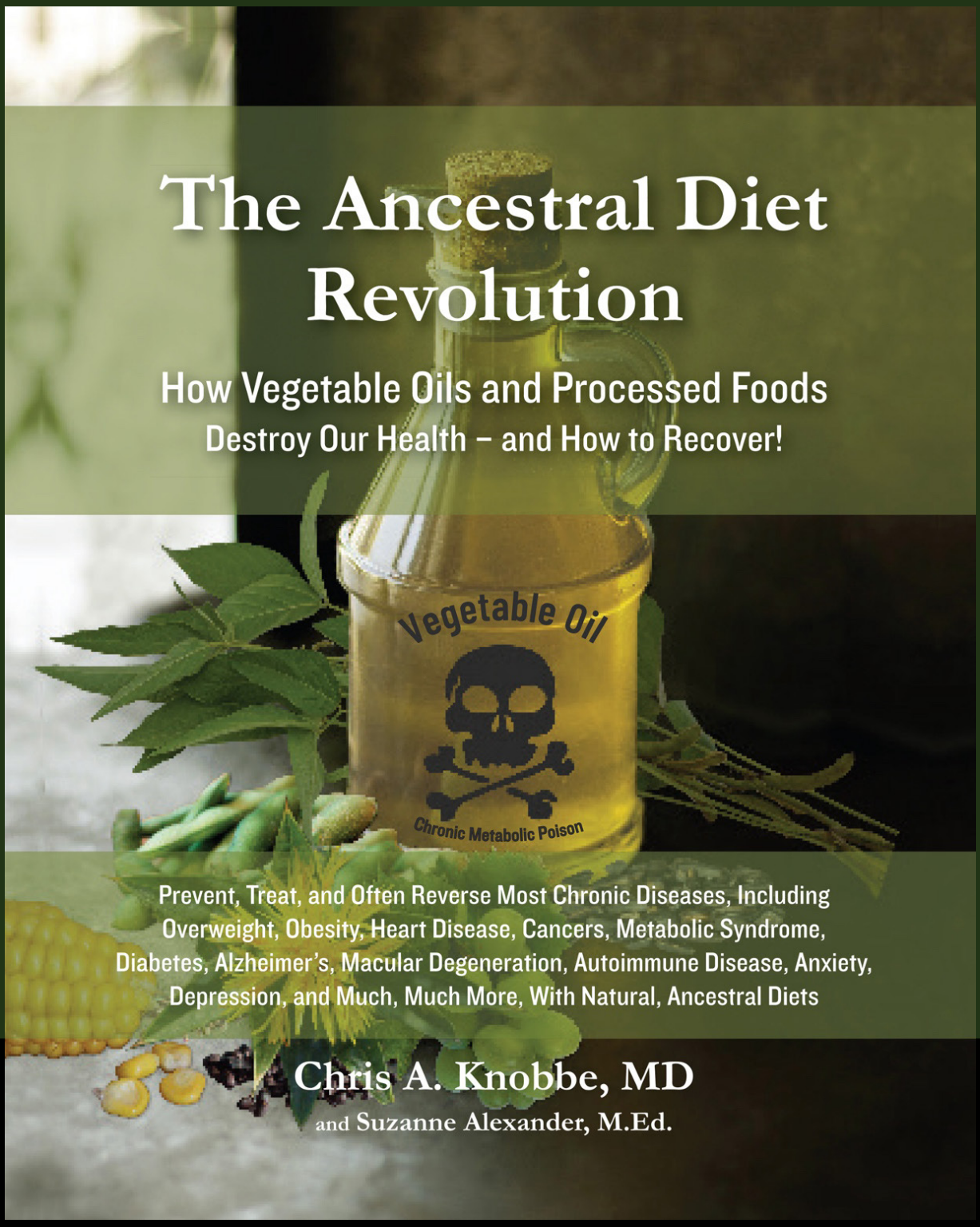
The Ancestral Diet Revolution

A Book Synopsis

With New Evidence Not Presented in the Book*

by Chris Knobbe, MD



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Industrial Seed Oils and Their Relationship to Obesity & Diabetes*

In nutrition circles today, there is a pervasive belief that either sugars, carbohydrates, or possibly processed carbohydrates are the primary drivers of obesity and diabetes. There is also a firmly held theory that the sugar fructose is the problem. There are lesser-held hypotheses that we just overeat or that wheat or grains, in general, are the primary culprits. Finally, many submit that we're lazy and lack of exercise is the primary driver.

