**Is Sugar Toxic?**

**By GARY TAUBES , New York Times Magazine**

On May 26, 2009, Robert Lustig gave a lecture called “Sugar: The Bitter Truth,” which was posted on YouTube the following July. Since then, it has been viewed well over 800,000 times, gaining new viewers at a rate of about 50,000 per month, fairly remarkable numbers for a 90-minute discussion of the nuances of fructose biochemistry and human physiology.

Lustig is a specialist on pediatric hormone disorders and the leading expert in childhood obesity at the University of California, San Francisco, School of Medicine, which is one of the best medical schools in the country. He published his first paper on childhood obesity a dozen years ago, and he has been treating patients and doing research on the disorder ever since.

The viral success of his lecture, though, has little to do with Lustig’s impressive credentials and far more with the persuasive case he makes that sugar is a “toxin” or a “poison,” terms he uses together 13 times through the course of the lecture, in addition to the five references to sugar as merely “evil.” And by “sugar,” Lustig means not only the white granulated stuff that we put in coffee and sprinkle on cereal — technically known as sucrose — but also high-fructose corn syrup, which has already become without Lustig’s help what he calls “the most demonized additive known to man.”

It doesn’t hurt Lustig’s cause that he is a compelling public speaker. His critics argue that what makes him compelling is his practice of taking suggestive evidence and insisting that it’s incontrovertible. Lustig certainly doesn’t dabble in shades of gray. Sugar is not just an empty calorie, he says; its effect on us is much more insidious. “It’s not about the calories,” he says. “It has nothing to do with the calories. It’s a poison by itself.”

If Lustig is right, then our excessive consumption of sugar is the primary reason that the numbers of obese and diabetic Americans have skyrocketed in the past 30 years. But his argument implies more than that. If Lustig is right, it would mean that sugar is also the likely dietary cause of several other chronic ailments widely considered to be diseases of Western lifestyles — heart disease, hypertension and many common cancers among them.

The number of viewers Lustig has attracted suggests that people are paying attention to his argument. When I set out to interview public health authorities and researchers for this article, they would often initiate the interview with some variation of the comment “surely you’ve spoken to Robert Lustig,” not because Lustig has done any of the key research on sugar himself, which he hasn’t, but because he’s willing to insist publicly and unambiguously, when most researchers are not, that sugar is a toxic substance that people abuse. In Lustig’s view, sugar should be thought of, like cigarettes and alcohol, as something that’s killing us.

This brings us to the salient question: Can sugar possibly be as bad as Lustig says it is? It’s one thing to suggest, as most nutritionists will, that a healthful diet includes more fruits and vegetables, and maybe less fat, red meat and salt, or less of everything. It’s entirely different to claim that one particularly cherished aspect of our diet might not just be an unhealthful indulgence but actually be toxic, that when you bake your children a birthday cake or give them lemonade on a hot summer day, you may be doing them more harm than good, despite all the love that goes with it. But Lustig, who has genuine expertise, has accumulated and synthesized a mass of evidence, which he finds compelling enough to convict sugar. His critics consider that evidence insufficient, but there’s no way to know who might be right, or what must be done to find out, without discussing it.

If I didn’t buy this argument myself, I wouldn’t be writing about it here. And I also have a disclaimer to acknowledge. I’ve spent much of the last decade doing journalistic research on diet and chronic disease — some of the more contrarian findings, [on dietary fat](http://www.nytimes.com/2002/07/07/magazine/what-if-it-s-all-been-a-big-fat-lie.html?scp=1&sq=gary%20taubes%20and%20fat&st=cse), appeared in this magazine —– and I have come to conclusions similar to Lustig’s.

The history of the debate over the health effects of sugar has gone on far longer than you might imagine. It is littered with erroneous statements and conclusions because even the supposed authorities had no true understanding of what they were talking about. They didn’t know, quite literally, what they meant by the word “sugar” and therefore what the implications were.

