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FEATURE

ESSAY

What if sugar is worse than just empty calories? An essay by Gary Taubes

Doctors have long suspected sugar is not simply a source of excess calories but a fundamental cause of obesity and type 2 diabetes. **Gary Taubes** argues we must do more to discourage consumption while we improve our understanding of sugar's role

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Discussions about the global epidemics of obesity and type 2 diabetes tend to assume that these are new phenomena, products of changes in the way we eat and live that go back perhaps 50 years. For some populations with diets and lifestyles that are only recently westernised that may be true. In the United States, however, the origins of the diabetes epidemic may be found in hospital records from the 19th century.

In 1898, the physician Elliot Joslin and the Harvard pathologist Reginald Fitz published an analysis of 74 years of case records from Boston's Massachusetts General Hospital. They identified only 172 cases of diabetes among the 48 000 inpatients documented, but prevalence of the disease seemed to have been increasing almost exponentially since the mid-1850s. At the time, they attributed the increase to the "wholesome tendency of diabetics to place themselves under careful medical supervision."[1]

By 1921, though, Joslin was using the word "epidemic" to describe what he was witnessing. "Statistics for the last thirty years show so great an increase in the number [of patients with diabetes] that, unless this were in part explained by a better recognition of the disease, the outlook for the future would be startling," he wrote.[2]

Joslin's observations were corroborated three years later by the Columbia University researchers Haven Emerson and Louise Larimore. Mortality from diabetes in some North American cities, they reported, had increased almost 15-fold since the civil war years.

Sucrose as prime suspect

"Better recognition of the disease," as Joslin had put it, may have played a part in explaining these large increases in diabetes mortality, but Emerson and Larimore had another suspect as well. They were not alone in pointing this out: a likely cause of the epidemic, they suggested, was refined sugar—namely, sucrose.

The candy, chocolate, and ice cream industries had all been founded in the 1840s; the soft drink industry was launched most prominently by Coca Cola and Pepsi in the 1880s. Per capita sugar consumption increased 16-fold in the United States over the course of the century. "Rises and falls in sugar consumption," Emerson and Larimore wrote, "are followed with fair regularity . . . by similar rises and falls in the death rates from diabetes."[3]

The idea that sugar could be a fundamental cause of diabetes, not just a source of empty calories, fell out of fashion over the years, but given the present dire situation the idea should be considered seriously again.

During a keynote address at the 2016 annual meeting of the US National Academy of Medicine, the World Health Organization's then director general, Margaret Chan, described the twin epidemics of obesity and diabetes worldwide as a "slow-motion disaster"—explosions in the prevalence of these related disorders that frankly strain the imagination.[4] Official estimates are that one in 11 people in the United States has diabetes; one in 16 in the UK. In some aboriginal populations half of adults aged over 60 have diabetes.[5] Worldwide, according to WHO, the number of adults living with diabetes has quadrupled in less than 40 years.[4] The latest estimates from the American Diabetes Association and the Centers for Disease Control and Prevention suggest that obesity and diabetes may now cost the US healthcare system as much as $1bn (£740m; €850m) a day.[6 7]

And yet Chan also suggested in her presentation that the likelihood that public health organisations such as WHO would succeed at preventing the current "bad situation" from getting "much worse" was "virtually zero."

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In the midst of such a huge public health crisis, and with the acknowledgment that failure is inevitable, the obvious question to ask is why. Why, despite all our best efforts, have these epidemics gone unchecked? Many reasons can be imagined for any public health failure, but no recent precedents exist for a failure of this magnitude when we, in theory, understand the cause of the disease. A simple explanation, then, could be that our understanding is flawed—perhaps fundamentally so.

Misplaced confidence in understanding

On the simplest level, we think that diabetes is "largely a penalty of obesity," as Joslin wrote, back in 1921, and obesity is caused by overconsumption of calories—too much food, rather than the wrong kind. By this thinking, many societal and behavioural factors are causing overconsumption, obesity, and diabetes, but ultimately these complex disorders result from calorific overload, working in concert with relative physical inactivity. This thinking is so widely accepted that it is typically considered beyond question.

The past decade has seen a renewed interest in the possibility that calorific sweeteners— particularly sugar and high fructose syrups—have major roles in causing obesity and diabetes, but still only in the context of this century old thinking that general overconsumption of calories is the problem.

