to outcomes at the individual level; hence, in this case, it is not possible to be sure that those individuals who are consuming less free sugars are the same individuals exhibiting a reduction in dental caries. Therefore, the evidence was downgraded to very low.
[d] There was no evidence of inconsistency. The results from all three studies found higher levels of dental caries with higher sugars intake (comparison of free sugars intake above 5% of total energy intake, to free sugars intake below 5% of total energy intake). As the data were not suitable for pooling, heterogeneity could not be formally assessed.
[e] All three studies assessed permanent dentition of children only; however, data were not downgraded for indirectness as the etiology of dental caries is the same in children and adults (although enamel of the primary dentition is softer and more vulnerable to demineralization by plaque acid), and the negative health effects of dental caries are cumulative with age. These studies were undertaken in populations with low fluoride exposure; however, there is no expectation of a difference in effect when extrapolating to populations with good fluoride exposure as the relationship between sugars intake and dental caries persists in the presence of exposure to fluoride.
[f] Total number of participants from the three population studies.
[g] All studies were conducted in Japan at a period when sugar availability dropped dramatically from 15 kg/person/year before the Second World War to a low of 0.2 kg/person/year (i.e. ≈0.1% of total energy) in 1946. In the study by Takeuchi *(54, 55)* and Takahashi *(56, 57)*, a log-linear relationship between sugars availability and dental caries increment was demonstrated between 0.2 kg/person/year and 5-7.5 kg/person/year in first permanent molars erupted for 7–8 years duration (r=0.8). In the study by Okuya *(58)*, when sugars availability decreased from 15 kg/person/year to <10 kg/person/year, dental caries also decreased, but not to zero. The correlation between sugars and dental caries in second permanent molars was r=0.7. In the study by Koike *(59, 60)*, when sugars availability decreased from 15 kg/person/year to <10 kg/person/year (and to a low of 0.2 kg/person/year), dental caries also decreased, but not to zero. Mean correlation between sugars availability and annual dental caries incidence rate was r=0.8 in the lower first permanent molars and r=0.6 for the upper first permanent molars. A straight line, log-linear relationship was shown between sugar availability and annual dental caries incidence between 0.1 kg/person/year and 15 kg/person/year.
[h] While the GRADE process is primarily used to assess evidence from controlled trials and cohort studies, the best available evidence in this case came from ecological studies. Under the guidance of a GRADE methodological expert, GRADE was used to assess the quality of evidence from the ecological studies, taking into consideration the limitations inherent in such studies (i.e. no adjustment for confounding and no information provided at the individual level), which precludes linking exposure data to outcomes along with any assessment of causality. A conservative approach was therefore taken and the evidence was rated as very low.

Appendix Table 4. Assessment of the Supporting Evidence for Each Recommendation (GRADE)

Guideline Title	Overall Recommendation	Specific Recommendations, Including Strength (if Reported)	Citations Supporting Recommendation, *n*	Study Design	GRADE Evidence Quality (Certainty in Estimates of Effect)
Sugars Intake for Adults and Children (WHO)*	–	"Reduced intake of free sugars throughout the life course—Strong Recommendation"	0	–	NA
		"In both adults and children, WHO recommends reducing the intake of free sugars to less than 10% of total energy intake—Strong Recommendation"	1	Systematic review	Low†
		"WHO suggests further reduction of the intake of free sugars to below 5% of total energy intake—Conditional Recommendation"	1	Systematic review	Very low
Carbohydrates and Health (Public Health England)‡	"The population average intake of free sugars should not exceed 5% of total dietary energy for age groups from 2 years upwards" and "The consumption of sugars-sweetened beverages should be minimised, in both children and adults."	"Greater sugar intake is associated with increased energy intake—Adequate Evidence" and "Sugar sweetened beverage intake is associated with risk of type-2 diabetes—Moderate Evidence"	1	Systematic review	Very low
		"Sugar consumption is associated with increased risk of dental caries—Moderate Evidence" and "Amount and frequency of SSB consumption is associated with dental caries—Adequate Evidence" and "Greater SSB consumption is associated with increased BMI—Limited Evidence"	1	Systematic review	Very low
Australian Dietary Guidelines	"Limit intake of foods and drinks containing added sugars such as confectionary, sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks"	"Consumption of sugar-sweetened beverages is associated with increased risk of weight gain in adults and children—Grade B"	15	Systematic review; randomized, controlled trial; observational study	Low, very low
		"High or frequent consumption of added sugars, particularly for infants and young children, is associated with increased risk of dental caries—Grade C"	1	Observational study	Very low
		"Consumption of soft drinks is associated with increased risk of dental caries in children—Grade C"	1	Observational study	Very low
		"Consumption of soft drinks is associated with increased risk of reduced bone strength—Grade C"	3	Randomized, controlled trial; observational study	Very low
Nordic Nutrition Recommendations	"Intake of added sugars should be kept below 10% of the energy intake"	–	14	Systematic review; observational study	Low, very low
Evidence-based Guideline of the German Nutrition Society: Carbohydrate Intake and the Prevention of Nutrition-Related Diseases	"The consumption of sugar-sweetened beverages should be limited, because they increase the risk of obesity and diabetes"	"The available cohort and intervention studies regarding adults mainly show that a higher consumption of SSB is accompanied by an increased risk of obesity—Probable"	6	Systematic review; randomized, controlled trial; observational study	Low, very low
		"The majority of prospective cohort studies and meta analysis indicate an increased risk of type 2 diabetes with regular consumption of sugar sweetened beverages—Probable"	5	Systematic review; observational study	Low, very low
Scientific Recommendations for Healthy Eating Guidelines in Ireland	"Healthy eating can be enjoyed with limited amounts of 'other foods' like biscuits, cakes, savoury snacks and confectionery. These foods are rich in calories, fat, sugar and salt so remember—NOT too MUCH and NOT too OFTEN"	–	6	Randomized, controlled trial; narrative review or report	Very low
Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids	A maximal intake level of 25% or less of energy is suggested to prevent the displacement of foods that are major sources of essential micronutrients.	–	7	Observational study	Very low
2015-2020 Dietary Guidelines for Americans§	"Consume less than 10% of calories per day from added sugars"				NA
Dietary Guidelines for the Brazilian Population	"Use oils, fats, salt, and sugar in small amounts for seasoning and cooking foods and to create culinary preparations"				NA

The Scientific Basis of Guideline Recommendations on Sugar Intake: A Systematic Review. Erickson J et al Ann Intern Med. 2017

BMI = body mass index; GRADE = Grading of Recommendations Assessment, Development and Evaluation; NA = not applicable; SSB = sugar-sweetened beverage; WHO = World Health Organization.