So let’s start by clarifying a few issues, beginning with Lustig’s use of the word “sugar” to mean both sucrose — beet and cane sugar, whether white or brown — *and* high-fructose corn syrup. This is a critical point, particularly because high-fructose corn syrup has indeed become “the flashpoint for everybody’s distrust of processed foods,” says Marion Nestle, a New York University nutritionist and the author of “Food Politics.”

This development is recent and borders on humorous. In the early 1980s, high-fructose corn syrup replaced sugar in sodas and other products in part because refined sugar then had the reputation as a generally noxious nutrient. (“Villain in Disguise?” asked a headline in this paper in 1977, before answering in the affirmative.) High-fructose corn syrup was portrayed by the food industry as a healthful alternative, and that’s how the public perceived it. It was also cheaper than sugar, which didn’t hurt its commercial prospects. Now the tide is rolling the other way, and refined sugar is making a commercial comeback as the supposedly healthful alternative to this noxious corn-syrup stuff. “Industry after industry is replacing their product with sucrose and advertising it as such — ‘No High-Fructose Corn Syrup,’ ” Nestle notes.

But marketing aside, the two sweeteners are effectively identical in their biological effects. “High-fructose corn syrup, sugar — no difference,” is how Lustig put it in a lecture that I attended in San Francisco last December. “The point is they’re each bad — equally bad, equally poisonous.”

Refined sugar (that is, sucrose) is made up of a molecule of the carbohydrate glucose, bonded to a molecule of the carbohydrate fructose — a 50-50 mixture of the two. The fructose, which is almost twice as sweet as glucose, is what distinguishes sugar from other carbohydrate-rich foods like bread or potatoes that break down upon digestion to glucose alone. The more fructose in a substance, the sweeter it will be. High-fructose corn syrup, as it is most commonly consumed, is 55 percent fructose, and the remaining 45 percent is nearly all glucose. It was first marketed in the late 1970s and was created to be indistinguishable from refined sugar when used in soft drinks. Because each of these sugars ends up as glucose and fructose in our guts, our bodies react the same way to both, and the physiological effects are identical. In a 2010 review of the relevant science, Luc Tappy, a researcher at the University of Lausanne in Switzerland who is considered by biochemists who study fructose to be the world’s foremost authority on the subject, said there was “not the single hint” that H.F.C.S. was more deleterious than other sources of sugar.

The question, then, isn’t whether high-fructose corn syrup is worse than sugar; it’s what do they do to us, and how do they do it? The conventional wisdom has long been that the worst that can be said about sugars of any kind is that they cause tooth decay and represent “empty calories” that we eat in excess because they taste so good.

By this logic, sugar-sweetened beverages (or H.F.C.S.-sweetened beverages, as the Sugar Association prefers they are called) are bad for us not because there’s anything particularly toxic about the sugar they contain but just because people consume too many of them.

Lustig’s argument, however, is not about the consumption of empty calories — and biochemists have made the same case previously, though not so publicly. It is that sugar has unique characteristics, specifically in the way the human body metabolizes the fructose in it, that may make it singularly harmful, at least if consumed in sufficient quantities. The phrase Lustig uses when he describes this concept is “isocaloric but not isometabolic.” This means we can eat 100 calories of glucose (from a potato or bread or other starch) or 100 calories of sugar (half glucose and half fructose), and they will be metabolized differently and have a different effect on the body. The calories are the same, but the metabolic consequences are quite different.

The fructose component of sugar and H.F.C.S. is metabolized primarily by the liver, while the glucose from sugar and starches is metabolized by every cell in the body. Consuming sugar (fructose and glucose) means more work for the liver than if you consumed the same number of calories of starch (glucose). And if you take that sugar in liquid form — soda or fruit juices — the fructose and glucose will hit the liver more quickly than if you consume them, say, in an apple (or several apples, to get what researchers would call the equivalent dose of sugar). The speed with which the liver has to do its work will also affect how it metabolizes the fructose and glucose.

In animals, or at least in laboratory rats and mice, it’s clear that if the fructose hits the liver in sufficient quantity and with sufficient speed, the liver will convert much of it to fat. This apparently induces a condition known as insulin resistance, which is now considered the fundamental problem in obesity, and the underlying defect in heart disease and in the type of diabetes, type 2, that is common to obese and overweight individuals. It might also be the underlying defect in many cancers.