Major public health organisations, including WHO, the American Heart Association, and Public Health England,[8] are now recommending strict limits to the consumption of these calorific sweeteners–what PHE calls "free sugars"–but they do so with the rationale only that these sugars cause dental caries and are a source of excess calories, "empty" of vitamins, minerals, protein, and fibre. As such, they are easy targets for restriction in a world in which far too many people overconsume.

But what if, as Emerson and Larimore suggested, the problem is the sugar itself. If so, then the failure to curb the epidemics is because of the failure to curb sugar consumption to any significant extent. The hypothesis is that sugar has deleterious effects on the human body independent of its calorific content and that a distinct causal pathway links consumption to disease. It is based on the fact that the fructose component of sucrose (and high fructose syrup), unlike the accompanying glucose, is metabolised primarily in the liver. This can lead to accumulation of hepatic fat—described in 1991 by the Israeli biochemist Eleazar Shafrir, as "the remarkable hepatic lipogenic capacity induced by fructose-rich diets"[9]—and from there, by this hypothesis, to the insulin resistance that is the fundamental biochemical disturbance in type 2 diabetes.

If sugar is a dietary trigger of insulin resistance, then it causes type 2 diabetes, whether it is only one of many causes or the primary one. If this hypothetical link really exists then sugar, high fructose syrups, and sugary beverages should also be prime suspects for causing non-alcoholic fatty liver disease, which associates with obesity and type 2 diabetes and is also increasing at epidemic proportions.

The wrong question gave the wrong answer

One way to conceptualise how sugar escaped appropriate censure is that, beginning in the 1950s, nutritionists and public health authorities asked the wrong question, and so they got the wrong answer. They focused on solving the problem they could see: why did there seem to be an epidemic of heart disease in the US and some European nations? What aspect of diet was responsible?

By asking these questions, they missed the bigger picture. Heart disease is associated with both obesity and diabetes and also with a cluster of metabolic abnormalities that are now known as "metabolic syndrome."

Wherever and whenever populations made the transition from traditional, preindustrial diets to westernised, industrialised diets, they have experienced epidemics of obesity and diabetes. The question is why. The saturated fat content of diet was an answer to the heart disease question. But it's not necessarily the answer to questions about obesity and diabetes.

The public relations campaign of the sugar industry, and the general exoneration of sugar that followed, set research on any link between sugar, heart disease, and diabetes back by 20 years—but didn't end it. As early as 1980, Stanford University researchers looking for animal models of metabolic syndrome and insulin resistance reported that they could cause these conditions, at least in rodents, by feeding them diets rich in sugar, although they could not dissociate any effect intrinsic to sugar from that of its calories. Other researchers studied fructose metabolism, interested in the possibility that fructose could be used as a sweetener by people with diabetes because it can be metabolised without insulin.

This research, mostly in animals, supported John Yudkin's contention that consuming large doses of sugar could cause a cluster of metabolic abnormalities that associate with heart disease: insulin resistance and type 2 diabetes, hepatic lipogenesis, dyslipidaemia, ectopic fat accumulation, and perhaps visceral fat accumulation as well.[9]

Shafrir and other biochemists suggested the mechanism: the hepatic metabolism of fructose, in the context of raised blood sugar and pancreatic insulin secretion caused by the accompanying glucose. By 1993, the *American Journal of Clinical Nutrition* dedicated an entire issue to the possible deleterious effects of sugar consumption. "Further studies are clearly needed to determine the metabolic alteration that may take place during chronic fructose or sucrose feeding," the Swiss physiologists Luc Tappy and Eric Jéquier wrote.[10] This research still needs be done

Ambiguous evidence

Whether sugar does induce these metabolic abnormalities through a pathway distinct from the effect of its calories, whether it does so in humans and not just laboratory animals, and ultimately whether it is responsible for the observed epidemics of obesity and diabetes are still valid questions. But if the hypothesis that the fructose content of sugar and high fructose syrups makes them uniquely toxic is found to be correct then the dietary prescriptions of the past 40 years have been very wrong.

The evidence is indeed far from definitive, as the sugar industry and some researchers will argue. Existing research has been incapable of unambiguously disassociating any deleterious effect of sugar from that of the calories it contains.[11] The US National Institutes of Health spent at least $750m testing the hypothesis that dietary fat consumption raises serum cholesterol and causes chronic disease. But it did not see the need to invest similar sums to answer the simple question, would we live longer and be healthier if we were given dietary advice to avoid sugar? This requires funding randomised controlled studies looking at hard endpoints such as mortality and cardiovascular events, as

Why was sugar missed?