I beg to differ with all of these competing hypotheses.

Why? The scientific evidence does not support any of these conclusions.

For example, in the United States, United Kingdom, Australia, Japan, and Israel, various combinations of total calories, carbohydrates, and total added sugars have shown declines over decades, while at the same time, obesity, overweight, and diabetes all markedly increased.

In the United States and Japan, for example, we have observed over decades decreasing consumption of total calories, carbohydrates, and total added sugar consumption, while obesity and diabetes have both escalated dramatically.

But what is the one food component that is rising? Industrial seed oils.

When we observe such relationships in a single population, e.g., the U.S., even a single time, it becomes exceedingly difficult to blame sugars or even refined carbohydrates for the induction or propagation of these conditions, despite the fact that added sugars and refined carbohydrates are indeed potentially problematic since they are nutrient deficient.

However, when we observe such relationships repeatedly in multiple populations over decades of time, it becomes beyond difficult to lay the blame on either too many calories, carbohydrates, or refined sugars.

Indeed, Weston A. Price first showed that refined sugars and flours are problematic in multiple scientific papers, culminating in the first and second editions of his textbook, *Nutrition and Physical Degeneration*, published in 1939 and 1945. Price's research firmly implicated refined flour, refined sugar, canned goods, sweets, confectionary, and "vegetable fats" as causal in dental decay, arthritis, cancers, congenital malformations ("birth defects"), and other physical degenerative disorders. However, he did not implicate these foods as etiologic agents of obesity and diabetes.

Price, however, did not address obesity in his classic text, *Nutrition and Physical Degeneration*, and he scarcely mentioned diabetes. These conditions appear to have been quite unusual or even rare in the 1930s throughout much of the world, particularly in the populations that Price studied.

Not only will I implicate seed oils as having major causal roles in obesity and diabetes but also in coronary heart disease (CHD), cancers, metabolic syndrome, dementia, age-related macular degeneration (AMD), autoimmune diseases, and many other chronic diseases. In this brief review, however, I'll focus on the proposed causal role of industrial seed oils in overweight, obesity, and diabetes.

* This writing is not fully referenced; however, all references are available via the source from which this was primarily taken, i.e., *The Ancestral Diet Revolution*, 2023, by Chris Knobbe, MD, and Suzanne Alexander.

The Seed Oil Hypothesis of Obesity and Chronic Disease

All relatively new theories have proponents and opponents, protagonists and antagonists, champions and critics. This hypothesis is no different.

For clarification, I didn't develop the hypothesis that seed oils are drivers of chronic disease. In fact, I wouldn't even credit Weston A. Price with initiating the theory. However, Price repeatedly named "vegetable fats" as one of the food groups of potential harm in his 1939 textbook *Nutrition and Physical Degeneration*. Price included vegetable oils in his list of "displacing foods of modern commerce" because, along with refined sugars, refined flour, canned goods, and sweets, vegetable oils are entirely devoid of nutrients in the form of vitamins A, D, and K2. They are altogether devoid of minerals except for trace amounts of iron.[1]

I would credit nutrition researcher E.V. McCollum, a researcher in the 1910s at the University of Wisconsin–Madison and subsequently at Johns Hopkins University, with first implicating vegetable oils as harmful due to their lack of fat-soluble vitamins. McCollum firmly established, by 1918, that young animals fed vegetable oils as their only source of fat would soon stop growing, develop rough coats, crooked and abnormal teeth, red and matted eyes, night blindness, infections, and finally, if the diet was continued, weight loss and death would ensue.

Since then, thousands of researchers have wrestled, experimented, and grappled with the different physiological and pathological outcomes of feeding (or consuming) animal fats versus vegetable oils.

At the time of this writing, I've spent most of the last thirteen years digging into this history and these questions. As you will soon see, if you are unfamiliar with my work, I am a self-professed "data junkie" – and I believe that, when it comes to Nutrition Science, it's impossible to support or refute a hypothesis without good, solid, verifiable data.

