

Robert Lustig is a pediatric endocrinologist at the University of California, San Francisco. He is the author of *Fat Chance: Beating the Odds against Sugar, Processed Food, Obesity, and Disease*.<sup>1</sup> He specializes in childhood obesity and studying the effects of sugar in the diet. He is the director of the UCSF Weight Assessment for Teen and Child Health Program and a member of the Obesity Task Force of the Endocrine Society.

Below are some quotes from Lustig. There is a recording of him saying this – otherwise it would not be possible to believe that a nutrition expert could actually make such ignorant statements.

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**Sugar - because of its unique composition is the only food on the planet that is both fat and carbohydrate at the same time.**

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**Is there one reaction in your body that actually requires sugar? Zero.**

**Even fatty fruits - coconut, olives, avocado - have no carbohydrates.**

**There is no foodstuff on this planet that have both fat and carbohydrate at the same time. It is one or the other because that is evolution - that is nature - that is what God did.**

Sugars are carbohydrates – they are not fats. This is basic chemistry. Fatty acids contain a carboxyl group (COOH) – sugars do not.

It is simply wrong to state that coconut, olives, avocados have no carbohydrates.

Below is a table showing the macronutrient composition of 100 g of food.<sup>2</sup>

Nutrient	Olives	Coconut dried	Avocado
NDB No	09194	12108	09037
Water (g)	84	3	73
Protein (g)	1	7	2
Fat (g)	7	65	15
Carbohydrate (g)	6	24	9
Fiber (g)	2	16	7

In *Fat Chance*, Lustig states that the browning of bananas is caused by the Maillard reaction

due to its fructose content. Browning of freshly cut fruit and vegetables is a reaction caused by enzymes. Maillard reaction is a non-enzymatic reaction between reducing sugars (glucose, fructose, galactose, maltose, lactose) and amino acids that occur from around 140°C to 165°C. At higher temperatures, caramelisation occurs. The golden crust of bread is due to Maillard reactions.

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Lustig featured in the BBC's documentary, *The Men Who Made Us Fat*, written by Jacques Peretti. At the beginning of the Peretti informs, "I am going to trace those responsible for a revolution in our eating habits. I'll be looking at how decisions made behind closed doors transformed food into an addiction." Brief images of Ancel Keys and George McGovern are shown as two of the perpetrators of this exploit.

Below are some comments by Lustig from the documentary.

This man, Ancel Keys, claimed he had the answer to heart disease. His theory had a decisive impact on what we would all eat. But it also had a devastating side effect—creating the conditions for obesity. Keys's theory was that fat alone caused heart disease. [...]

In 1952, Keys did a sabbatical in England where he saw the epidemic of heart disease himself and correlated it with the enormously poor British diet of fish and chips, etc.—you know what I'm talking about—and decided that saturated fat had to be the culprit. And he actually said that back in the fifties before he did any studies. And he spent the next fifty years attempting to prove himself right.

Keys won the battle. Yudkin was thrown under the bus. And—well, he was discredited by numerous societies basically saying that he did not have the data to make his claims about the importance of sugar.<sup>3</sup>

Much of what a rather chubby Robert Lustig states is false.

Firstly, Keys's research was not the starting point for nutritional and cholesterol research, which had its foundations in the early years of the twentieth century.

Keys's early views on diet were formed in Italy and Spain, not in England. He developed his ideas about diet and heart disease when he was invited to Naples in the early 1950s. His studies showed dramatically lower rates of coronary heart disease in Italy and Spain. He introduced the concept of the Mediterranean diet to America—a diet he described as mainly vegetarian.

Initially, Keys did focus on fats in the diet—not saturated fats—as Lustig states above. Keys conducted many trials and experiments, both before and after he came to his initial conclusions regarding fat.

A number of other researchers, including Jeremiah Stamler, Gerry Shaper, Michael Oliver, and Geoffrey Rose, were of the opinion that “there was no firm evidence linking intake of dietary sugar and CHD.”<sup>4</sup>

The claim that “Keys's theory was that fat alone caused heart disease” is false and deceptive. Keys noted in 1980, “Responsible students of the coronary problem long ago abandoned the idea of seeking the cause of the disease, agreeing that several, perhaps many, variables are involved in almost all cases.”<sup>5</sup> As the title of this report (*Seven Countries: A Multivariate Analysis of Death and Coronary Heart Disease*) indicates, Keys and his colleagues were examining a number of different variables in relation to heart disease.

Lustig states,

Keys was already pretty famous in America because he was the originator, inventor, of the K-Ration. The K-ration was a way of getting 12,000 calories in a very small, compact little box.