To propose sucrose as a major cause of the epidemics of obesity and diabetes requires an explanation for why mainstream medical and nutrition authorities have only recently come to consider the possibility seriously. This becomes clear with the benefit of hindsight. After Emerson and Larimore suggested sugar as the prime suspect in 1924, the hypothesis was rejected in the US by Joslin himself, whose textbook *The Treatment of Diabetes Mellitus* was becoming the bible in the discipline, and in the UK by Harold Himsworth, the future director of the Medical Research Council.

As a young physician in his 20s, Himsworth initially argued that sugar was healthy for people with type 2 diabetes because it "must be given" to treat diabetic coma, and that apparently shaped his thinking.[6] He then decided that dietary fat was the cause of diabetes, based largely on two observations: his patients with diabetes reported consuming relatively fatty diets, while people in Japan tended to consume very low fat diets and had very low rates of diabetes.[14]

Joslin was swayed by Himsworth's observations as well as the Japanese experience, assuming incorrectly that all carbohydrates are alike in how they are metabolised—whether from rice or sugar. So a population that ate a carbohydrate rich diet and had low rates of diabetes provided compelling reason to believe, so Joslin argued, that sugar was not a cause.[15] (Neither Joslin nor Himsworth apparently considered the fact that the Japanese diet also contained relatively little sugar.)

With such influential authorities arguing that sugar was benign, it took on the aura of undisputed truth. Just as other physicians and nutritionists in the 1960s, led by the British researchers Peter Cleave and John Yudkin,[16] began to suggest that sugar was indeed a likely cause of obesity, diabetes, and now heart disease as well, diabetes specialists would assume that the possibility was unworthy of their attention. This attitude was reinforced by the growing conviction among these authorities that heart disease could be adequately explained by the saturated fat content of diet, leaving no role for sugar. The two hypotheses were widely considered mutually exclusive.[17]

This in turn gave the sugar industry the ammunition to defend its product. Beginning in the mid-1960s, the industry engaged in a successful public health campaign based on hiring influential researchers, most prominently Fred Stare, the founding director of the nutrition department at the Harvard School of Public Health, to write articles and authoritative reports disseminating their belief that dietary fat was the primary evil in American diets and that sugar was indeed benign.[18]

By 1980, dietary guidelines from the US Department of Agriculture were advising consumers simply to "avoid too much sugar"—a statement that could be said about any food. By 1985 the guidelines stated dogmatically that "too much sugar in your diet does not cause diabetes." A subsequent series of authoritative US and UK government reports institutionalised the hypothesis that dietary fat and serum cholesterol were the agents of heart disease, and that we should worry about the fat content of our diets and not the carbohydrates—sugar or otherwise. Evolving science on insulin resistance and metabolic syndrome that implicated the sugar content of diet remained on the fringes of nutrition discussions.

it did with dietary fat, rather than short term studies examining risk factors alone.[11]

Evidence suggests that when people severely restrict carbohydrate intake–not just sugars, but also grains and starchy vegetables–diabetes can be reversed or disappears.[12] This is consistent with the hypothesis that sugar is a cause of diabetes. However, the studies are incapable of determining whether this beneficial effect is due to the carbohydrate restriction or the calorie restriction that might go with it.[12]

What is most likely?

Ultimately, science is not about proving what is true or what is not, as the Nobel laureate physicist Richard Feynman has said, but what is more or less likely.[13] The hypothesis that sugar consumption causes diabetes through a mechanism other than its contribution to calorific overload could be true. Whether it is likely or not is still a judgment call. If it is true, though, it changes how we must communicate the dangers of sugar consumption.

Setting an upper limit to the amount of sugar that should be consumed in a healthy diet is a good start, as Public Health England and other organisations now do. But we don't know if the level recommended is safe for everyone, and individuals do not know whether they may have crossed some threshold beyond which the damage caused by the sugar is irreversible. It could be that for people who have obesity or diabetes, or both, even a little is too much. And the ubiquity of sugar rich products may make it difficult for many people to maintain a healthy level of sugar consumption.

If sugar can trigger accumulation of liver fat, insulin resistance, and type 2 diabetes, then that's what has to be said. If the evidence is ambiguous, as it still is, and given the scale of the obesity and diabetes epidemics, then a concerted programme of research to establish reliable knowledge on this subject should be among our highest priorities. Meanwhile, we can acknowledge the uncertainties while still recommending strongly against consumption.