Food consumption data, plotted against disease prevalence (in graphic form), has led me to believe that seed oils are the single greatest harmful component in the food supply.

Industrial seed oils, in fact, vegetable oils in general, represent the single greatest change to the diet of mankind in all of history.[2]

In numerous countries as well as the entire world, our works have shown that total vegetable oil consumption has risen in direct correlation to obesity, diabetes, and most chronic diseases.

Given these facts (to be reviewed in detail subsequently), wouldn't it make sense to infer that vegetable oils are indeed the primary driver, particularly if no other major food components show such solid, repetitive, and reliable correlations?

With that said, consider the following: If sugar and carbohydrate consumption are on the decline for an extended period, e.g., many years, while obesity and diabetes are on the rise in the same population(s) and during the same time frame(s), isn't this evidence against their etiology in these disorders?

Furthermore, if most of the population is affected by the condition in question, e.g., overweight and obesity (which affected a total of 73.6% of American adults in 2018), we can reasonably infer that most of the population, on average, is consuming the major dietary constituents (e.g., proteins, carbohydrates, fats, seed oils, sugars, flours, etc.). Then, if sugar and carbohydrates are on the decline while overweight, obesity, and diabetes are on the rise, wouldn't it make more sense to infer, or at least suggest, that sugar and carbohydrates might prevent these conditions rather than cause them?

It is not my suggestion that sugar consumption is protective against overweight, obesity, and diabetes, but I want to pose this question since, as mentioned previously, in a number of populations, sugar consumption is negatively correlated with obesity.

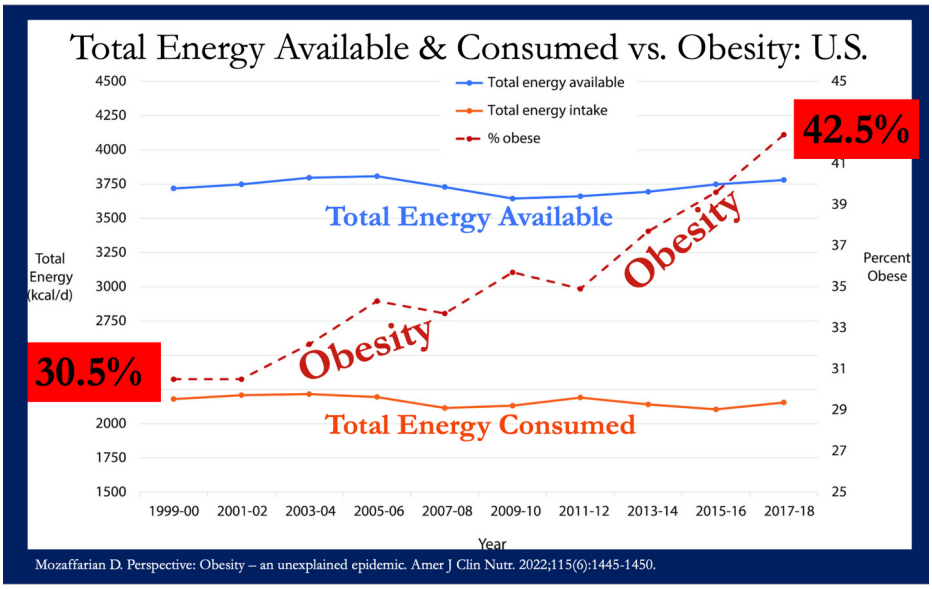
One by one, let's examine some of the most popular competing hypotheses for the primary cause of obesity and diabetes.

Total Calories Consumed and Obesity in the U.S.

Let's examine the relationship between total calories consumed and obesity.

Observe the graph below, published by Dariush Mozaffarian, MD, MPH, of Tufts University, in a paper entitled "Perspective: Obesity, an Unexplained Epidemic."[3] In the graph presented in this paper, Mozaffarian shows data indicating that total energy (food) availability has shown almost no change from 1999 through 2018. Furthermore, total energy consumed has declined slightly since 2003-04. Mozaffarian states, "NHANES data suggest small but statistically significant declines in energy intake over this period [1999-2018]."[3]

Yet, obesity has steadily risen during this entire period, rising from 30.5% in 1999 to 42.5% in 2018.