Lustig had overestimated the amount of energy in the K-Ration by three to four times. The K-Ration was an emergency survival ration consisting of non-perishable food designed for a few days' use only. The program claims that the K-Ration contained a lot of sweet food like chocolate, “never for one moment [realising that] it could be harmful.”

As well as chocolate bars, it contained pemmican biscuits, veal meat, sausage, toilet paper, chewing gum, and cigarettes. The K-Ration was never designed for long-term use.

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Lustig's claim that Keys made his assertion regarding the implications of fats in the diet with

heart disease without the backing of research is not true.

In 1922, de Langen, working with Javanese men in the East Indies, showed that a diet high in eggs, butter, and meat raised serum cholesterol.

In 1947, Keys commenced the Minnesota Business and Professional Men Study to determine why apparently healthy middle-age men were dying from heart attacks. A number of variables were examined, with serum cholesterol being the most significant variable.

Keys performed studies with his wife, Margaret, in Naples, Rome and Madrid in 1952.

In 1955, Brian Bronte-Stewart, John Block (professor of medicine at University of Cape Town), Ancel and Margaret Keys and colleagues published a paper examining serum cholesterol, diet, income and cardiovascular mortality in Europeans, "Coloured" and Bantu groups in Cape Town.<sup>6</sup>

The mean values for total cholesterol were 234 mg/dL (6.1 mmol/L), 204 mg/dL (5.3 mmol/L) and 166 mg/dL (4.3 mmol/L) respectively. Heart disease for Europeans was more than twice that of Cape Coloured and among Bantu it was "exceedingly rare as a cause of death". This was the first study to show that the increase in LDL cholesterol was related to the consumption of animal fats.

In 1958, Keys and his colleagues published a paper examining serum cholesterol, diet and cardiovascular disease in Japanese living in Japan, Hawaii and Los Angeles. In Japan, "heart disease is rare, in Hawaii, where it is fairly common but less so than among local Caucasians, and in California, where the local Japanese are similar to the local Caucasians in regard to the frequency of the disease. In middle age, coronary heart disease is at least 10 times as common in the United States as in Japan." <sup>7</sup>

A number of researchers (J Groen, LW Kinsell, EH Ahrens, A Keys, JM Beveridge and B Bronte-Stewart) studied the relationship of saturated fat to serum cholesterol during the 1950s using controlled feeding studies.

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Lustig's admiration for Yudkin is unfounded. Yudkin was unable to produce the data to support his contention that sugar causes heart disease.

During the 1960s, John Yudkin noted that the consumption of sugar increased in Britain more than any other food item in the last 100 years. Both sugar consumption and total fat

consumption (note the correlation is with total fat and not saturated fat) correlated with heart disease but Yudkin concluded that sugar was the more likely cause.<sup>8</sup>

Yudkin performed a small study recording the intakes of sugar 70 men: 20 with a recent first heart attack, 25 with peripheral arterial disease and 25 healthy men. The mean daily intakes were 132, 141 and 77 g, with medians 113, 128 and 56g, respectively. The intakes of the patients were significantly higher than those of the healthy controls. Both groups of patients took more sugar in more cups of tea or coffee per day than controls.<sup>9</sup>

Yudkin also performed a feeding experiments with rats. A diet high in sugar increased serum triglycerides. Triglycerides are no longer considered to be a high risk factor for heart disease.

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**This was the extent of Yudkins's research.**

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His best-selling book *Pure, White and Deadly: the problem with sugar*<sup>10</sup> contained no references.

Lustig wrote the introduction for the reissue of the book in 2012.

Ansel Keys, Geoffrey Rose and many others criticised Yudkin because “he did not have the data” to support his contention that sugar caused heart disease. Mortality from heart disease started reducing in 1966 in U.S., Finland, and Australia. It was another 10 years before this happened in the United Kingdom because of Yudkin's influence.<sup>11</sup>

Rose believed that there would have been 25,000 fewer deaths in England and Wales if the gains made in Australia and America were duplicated in the United Kingdom.<sup>12</sup>

This does not mean that Keys approved of the high level of sugar consumption:

None of what is said here should be taken to mean approval of the common high level of sucrose in many diets. But there are plenty of good arguments to reduce the flood of dietary sucrose without building a mountain of nonsense about coronary heart disease.<sup>13</sup>

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Sweeney showed in 1927 that high fat diets increase insulin resistance.<sup>14 15</sup>

Students were fed their diets four highly improbable diets for two days: a high fat diet, high carbohydrate diet, high-protein diet and a fasting diet. A glucose tolerance test was performed on the morning of the third day.

After only two days on their experimental diets, the only group showing a normal, healthy response to the glucose tolerance test was the high-carbohydrate group. Both the high-fat and fasting diet showed – after only two days – a significant increase in insulin resistance.