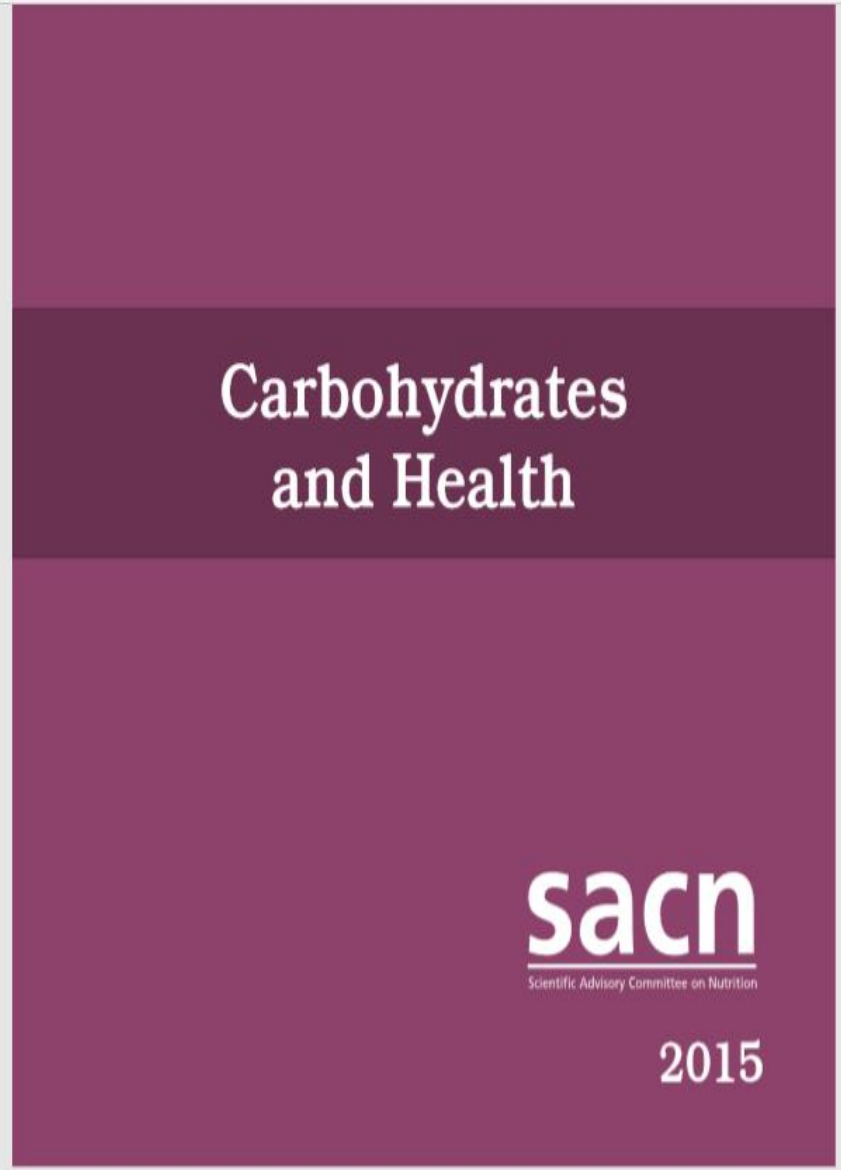
* A systematic review on sugars and weight was conducted and referenced. However, authors did not look specifically at 10% reduction; only the effect of sugar on dental caries was cited for the final 2 of 3 recommendations.

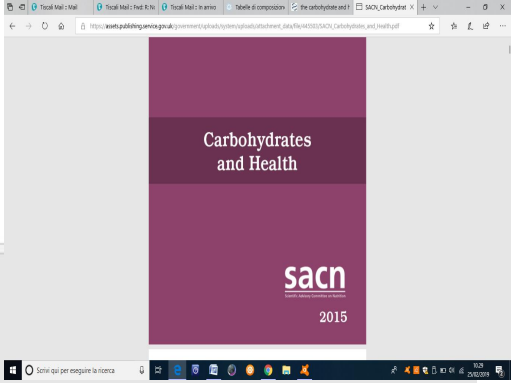
† The WHO rated the quality of evidence as "moderate"; however, in our independent assessment, we considered WHO's reasoning for rating up from low to be inappropriate.

‡ Public Health England conducted its own systematic reviews that were unpublished.

§ A rigorous scientific report of unpublished systematic reviews was conducted but was not used to make recommendation.

Scientific Advisory Committee on Nutrition Report





Sugars (g/day) and type 2 diabetes mellitus
• No association • Limited evidence

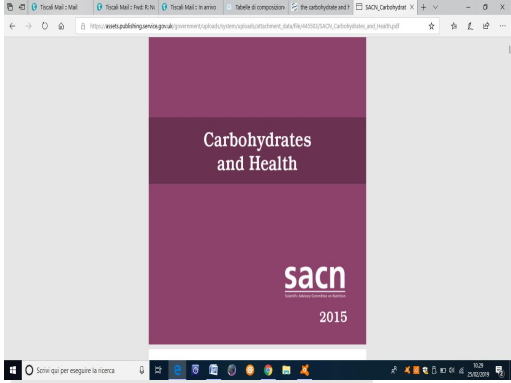
Blood glucose

6.25 Five randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to fasting blood glucose (Ryle *et al.*, 1990; Surwit *et al.*, 1997; Bantle *et al.*, 2000; Saris *et al.*, 2000; Black *et al.*, 2006). No further trials were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter).