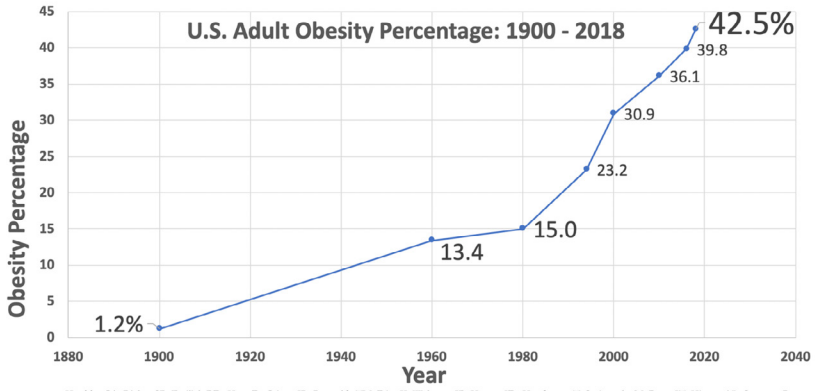
(Graph in its original form; published here with additional labeling to clarify understanding.)

In the opening abstract of this paper, Mozaffarian wrote, "Since 1980, obesity prevalence among U.S. adults has soared from 14% to 42%. The commonly accepted explanation is pervasive overeating: ever-increasing energy intake as the population gains weight, year after year. However, evidence does not support this hypothesis."[3] He continues, "Americans appear to be eating relatively less since 2000, for ever-increasing body sizes, as time has progressed." Finally, Mozaffarian laments, "It is time to share a striking, and not widely appreciated, secret: we do not have a clear explanation for the obesity epidemic."

Might it be that, perhaps before 1999, total calories consumed might explain the obesity epidemic, but for unknown reasons, it does not now?

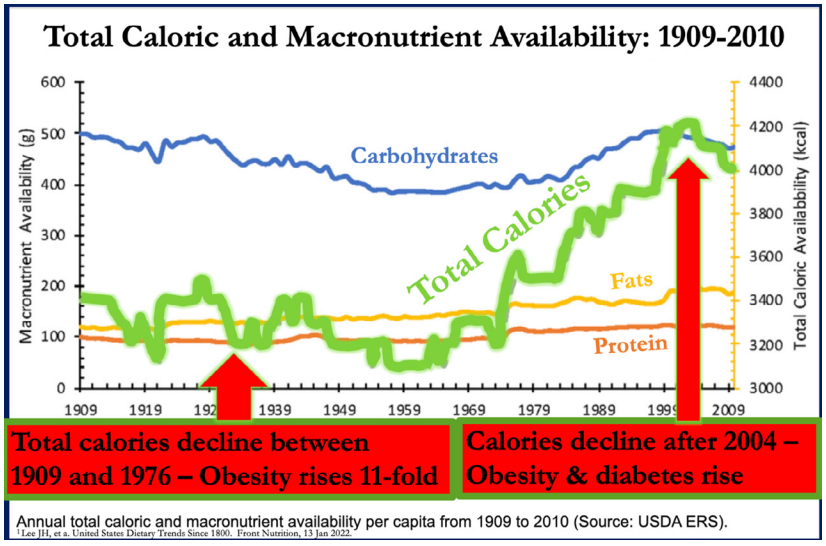
To evaluate the "overeating hypothesis" of obesity, we must first understand the known history of obesity, in this case, in the U.S.

Below is a graph of obesity (BMI ≥ 30) in the United States. Scott Alan Carson's work shows that obesity was 1.2% in men aged 18 to 80 in the late 19th century. No data was available for women in the 19th century. Obesity steadily increased from this 1.2% level to 13.4% by 1961, 15.0% by 1980, 30.9% by 2000, and finally, to 42.5% by 2018.[2]



Knobbe CA, Richer SP, Freilich BD, Kent D, Cripps JR, Reynolds TM, Dito JJ, Walgama JP, Hunter JD, Henderson T, Stojanoska M, Barry JT, Kiwara AL, Sommer R, Ross RD, Kaushal S, Luff AK. Westernized Diet, Omega-6 Rich Vegetable Oils, and Nutrient-Deficient Diets: Epidemiologic Evidence and Pathophysiologic Mechanisms for Age-Related Macular Degeneration and Associated Chronic Diseases. Manuscript submitted for publication. University of Texas Southwestern Medical Center, Dallas, U.S., 2022. © C. Knobbe, 2022. Ancestral Health Foundation.