If what happens in laboratory rodents also happens in humans, and if we are eating enough sugar to make it happen, then we are in trouble.

When Glinsmann and his F.D.A. co-authors decided no conclusive evidence demonstrated harm at the levels of sugar then being consumed, they estimated those levels at 40 pounds per person per year beyond what we might get naturally in fruits and vegetables — 40 pounds per person per year of “added sugars” as nutritionists now call them. This is 200 calories per day of sugar, which is less than the amount in a can and a half of Coca-Cola or two cups of apple juice. If that’s indeed all we consume, most nutritionists today would be delighted, including Lustig. But 40 pounds per year happened to be 35 pounds less than what Department of Agriculture analysts said we were consuming at the time — 75 pounds per person per year — and the U.S.D.A. estimates are typically considered to be the most reliable. By the early 2000s, according to the U.S.D.A., we had increased our consumption to more than 90 pounds per person per year.

That this increase happened to coincide with the current epidemics of obesity and diabetes is one reason that it’s tempting to blame sugars — sucrose and high-fructose corn syrup — for the problem. In 1980, roughly one in seven Americans was obese, and almost six million were diabetic, and the obesity rates, at least, hadn’t changed significantly in the 20 years previously. By the early 2000s, when sugar consumption peaked, one in every three Americans was obese, and 14 million were diabetic.

**This correlation** between sugar consumption and diabetes is what defense attorneys call circumstantial evidence. It’s more compelling than it otherwise might be, though, because the last time sugar consumption jumped markedly in this country, it was also associated with a diabetes epidemic.

In the early 20th century, many of the leading authorities on diabetes in North America and Europe (including Frederick Banting, who shared the 1923 Nobel Prize for the discovery of insulin) suspected that sugar causes diabetes based on the observation that the disease was rare in populations that didn’t consume refined sugar and widespread in those that did. In 1924, Haven Emerson, director of the institute of public health at Columbia University, reported that diabetes deaths in New York City had increased as much as 15-fold since the Civil War years, and that deaths increased as much as fourfold in some U.S. cities between 1900 and 1920 alone. This coincided, he noted, with an equally significant increase in sugar consumption — almost doubling from 1890 to the early 1920s — with the birth and subsequent growth of the candy and soft-drink industries.

carbohydrate, does not cause diabetes. But sugar and rice are not identical merely because they’re both carbohydrates. Joslin could not know at the time that the fructose content of sugar affects how we metabolize it.

At the time, many of the key observations cited to argue that dietary fat caused heart disease actually support the sugar theory as well. During the Korean War, pathologists doing autopsies on American soldiers killed in battle noticed that many had significant plaques in their arteries, even those who were still teenagers, while the Koreans killed in battle did not. The atherosclerotic plaques in the Americans were attributed to the fact that they ate high-fat diets and the Koreans ate low-fat. But the Americans were also eating high-sugar diets, while the Koreans, like the Japanese, were not.

In 1970, Keys published the results of a landmark study in nutrition known as the Seven Countries Study. Its results were perceived by the medical community and the wider public as compelling evidence that saturated-fat consumption is the best dietary predictor of heart disease. But sugar consumption in the seven countries studied was almost equally predictive. So it was possible that Yudkin was right, and Keys was wrong, or that they could both be right. The evidence has always been able to go either way.

Physicians and medical authorities came to accept the idea that a condition known as metabolic syndrome is a major, if not *the* major, risk factor for heart disease and diabetes. The Centers for Disease Control and Prevention now estimate that some 75 million Americans have metabolic syndrome. For those who have heart attacks, metabolic syndrome will very likely be the reason.

The first symptom doctors are told to look for in diagnosing metabolic syndrome is an expanding waistline. This means that if you’re overweight, there’s a good chance you have metabolic syndrome, and this is why you’re more likely to have a heart attack or become diabetic (or both) than someone who’s not. Although lean individuals, too, can have metabolic syndrome, and they are at greater risk of heart disease and diabetes than lean individuals without it.