6.26 Due to the diverse range of sugars interventions used in these trials, it is not appropriate to combine them in a meta-analysis. One compares a higher glucose diet to a higher fructose diet (Bantle *et al.*, 2000); one compares a higher glucose, lower soluble fibre diet, which includes a guar preparation, to a lower glucose, higher soluble fibre diet (Ryle *et al.*, 1990); one compares a higher sucrose group and a lower sucrose group (Black *et al.*, 2006) and one randomises subjects to three diets: lower fat, higher 'simple carbohydrates'; lower fat, higher 'complex carbohydrates'; and a control group (Saris *et al.*, 2000). Two of the trials are weight loss trials (Bantle *et al.*, 2000; Saris *et al.*, 2000). All the trials report no significant effect of diets differing in the proportion of sugars in relation to fasting blood glucose concentration. One of the trials measured day-long area under the curve

insulin (Bantle *et al.*, 2000), whereas the other trials measured both fasting insulin and area under the curve oral glucose tolerance test insulin.

Sugars and fasting blood glucose concentration
• No effect • Limited evidence



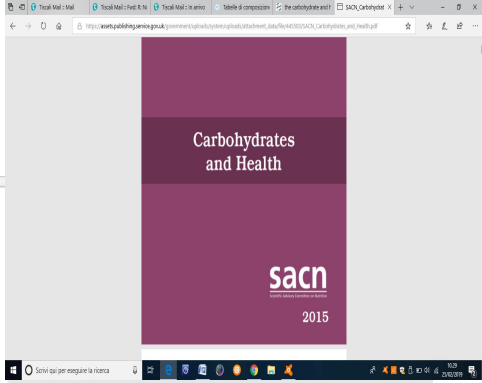
Sugars

Coronary events

6.6 Three cohort studies were identified that presented evidence on sugars intake and incidence of coronary events (Fehily *et al.*, 1993; Liu *et al.*, 2000; Beulens *et al.*, 2007). The exposure measures used in the studies were not sufficiently comparable to enable a meta-analysis to be performed. Two cohort studies were subsequently identified in the update search (Sieri *et al.*, 2010; Burger *et al.*, 2011) (Cardio-metabolic review, cardiovascular disease chapter; Update search).

6.7 No significant association is observed between sugars consumption and incidence of coronary events in any of the studies.

Sugars (g/day) and coronary events
• No association • Moderate evidence



6.7 No significant association is observed between sugars consumption and incidence of coronary events in any of the studies.

Sugars (g/day) and coronary events
• No association • Moderate evidence

Blood pressure

6.8 Five randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to blood pressure (Surwit *et al.*, 1997; Vasilaras *et al.*, 2001; Poppitt *et al.*, 2002; Raben *et al.*, 2002; Black *et al.*, 2006), three of which were included in a meta-analysis (Surwit *et al.*, 1997; Raben *et al.*, 2002; Black *et al.*, 2006). Two very small trials could not be included in a meta-analysis as they did not report the necessary data (Vasilaras *et al.*, 2001; Poppitt *et al.*, 2002). No further trials were identified in the update search (Cardio-metabolic review, incident hypertension and blood pressure chapter).

6.9 No significant effect is demonstrated for diets differing in the proportion of sugars on systolic blood pressure (1.4mmHg, 95% CI -5.4, 8.3; p=0.69) or diastolic blood pressure (3.1mmHg, 95% CI -0.2, 6.3; p=0.06); although the result is of borderline statistical significance. One trial included in the meta-analysis compared the consumption of sucrose-sweetened foods and drinks to non-calorically sweetened foods and beverages (Raben *et al.*, 2002). The other two trials compared higher and lower sucrose diets, one of which was a weight loss trial (Surwit *et al.*, 1997).

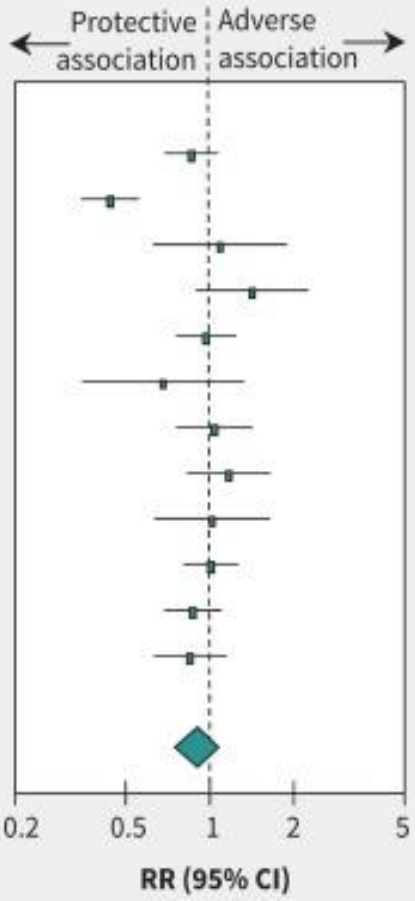
Sugars and systolic and diastolic blood pressure
• No effect • Limited evidence

Relation between intake of total sugars and incident type 2 diabetes
(highest v. lowest level of intake)

Study	No. of participants	No. of cases	Weight, %	RR (95% CI)
Janket et al., 2003[26]	38 480	918	10.20	0.86 (0.69–1.07)
Hodge et al., 2004[27]	31 641	365	9.90	0.44 (0.35–0.55)
Barclay et al., 2007[28]	1 833	138	5.70	1.09 (0.63–1.88)
Montonen et al., 2007[29]	4 284	175	6.80	1.42 (0.90–2.24)
Sluijs et al., Denmark, 2013[31]	4 037	2055	9.80	0.97 (0.76–1.23)
Sluijs et al., France, 2013[31]	867	288	4.60	0.68 (0.35–1.32)
Sluijs et al., Germany, 2013[31]	3 578	1584	8.80	1.04 (0.76–1.42)
Sluijs et al., Italy, 2013[31]	3 393	1437	8.40	1.17 (0.83–1.64)
Sluijs et al., Netherlands, 2013[31]	2 290	828	6.60	1.02 (0.64–1.63)
Sluijs et al., Spain, 2013[31]	5 889	2564	10.10	1.01 (0.81–1.25)
Sluijs et al., Sweden, 2013[31]	5 401	2622	10.00	0.87 (0.69–1.09)
Ahmadi-Abhari et al., 2014[32]	4 153	753	9.10	0.85 (0.63–1.14)
Total (95% CI)				**0.91 (0.76–1.09)**