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1 Fitz RH, Joslin EP. Diabetes mellitus at the Massachusetts General Hospital from 1824 to 1898: a study of the medical records. *JAMA* 1898;31:165-71doi:10.1001/jama.1898.92450040015001a.
2 Joslin EP. The prevention of diabetes mellitus. *JAMA* 1921;76:79-84doi:10.1001/jama.1921.02630020001001.
3 Emerson H, Larimore LD. Diabetes mellitus—a contribution to its epidemiology based chiefly on mortality statistics. *Arch Intern Med* 1924;34:585-630doi:10.1001/archinte.1924.00120050002001.
4 Chan M. Obesity and diabetes: the slow-motion disaster. Keynote address at the 47th meeting of the National Academy of Medicine. 2016. http://www.who.int/dg/speeches/2016/obesity-diabetes-disaster/en/
5 Dyck R, Osgood N, Lin TH, Gao A, Stang MR. Epidemiology of diabetes mellitus among First Nations and non-First Nations adults. *CMAJ* 2010;182:249-56. doi:10.1503/cmaj.090846 pmid:20083562.
6 American Diabetes Association. Economic costs of diabetes in the US in 2012. *Diabetes Care* 2013;36:1033-46. doi:10.2337/dc12-2625 pmid:23468086.
7 Finkelstein EA, Trogdon JG, Cohen JW, Dietz W. Annual medical spending attributable to obesity: payer-and service-specific estimates. *Health Aff (Millwood)* 2009;28:w822-31.. doi:10.1377/hlthaff.28.5.w822 pmid:19635784.
8 Scientific Advisory Committee on Nutrition. Carbohydrates and health. 2015. https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/445503/SACN_Carbohydrates_and_Health.pdf
9 Shafrir E. Fructose/sucrose metabolism: its physiological and pathological implications. In: Kretchmer N, Hollenbeck CB, eds. *Sugars and sweeteners.* CRC Press, 1991: 63-98.
10 Tappy L, Jéquier E. Fructose and dietary thermogenesis. *Am J Clin Nutr* 1993;58(Suppl):766S-70S.pmid:8213608.
11 Khan TA, Sievenpiper JL. Metabolic improvement with fructose restriction: is it the fructose or the weight loss?*Obesity (Silver Spring)* 2016;24:549. doi:10.1002/oby.21431 pmid:26857209.
12 McKenzie AL, Hallberg SJ, Creighton BC, et al. A novel intervention including individualized nutritional recommendations reduces hemoglobin A1c level, medication use, and weight in type 2 diabetes. *JMIR Diabetes* 2017;2:e5doi:10.2196/diabetes.6981.
13 Feynman RP. *The character of physical law.* MIT Press, 1967.
14 Himsworth HP. Diet and the incidence of diabetes mellitus. *Clin Sci* 1935;(Sep):117-48.
15 White P, Joslin EP. The etiology and prevention of diabetes. In: Joslin EP, Root HF, White P, Marble A, eds. *The treatment of diabetes mellitus.* 10th ed. Lea & Febiger, 1959: 47-98.
16 Yudkin J. *Pure white and deadly.* Davis-Poynter, 1972.
17 Keys A. Sucrose in the diet and coronary heart disease. *Atherosclerosis* 1971;14:193-202. doi:10.1016/0021-9150(71)90049-9 pmid:4940760.
18 Kearns CE, Schmidt LA, Glantz SA. Sugar industry and coronary heart disease research: a historical analysis of internal industry documents. *JAMA Intern Med* 2016;176:1680-5. doi:10.1001/jamainternmed.2016.5394 pmid:27617709.

Key messages

- Physicians and public health authorities have long hypothesised that dietary sugar could cause obesity and type 2 diabetes
- Until recently, fat consumption and total energy balance have dominated the debate about obesity and health
- Recent recommendations on consumption target sugar only for its calories rather than as a potential causal agent of disease
- The evidence that sugar has harmful qualities independent of its calories is ambiguous
- While we develop better science, we should strengthen recommendations against consumption

Biography

Gary Taubes is the author of *The Case Against Sugar* (Portobello, 2017). He is cofounder of the not-for-profit Nutrition Science Initiative, a senior editor at CrossFit Health, and an award winning science and health journalist. He is the recipient of a Robert Wood Johnson Foundation investigator award in health policy research and the author of *Why We Get Fat and What to Do About It* (Knopf, 2011) and *Good Calories, Bad Calories: Fats, Carbs, and the Controversial Science of Diet and Health* (Knopf, 2007), published in the UK as *The Diet Delusion*