The relationship between an increase in fats in muscle cells and insulin resistance has been documented since the late 1990s.<sup>16 17 18</sup>

Normally, insulin attaches to protein receptors on the cell's surface and signals the cell membrane to allow glucose to enter. If there is an accumulation of fat in the cell, it interferes with insulin's signaling process and glucose cannot enter the cell. Fat can accumulate inside muscle cells even in slim people. The real cause of type 2 diabetes is not an excess of sugar or carbohydrates. It is an accumulation of fat inside the cells that interferes with the muscle cells ability to respond to insulin. The muscle cells are unable to access glucose, which is required for energy production.

Type 2 diabetes does result in an increase in glucose in the blood and urine but this does not mean that Type 2 diabetes is caused by a consumption of sugars or carbohydrates.

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The food we eat is a complex combination of many components—many different types of fat, carbohydrates, amino acids, and dietary fiber along with a multitude of micro-nutrients including vitamins, minerals, carotenoids, and polyphenols. Focusing on one component such as sugar, carbohydrates, fats or saturated fats is not very productive.

A whole-food, plant-based is low in simple sugars, fats and saturated fats and high in fibre, carbohydrates and antioxidants.

### **Related articles**

[Ancel Keys and the High-Fat Diet “Experts”](#)

[Ancel Keys did not manipulate his data](#)

[The Big Fat Surprise](#)

[TIME Magazine Article – Eat Butter](#)

[TIME Magazine Article – Eat Butter – Part 2](#)

[Heart of the Matter – ABC Catalyst](#)

## The Pioppi Diet

## Footnotes

1. Lustig, R. (2013) *Fat Chance: Beating the Odds Against Sugar, Processed Food, Obesity, and Disease*. New York: Penguin Group
2. U.S. Department of Agriculture (n.d.) USDA Food Composition Databases.
3. Boulding, C. (2012) *The Men Who Made Us Fat*.
4. Truswell, A. S. (2010) *Cholesterol and Beyond: The Research on Diet and Coronary Heart Disease 1900-2000*. Springer Netherlands
5. Keys, A. et al. (1980) *Seven Countries: A Multivariate Analysis of Death and Coronary Heart Disease*. Cambridge, Massachusetts and London, England: Harvard University Press, 335.
6. Bronte-Stewart, B. et al. (1955) Serum-cholesterol, diet, and coronary heart-disease. An inter-racial survey in the Cape Peninsula. *Lancet*. 2691103-1108.
7. Keys, A. et al. (1958) Lessons from serum cholesterol studies in Japan, Hawaii and Los Angeles. *Annals of Internal Medicine*. 48 (1), 12.
8. Yudkin, J. (1963) Nutrition and palatability with special reference to obesity, myocardial infarction, and other diseases of civilisation. *Lancet*. 281 (7295), 1335-1338.
9. Yudkin, J. & Roddy, J. (1964) Levels of dietary sucrose in patients with occlusive atherosclerotic disease. *Lancet*. 284 (7349), 6-8.
10. Yudkin, J. (1972) *Pure, White and Deadly: the problem of sugar*. London: Davis-Poynter Limited.
11. Truswell, A. S. (2010) *Cholesterol and Beyond: The Research on Diet and Coronary Heart Disease 1900-2000*. Springer Netherlands, 9.2.
12. Truswell, A. S. (2010) *Cholesterol and Beyond: The Research on Diet and Coronary Heart Disease 1900-2000*. Springer Netherlands, 31.4.
13. Keys, A. (1971) Sucrose in the Diet and Coronary Heart Disease. *Atherosclerosis*. 14 (1), 200.
14. Sweeney, J. S. (1927) Dietary Factors that Influence the Dextrose Tolerance Test. *Archives of Internal Medicine*. 40 (6), 818-830
15. Sweeney, J. S. (1928) A comparison of the effects of general diets and of standardized diets on tolerance for dextrose. *Archives of Internal Medicine*. 42 (6), 872-876.
16. Jacob, S. et al. (1999) Association of Increased Intramyocellular Lipid Content With Insulin Resistance in Lean Nondiabetic Offspring of Type 2 Diabetic Subjects. *Diabetes*. 48 (21), 1113-1119.
17. Bachmann, O. P. et al. (2001) Effects of Intravenous and Dietary Lipid Challenge on Intramyocellular Lipid Content and the Relation With Insulin Sensitivity in Humans. *Diabetes*. 50 (13), 2579-2584.
18. Krssak, M. et al. (1999) Intramyocellular lipid concentrations are correlated with insulin sensitivity in humans: a <sup>1</sup>H NMR spectroscopy study. *Diabetologia*. 42 (1), 113-116.