$I^2 = 76$

Test for overall effect: $Z = 1.07$

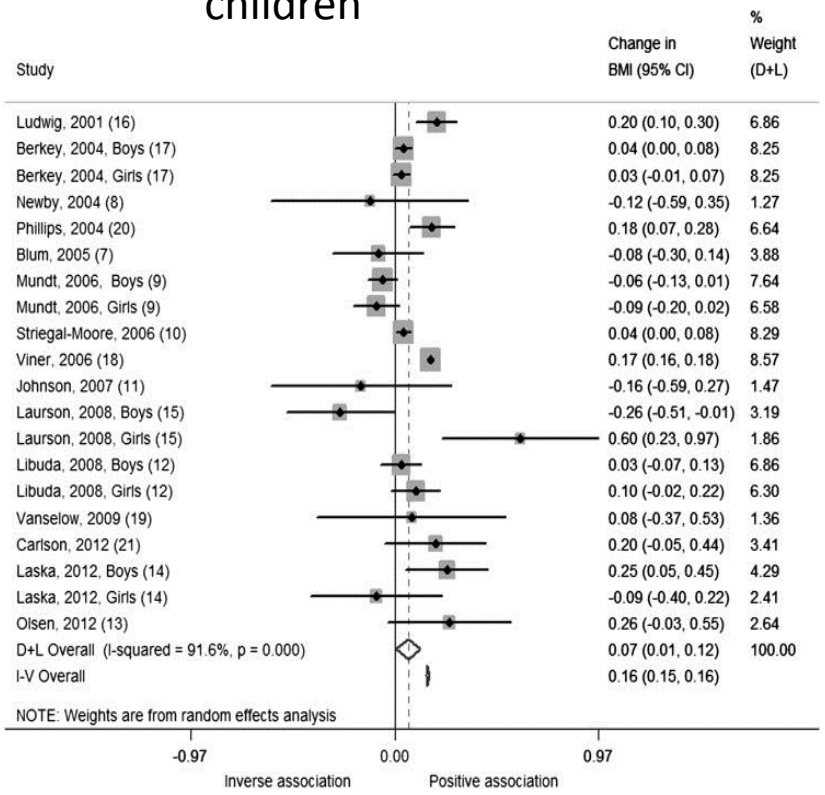


Tsilas et al. CMAJ. 2017;189:E711-E720

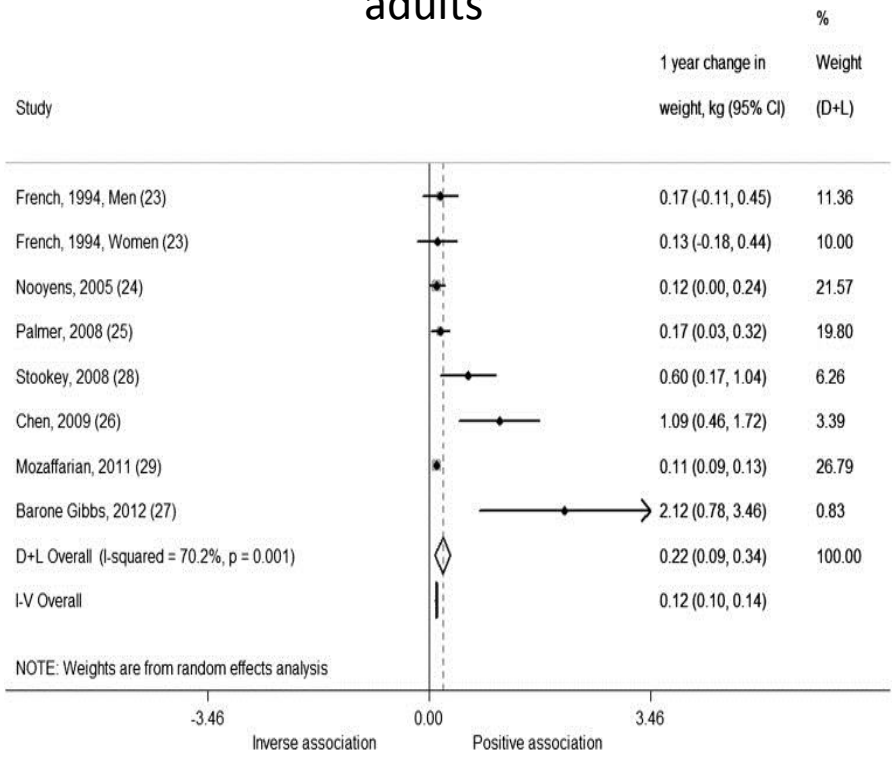
Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis.