Having metabolic syndrome is another way of saying that the cells in your body are actively ignoring the action of the hormone insulin — a condition known technically as being insulin-resistant. Because insulin resistance and metabolic syndrome still get remarkably little attention in the press (certainly compared with cholesterol), let me explain the basics.

You secrete insulin in response to the foods you eat — particularly the carbohydrates — to keep blood sugar in control after a meal. When your cells are resistant to insulin, your body (your pancreas, to be precise) responds to rising blood sugar by pumping out more and more insulin. Eventually the pancreas can no longer keep up with the demand or it gives in to what diabetologists call “pancreatic exhaustion.” Now your blood sugar will rise out of control, and you’ve got diabetes.

Not everyone with insulin resistance becomes diabetic; some continue to secrete enough insulin to overcome their cells’ resistance to the hormone. But having chronically elevated insulin levels has harmful effects of its own — heart disease, for one. A result is higher triglyceride levels and blood pressure, lower levels of HDL cholesterol (the “good cholesterol”), further worsening the insulin resistance — this is metabolic syndrome.

When physicians assess your risk of heart disease these days, they will take into consideration your LDL cholesterol (the bad kind), but also these symptoms of metabolic syndrome. The idea, according to Scott Grundy, a University of Texas Southwestern Medical Center nutritionist and the chairman of the panel that produced the last edition of the National Cholesterol Education Program guidelines, is that heart attacks 50 years ago might have been caused by high cholesterol — particularly high LDL cholesterol — but since then we’ve all gotten fatter and more diabetic, and now it’s metabolic syndrome that’s the more conspicuous problem.

This raises two obvious questions. The first is what sets off metabolic syndrome to begin with, which is another way of asking, What causes the initial insulin resistance? There are several hypotheses, but researchers who study the mechanisms of insulin resistance now think that a likely cause is the accumulation of fat in the liver. When studies have been done trying to answer this question in humans, says Varman Samuel, who studies insulin resistance at Yale School of Medicine, the correlation between liver fat and insulin resistance in patients, lean or obese, is “remarkably strong.” What it looks like, Samuel says, is that “when you deposit fat in the liver, that’s when you become insulin-resistant.”

That raises the other obvious question: What causes the liver to accumulate fat in humans? A common assumption is that simply getting fatter leads to a fatty liver, but this does not explain fatty liver in lean people. Some of it could be attributed to genetic predisposition. But harking back to Lustig, there’s also the very real possibility that it is caused by sugar.

**As it happens,** metabolic syndrome and insulin resistance are the reasons that many of the researchers today studying fructose became interested in the subject to begin with. If you want to cause insulin resistance in laboratory rats, says Gerald Reaven, the Stanford University diabetologist who did much of the pioneering work on the subject, feeding them diets that are mostly fructose is an easy way to do it. It’s a “very obvious, very dramatic” effect, Reaven says.

By the early 2000s, researchers studying fructose metabolism had established certain findings unambiguously and had well-established biochemical explanations for what was happening. Feed animals enough pure fructose or enough sugar, and their livers convert the fructose into fat — the saturated fatty acid, palmitate, to be precise, that supposedly gives us heart disease when we eat it, by raising LDL cholesterol. The fat accumulates in the liver, and insulin resistance and metabolic syndrome follow.

Similar effects can be shown in humans, although the researchers doing this work typically did the studies with only fructose — as Luc Tappy did in Switzerland or Peter Havel and Kimber Stanhope did at the University of California, Davis — and pure fructose is not the same thing as sugar or high-fructose corn syrup. When Tappy fed his human subjects the equivalent of the fructose in 8 to 10 cans of Coke or Pepsi a day — a “pretty high dose,” he says —– their livers would start to become insulin-resistant, and their triglycerides would go up in just a few days. With lower doses, Tappy says, just as in the animal research, the same effects would appear, but it would take longer, a month or more.