To examine the hypothesis that we're all just overeating, that is, that we "just eat too much," we'll draw on food consumption data from a paper by Joyce H. Lee et al. published in *Frontiers in Nutrition*, which reviewed evidence of dietary trends in Americans since 1800.[17] Observe the following graph (with enhancements added) from this paper entitled, "Total Caloric and Macronutrient Availability: 1909-2010."



Note from this graph that while caloric consumption declined between 1909 and about 1976, obesity climbed from about 1.2% to around 14%, or at least 11-fold. Caloric consumption then rose from about 1977 through 2004 before falling again through 2018, as reviewed by Mozaffarian.

Thus, the only period in recorded history in which obesity increased while caloric consumption increased was between 1977 and 2004. The remainder of the time, from 1909 to 1976 and from 2004 to 2018, caloric consumption declined while obesity increased.

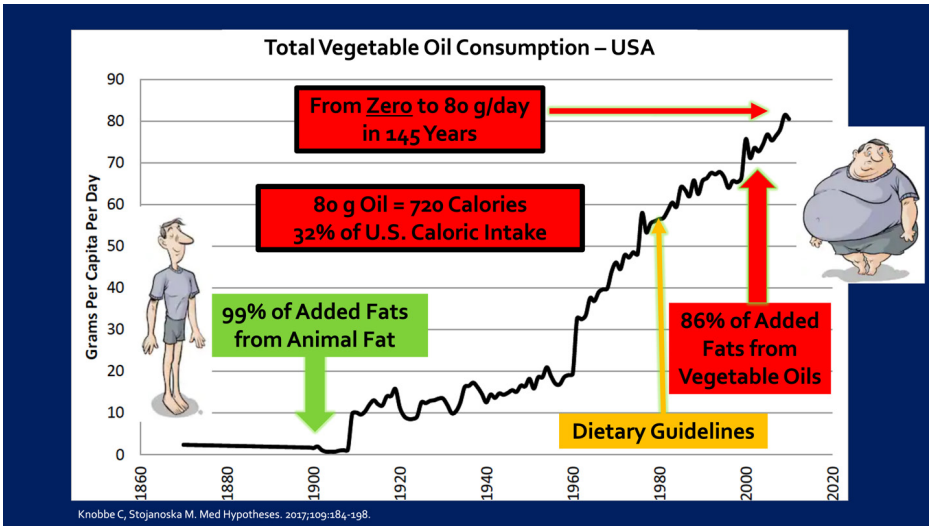
Given this evidence, I will submit that we cannot blame overeating per se as the cause of the obesity epidemic. The 'calories in, calories out' hypothesis of obesity, while valid from a thermodynamic standpoint, does not explain *why* we're becoming progressively more overweight. Something else is at play.

Industrial Seed Oils vs. Obesity

Five years ago at the time of this writing, in August 2019, at the Ancestral Health Symposium held at the University of California, San Diego, I presented a lecture with evidence in support of the hypothesis that industrial seed oils are the primary driver of obesity, coronary heart disease (CHD), cancer, type 2 diabetes (T2DM), metabolic syndrome, and age-related macular degeneration (AMD). Since then, I have given dozens of supportive presentations, many for continuing medical education (CME), in eight countries supporting this hypothesis. Let's observe some of this evidence.

In the graph below, published by our group in 2017 in the journal *Medical Hypotheses*, we observe the history of vegetable oil consumption in the U.S.[4] Please note that Americans consumed virtually no vegetable oils in the year 1865 and that by 2010, vegetable oil consumption had risen to 80 g/capita/day (vegetable oil and sugar data after 1961 is provided by FAO/FAOSTAT food balance sheets, corrected for losses, and provides the amount of food that "reaches the consumer").