Malik et al. Am J Clin Nutr. 2013;98:1084-1020

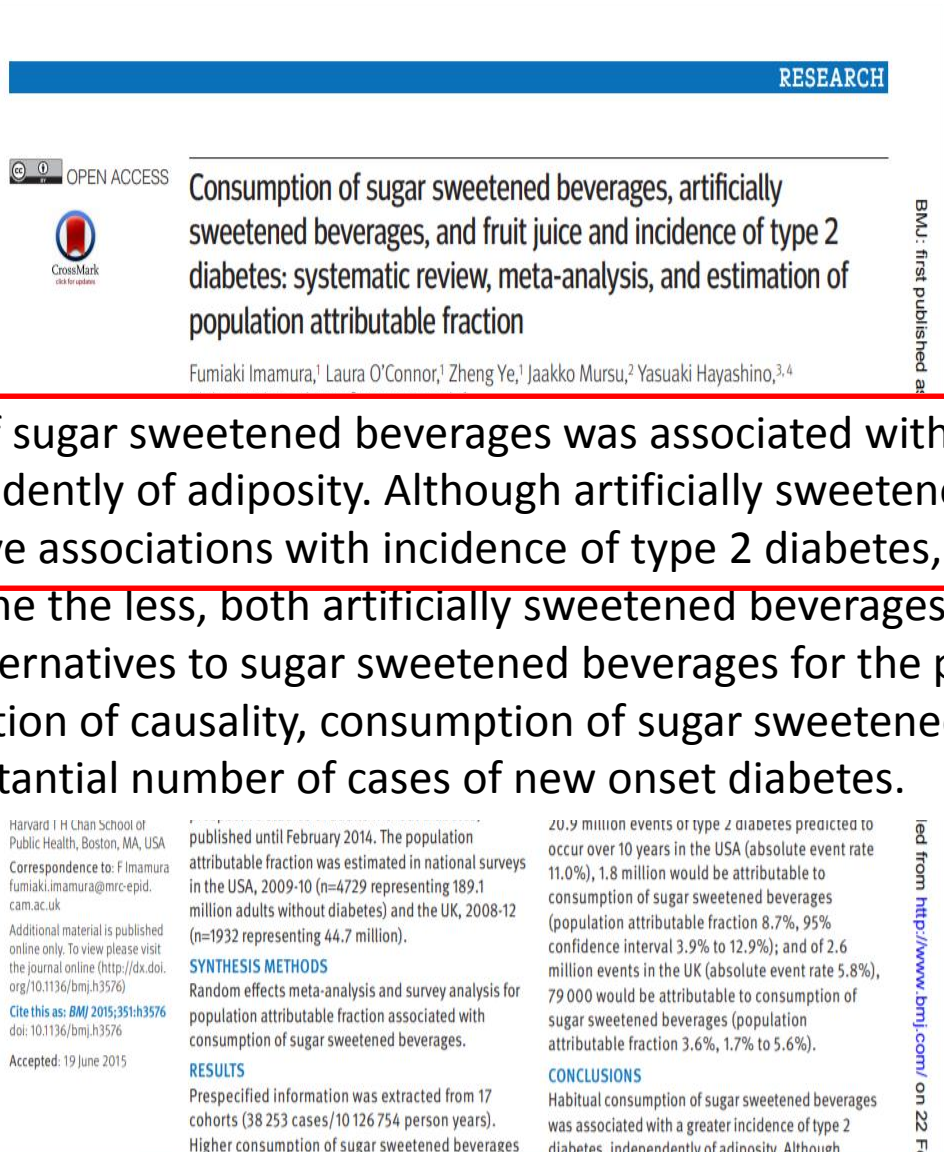
children



adults



One-year changes (95% CI) in weight (kg) per 1-serving/d increase in sugar-sweetened beverages from prospective cohort studies



RESEARCH

OPEN ACCESS

Consumption of sugar sweetened beverages, artificially sweetened beverages, and fruit juice and incidence of type 2 diabetes: systematic review, meta-analysis, and estimation of population attributable fraction

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Additional material is published online only. To view please visit the journal online (http://dx.doi.org/10.1136/bmj.h3576)

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Accepted: 19 June 2015

published until February 2014. The population attributable fraction was estimated in national surveys in the USA, 2009-10 (n=4729 representing 189.1 million adults without diabetes) and the UK, 2008-12 (n=1932 representing 44.7 million).

SYNTHESIS METHODS

Random effects meta-analysis and survey analysis for population attributable fraction associated with consumption of sugar sweetened beverages.

RESULTS

Prespecified information was extracted from 17 cohorts (38 253 cases/10 126 754 person years). Higher consumption of sugar sweetened beverages

20.9 million events of type 2 diabetes predicted to occur over 10 years in the USA (absolute event rate 11.0%), 1.8 million would be attributable to consumption of sugar sweetened beverages (population attributable fraction 8.7%, 95% confidence interval 3.9% to 12.9%); and of 2.6 million events in the UK (absolute event rate 5.8%), 79 000 would be attributable to consumption of sugar sweetened beverages (population attributable fraction 3.6%, 1.7% to 5.6%).

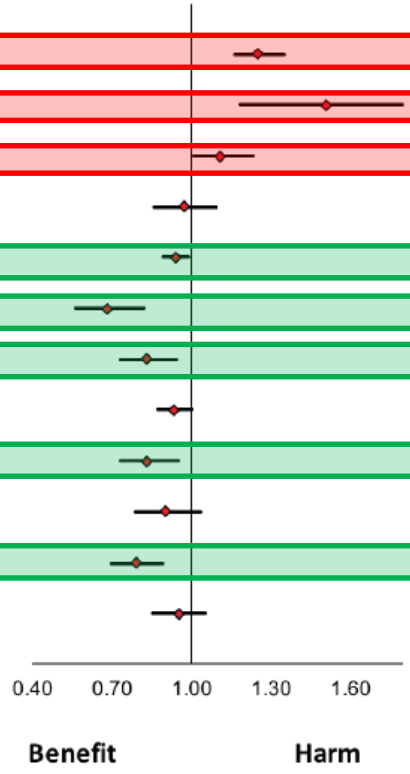
CONCLUSIONS

Habitual consumption of sugar sweetened beverages was associated with a greater incidence of type 2 diabetes, independently of adiposity. Although

Habitual consumption of sugar sweetened beverages was associated with a greater incidence of type 2 diabetes, independently of adiposity. Although artificially sweetened beverages and fruit juice also showed positive associations with incidence of type 2 diabetes, the findings were likely to involve bias. None the less, both artificially sweetened beverages and fruit juice were unlikely to be healthy alternatives to sugar sweetened beverages for the prevention of type 2 diabetes. Under assumption of causality, consumption of sugar sweetened beverages over years may be related to a substantial number of cases of new onset diabetes.