In simpler language, how much of this stuff do we have to eat or drink, and for how long, before it does to us what it does to laboratory rats? And is that amount more than we’re already consuming? Unfortunately, we’re unlikely to learn anything conclusive in the near future. As Lustig points out, sugar and high-fructose corn syrup are certainly not “acute toxins” of the kind the F.D.A. typically regulates and the effects of which can be studied over the course of days or months. The question is whether they’re “chronic toxins,” which means “not toxic after one meal, but after 1,000 meals.” This means that what Tappy calls “intervention studies” have to go on for significantly longer than 1,000 meals to be meaningful.

Only one study in this country, by Havel and Stanhope at the University of California, Davis, is directly addressing the question of how much sugar is required to trigger the symptoms of insulin resistance and metabolic syndrome. Havel and Stanhope are having healthy people drink three sugar- or H.F.C.S.-sweetened beverages a day and then seeing what happens. The catch is that their study subjects go through this three-beverage-a-day routine for only two weeks. That doesn’t seem like a very long time — only 42 meals, not 1,000 — but Havel and Stanhope have been studying fructose since the mid-1990s, and they seem confident that two weeks is sufficient to see if these sugars cause at least some of the symptoms of metabolic syndrome.

So the answer to the question of whether sugar is as bad as Lustig claims is that it certainly could be. It very well may be true that sugar and high-fructose corn syrup, because of the unique way in which we metabolize fructose and at the levels we now consume it, cause fat to accumulate in our livers followed by insulin resistance and metabolic syndrome, and so trigger the process that leads to heart disease, diabetes and obesity. They could indeed be toxic, but they take years to do their damage. It doesn’t happen overnight. Until long-term studies are done, we won’t know for sure.

The second observation was that malignant cancer, like diabetes, was a relatively rare disease in populations that didn’t eat Western diets, and in some of these populations it appeared to be virtually nonexistent. In the 1950s, malignant cancer among the Inuit, for instance, was still deemed sufficiently rare that physicians working in northern Canada would publish case reports in medical journals when they did diagnose a case.

In 1984, Canadian physicians published an analysis of 30 years of cancer incidence among Inuit in the western and central Arctic. While there had been a “striking increase in the incidence of cancers of modern societies” including lung and cervical cancer, they reported, there were still “conspicuous deficits” in breast-cancer rates. They could not find a single case in an Inuit patient before 1966; they could find only two cases between 1967 and 1980. Since then, as their diet became more like ours, breast cancer incidence has steadily increased among the Inuit, although it’s still significantly lower than it is in other North American ethnic groups. Diabetes rates in the Inuit have also gone from vanishingly low in the mid-20th century to high today.

Now most researchers will agree that the link between Western diet or lifestyle and cancer manifests itself through this association with obesity, diabetes and metabolic syndrome — i.e., insulin resistance. This was the conclusion, for instance, of a 2007 report published by the World Cancer Research Fund and the American Institute for Cancer Research — “Food, Nutrition, Physical Activity and the Prevention of Cancer.”

So how does it work? Cancer researchers now consider that the problem with insulin resistance is that it leads us to secrete more insulin, and insulin (as well as a related hormone known as insulin-like growth factor) actually promotes tumor growth.

But some researchers will make the case, as Cantley and Thompson do, that if something other than just being fatter is causing insulin resistance to begin with, that’s quite likely the dietary cause of many cancers. If it’s sugar that causes insulin resistance, they say, then the conclusion is hard to avoid that sugar causes cancer — some cancers, at least — radical as this may seem and despite the fact that this suggestion has rarely if ever been voiced before publicly. For just this reason, neither of these men will eat sugar or high-fructose corn syrup, if they can avoid it.

“I have eliminated refined sugar from my diet and eat as little as I possibly can,” Thompson told me, “because I believe ultimately it’s something I can do to decrease my risk of cancer.” Cantley put it this way: “Sugar scares me.”

*Gary Taubes (*[*gataubes@gmail.com*](mailto:gataubes@gmail.com)*), Robert Wood Johnson Foundation  
Text abridged by L. Marti*