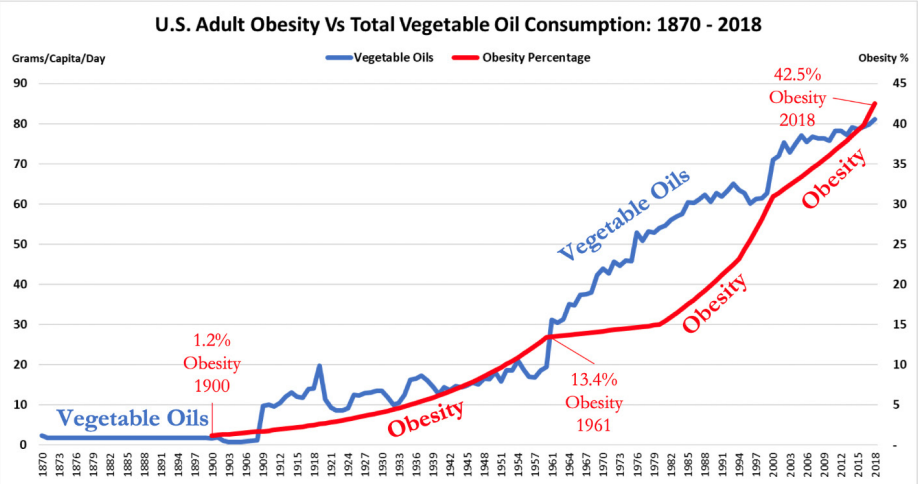
Eighty grams of seed oils is 720 calories per person per day, or about 32% of U.S. caloric intake for the year 2010. With expected plate losses and other waste after reaching the consumer, consumption still accounts for around one-fourth of the average calories consumed by Americans in recent years. Given that some people consume almost zero seed oils, others assuredly get one-third or more of their calories from seed oils.



Knobbe C, Stojanoska M. Medical Hypotheses. 2017;109:184-198.
Graph in its original form, published here with PowerPoint enhancements to clarify understanding.

It is critical to observe that, in 1900, 99% of consumed added fats were derived from animal fats, such as lard, butter, and beef tallow. However, by 2005, 86% of added fats in the diet were derived from vegetable oils.

In the following graph, we observe the relationship between vegetable oil consumption and obesity in the U.S., with data back to the 1860s. Vegetable oil consumption, except for trivial amounts (a small fraction of a gram/day) of olive oil consumed for hundreds of years, began with cottonseed oil shortly after the end of the American Civil War, in approximately 1866.



References: 1) Knobbe C, Stojanoska M. The 'Displacing Foods of Modern Commerce' Are the Primary and Proximate Cause of Age-Related Macular Degeneration: A Unifying Singular Hypothesis. 2) Obesity Statistics: See references herein. © C. Knobbe, 2022. Ancestral Health Foundation.

In 1900, when obesity was 1.2% in U.S. men, vegetable oil consumption was about 1.0 gram/capita/day. By 1961, obesity had risen to 13.4%, an 11-fold increase from the prevalence in 1900, and vegetable oil consumption was 19 g/day, which is approximately a 19-fold increase from the 1900 level.

By 2018, obesity had risen to 42.5%, and vegetable oil consumption had increased to about 80g/day (the 2010 level), which is about 80-fold higher than in 1900 and 4-fold higher than in 1961.

In short, we observe an exquisitely high degree of correlation between industrial seed oil consumption and obesity.

Furthermore, as noted earlier, the 80 g of seed oils per day is 720 calories worth of food, none of which existed in 1865. Thus, in about 150 years, roughly one-fourth to one-third of the food supply was supplanted and replaced by industrial seed oils.

Thus, industrial seed oil consumption represents the single greatest change to the diet of mankind, in all of history.[2]

At this point, let's define "vegetable oils" as used in this writing. These are the oils of soybean, corn, canola, cottonseed, rapeseed, grapeseed, sunflower, safflower, rice bran, sesame, groundnut (peanut), palm, palm kernel, coconut, olive, and avocado oils, with trivial amounts of other oils. The enormous bulk of the consumed oil is made up of "industrial seed oils," which, for the sake of this discussion, includes the oils of soybean, corn, canola, rapeseed, grapeseed, sunflower, safflower, and rice bran. Most of the oil consumed in restaurants and fast-food restaurants in the U.S. is derived from soybean and canola oil.