Relation of food sources of sugars with diabetes incidence: 84 cohort comparisons, n=3,899,203 (99,668 cases), FU=14y

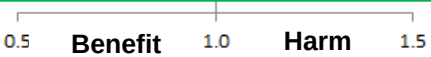
Food source	Cohort comparisons	Cases	Subjects	Risk ratio [95% CIs]	P (Association)	I^2	P (Heterogeneity)
Sugar-sweetened beverages	16	26,884	411,739	1.25 [1.16, 1.35]	<0.0001	55%	0.005
Fruit drinks	3	2,898	67,906	1.51 [1.18, 1.92]	0.001	50%	0.14
Mixed fruit drinks	7	18,959	364,321	1.11 [1.01, 1.23]	0.033	75%	0.001
100% fruit juice	5	13,739	206,899	0.97 [0.86, 1.09]	0.62	0%	0.59
Fruit	21	65,989	1,847,583	0.94 [0.89, 0.99]	0.04	41%	0.038
Whole grain breakfast cereal	4	3,779	237,694	0.68 [0.56, 0.82]	0.016	71%	0.016
Yoghurt	10	6,152	366,208	0.83 [0.73, 0.94]	0.008	58%	0.008
Jams, syrups, honey	4	993	27,847	0.93 [0.87, 1.0]	0.39	0%	0.39
Ice cream	2	1,499	150,477	0.83 [0.73, 0.95]	0.007	0%	0.37
Sherbert	2	1,548	172,015	0.90 [0.79, 1.03]	0.12	0%	0.6
Chocolate	5	1,630	26,737	0.79 [0.70, 0.89]	<0.0001	0%	0.83
Confectionary (biscuits, cakes, desserts)	3	837	23,531	0.95 [0.85, 1.05]	0.62	0%	0.95



Khan et al. Unpublished

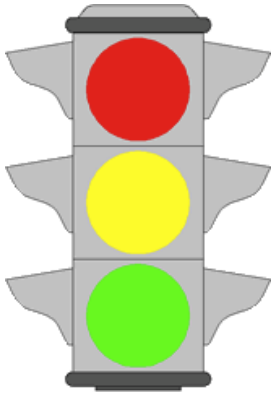
Relation of food sources of sugars with CV incidence : 134 cohort comparisons, n=5,311,852 (181,925 cases), FU=14y

Outcome and Food Source	Cohort Comparisons	Follow up (yrs)	Events	N	Relative Risk (95% CIs)	I^2	P-value I^2	GRADE Assessment
CVD incidence								
Sugar-Sweetened Beverage	6	10.0	4,856	94,784	1.04 [0.97, 1.13]	0%	0.58	●○○○ Very Low
Fruit	14	10.0	22,438	524,940	0.90 [0.86, 0.94]	0%	0.57	●●●○ Moderate
Fruit Drink	-				-	-	-	-
Fruit Juice	2	17.5	4,255	133,319	1.02 [0.93, 1.11]	0%	0.98	●○○○ Very Low
Breakfast Cereal	1	8.0	363	23,531	0.54 [0.28, 1.05]	n/a	-	●○○○ Very Low
Cookies and Cakes	3	12.0	2,883	49,946	0.94 [0.85, 1.04]	7%	0.34	●○○○ Very Low
Yogurt/Sweetened Dairy	3	24.0	7,605	100,575	0.84 [0.78, 0.91]	0%	0.62	●○○○ Very Low
Ice Cream/Sherbert	-				-	-	-	-
Chocolate	2	10.0	2,509	40,308	0.74 [0.56, 0.97]	59%	0.12	●○○○ Very Low
CHD incidence								
Sugar-Sweetened Beverage	6	18.0	9,889	199,942	1.17 [1.08, 1.26]	0%	0.73	●●●○ Moderate
Fruit	20	8.8	19,214	1,147,139	0.87 [0.83, 0.92]	0%	0.53	●●●○ Moderate
Fruit Drink	3	22.0	11,167	153,358	1.05 [0.98, 1.14]	86%	<0.01	●○○○ Very Low
Fruit Juice	5	11.0	10,137	227,927	1.01 [0.92, 1.10]	0%	0.79	●○○○ Very Low
Breakfast Cereal	3	8.6	1,933	153,770	0.80 [0.70, 0.92]	0%	0.84	●○○○ Very Low
Cookies and Cakes	2	12.5	2,924	111,285	1.15 [0.69, 1.91]	81%	0.02	●○○○ Very Low
Yogurt/Sweetened Dairy	8	14.8	6,973	144,731	0.96 [0.83, 1.10]	76%	<0.01	●○○○ Very Low
Ice Cream/Sherbert	2	16.2	451	1,759	1.12 [0.85, 1.46]	0%	0.34	●○○○ Very Low
Chocolate	6	10.7	12,843	283,819	0.88 [0.83, 0.94]	0%	0.46	●●○○ Low
Stroke incidence								
Sugar-Sweetened Beverage	10	14.0	11,026	267,669	1.05 [0.96, 1.16]	25%	0.22	●●○○ Low
Fruit	18	12.8	28,368	958,203	0.83 [0.78, 0.89]	28%	0.13	●●●○ Moderate
Fruit Drink	-				-	-	-	-
Fruit Juice	2	11.0	570	114,279	0.65 [0.51, 0.84]	0%	0.47	●○○○ Very Low
Breakfast Cereal	2	25.0	2,458	114,573	0.88 [0.80, 0.97]	0%	0.57	●○○○ Very Low
Cookies and Cakes	2	14.0	1,176	26,415	0.84 [0.70, 1.00]	0%	0.44	●○○○ Very Low
Yogurt/Sweetened Dairy	7	13.6	10,263	292,091	0.96 [0.87, 1.05]	57%	0.03	●○○○ Very Low
Ice Cream/Sherbert	2	13.8	2,627	112,320	0.90 [0.79, 1.03]	4%	0.31	●○○○ Very Low
Chocolate	5	11.9	6,337	157,219	0.85 [0.76, 0.94]	19%	0.29	●●●○ Moderate



Au Yeung et al. Unpublished

Misleading warnings: Food lights? Sugar tax? Nutriscore?



- Olive oil (99% fats)

(most important source of polyphenols, vitamin E, phytosterols etc.)

- Parmigiano, pecorino, grana (too much salt)

(main source of high quality proteins, CLA, satisfies by itslef daily needs of calcium and vitamin D)

- Light colas (no sugar)

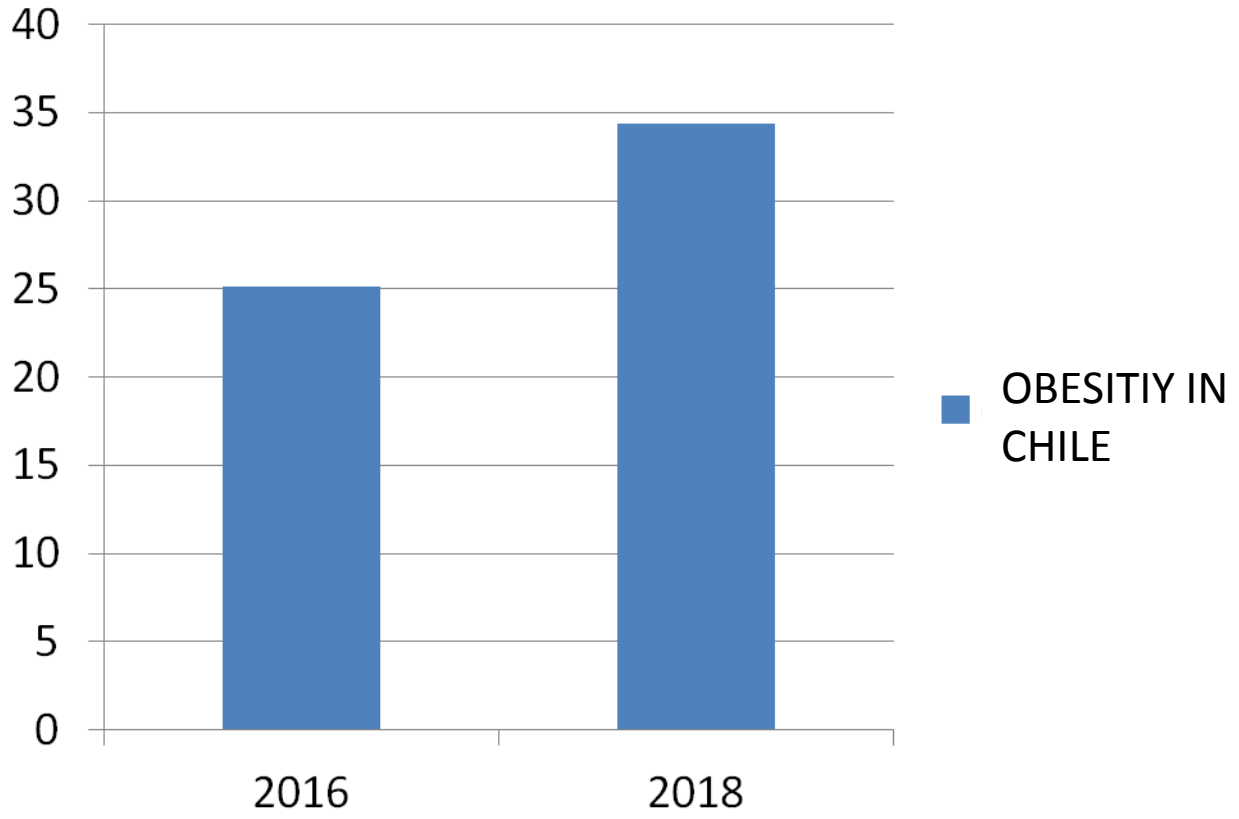
(edulcorants are involved in sweet taste dependance and do not improve diabetes risk)

Catena Jumbo Santiago - Chile

All chocolate characters loved by children (bunnies, Santa Claus, other figures) are hidden under white numbered bags. The contents of the product are declared on shelf tags according to the number on the product packaging.

Black labels in Chile

In 2016 in Chile begins the campaign of application of black labels on foods rich in salt, fats and sugars



FAO 2018

LARN 2014 (Italian Guidelines)

Based on epidemiological evidence of risk for the Italian population were established reference intervals (RI) (pointing to the middle of the range) for carbohydrates and lipids

- CHO 45-60% of calories (healthy adult)
  (<10% of calories for free sugars)

- Lipids 25-30% of calories (healthy adult)

For proteins it has been established a population recommended intake value (PRI) which corresponds to the level of intake sufficient to meet the nutrient requirements of nearly all (97.5%) healthy individuals in a specific population group.

- Proteins 0,90 gr/kg/die (healthy adult)

More than a diet… an educational model

New Pictorial Proposal for an Environmental, Sustainable Mediterranean Diet

p=portion Serving or portion size based on frugality and local habits

Regular physical activity
Adequate rest
Conviviality
Wine (and other alcoholic fermented beverages) in moderation and respecting social beliefs

Biodiversity and seasonality
Traditional, local and eco-friendly products
Culinary activities

IFMeD International Foundation of Mediterranean Diet
A healthy and sustainable Future

Sales of low-fat and low-sugar products in Italy

Oi 2017 • 2 – Free from

I NUMERI DEL MONDO FREE FROM: LA SEGMENTAZIONE DELLE CARATTERISTICHE

	% PRODOTTI	% VENDITE IN VALORE	TREND % VENDITE IN VALORE	PRESSIONE PROMO
Senza conservanti	7,7	12,1	-1,0	39,5
Pochi grassi	4,9	8,2	-0,1	35,8
Senza coloranti	3,8	5,1	-3,3	34,6
Pochi zuccheri	2,5	2,4	3,3	24,8
Senza olio di palma	2,3	6,4	17,6	35,4
Senza additivi	1,9	1,7	3,4	32,7
Senza OGM	1,7	1,6	-6,6	24,0
Senza grassi idrogenati	1,7	1,9	-3,9	40,5
Senza zuccheri aggiunti	1,3	0,8	6,1	26,2
Senza glutammato	1,0	1,0	1,1	35,6
A ridotto contenuto/ senza sale	0,8	0,7	7,2	30,8
Poche calorie	0,6	1,0	3,4	35,4
Senza/a ridotto contenuto di grassi saturi	0,5	2,1	7,6	35,8
Senza aspartame	0,1	0,1	-1,5	15,0

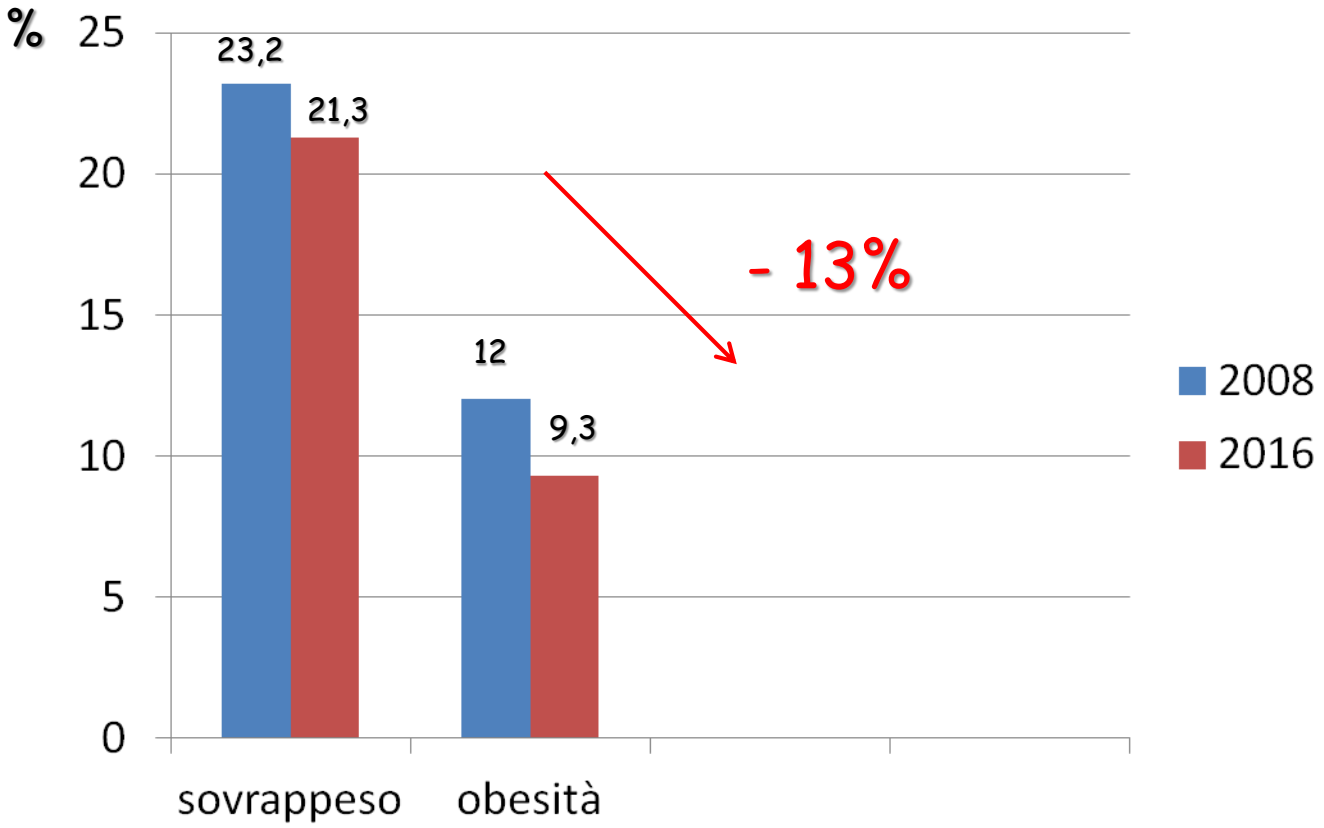
"Il mondo del "cibo senza" è diventato il più rappresentativo in termini di giro d'affari"

e creme spalmabili), dell'assenza di additivi (importante in particolare nei piatti pronti freschi, cresciuti molto nel corso dell'ultimo anno) e dell'assenza di olio di palma, che è molto sostenuto dall'offerta perché ha conquistato diverse categorie di prodotti.
La lettura effettuata dall'**Osservatorio**

Osservatorio Immagino Nielsen GS1 Italy - 2017 • 2

Overweight and obesity in childhood from 2008 to 2016 (Italy)

OKkio alla Salute:
National data 2016 (Istituto Superiore di Sanità)



Over 48.400 parents and 48.900 children involved all over National territory

The ITALIAN PROPOSAL: RI/GDA VS TRAFFIC LIGHT

There are some fundamental reasons why RI/GDA labels enjoy interest both from consumers and nutritionists, namely:

1. RI/GDA labelling is based on **per portion** information whilst traffic light labelling is applied on 100g. Combining the two approaches across all categories provides some confusing results. Products which typically are consumed in portions larger than 100g may get inferred traffic light endorsement when in reality their consumption levels merit careful consideration. For example, a 10g portion of a spread containing 0.2g of salt would have a red label for salt, whilst a ready meal containing 2.2g of salt would have an amber label.
2. RI/GDA labels is informative and encourage consumers to commit to positive incentives rather than pay heed to negative warnings, thus **contributing to the development of a nutrition education** among the population.
3. Some **foods** that are **recommended** to be included in healthy balanced diets – such as oily fish, nuts or cheese – can become **unfairly demonized** with a swathe of red warning signs.

- The scheme has been developed based on the **Article 35 criteria** of EU Regulation 1169 – Food Information to Consumers

Take home message

Sugar : yes or not? This is NOT the question.

The matter is

1) «how much?»
2) «which source?»
3) “sugar in the whole diet/lifestyle”