In 2010, the oils of soybean (soyabean), groundnut, sunflower, rape (canola), mustard, cottonseed, sesame, rice bran, and corn oils made up 88.7% of the total consumed oils.[11] The remaining 11.3% comprises mostly tropical oils (palm, palm kernel, and coconut) and fruit oils (olive and avocado), with just trivial amounts of other oils.

While the public and many healthcare practitioners applaud the benefits of olive oil, between 1990 and 2010, the average American consumed an average of just 1.6 g of olive oil per day, amounting to 14.5 cal/day.[11] In 2010, out of 80 g of total vegetable oils consumed per day, just 2.4 g (21.6 calories) was olive oil, which amounts to about one-half teaspoon per day and just 1.0% of calories.[11] [5] That is, as a nation, the consumption of olive oil is virtually trivial. The same is true globally.

Let's face it. As a nation, about 89% of the vegetable oils we've consumed in recent years are industrial seed oils, with the remaining 11% being those of olive, coconut, and palm kernel, which I would consider the healthiest options.

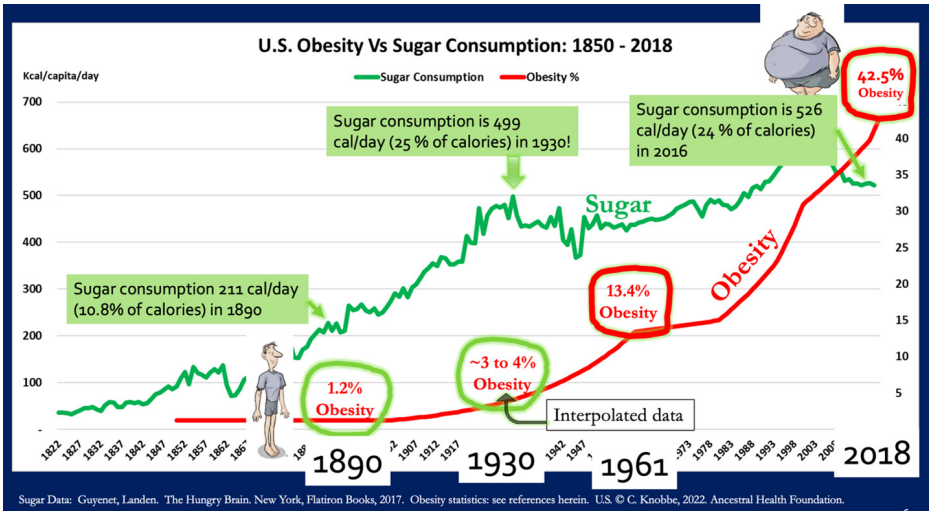
Below, we observe the image of an industrial seed oil refinery.[6] To be fair, most don't look quite like this. However, the very sight of such a factory might suggest that the end products are anything but natural and healthy food. The industrial seed (and soybean) oils produced in these factories are subjected to an intense refining process that includes, initially, crushing, heating, and pressing of the seeds; second, a petroleum-derived hexane solvent bath; third, steam or heat evaporation and distillation, and finally, chemical alkalinization, bleaching, and deodorization.[7] [8]

This industrial process is the simplified version of a very intensive mechanical, chemical, and heating process. The end product oils would reek of extreme rancidity (oxidation) if not for the final deodorization phase, which subjects such oils to temperatures often exceeding 500° F. In *The Ancestral Diet Revolution*, I've revealed in great detail how consumption of such factory-produced oils induces a metabolic environment that is pro-oxidative, pro-inflammatory, toxic, and nutrient deficient.

Source: How a Vegetable Oil Refinery Plant Works. Oil Refinery Plant. 27 Mar 2012.[6]

Sugar Consumption vs. Obesity

Next, let's examine sugar consumption versus obesity. Please observe the following graph, "U.S. Obesity vs Sugar Consumption: 1850 – 2018."



In 1890, when obesity in men was 1.2%, sugar consumption had already risen to 211 cal/day, which was 10.8% of calories consumed. Today, the World Health Organization (WHO) recommends no more than 10% of calories be consumed as added sugar while advising that reducing to less than 5% of calories as sugar "would provide additional health benefits."[9]

However, Americans had already exceeded the 10% level of sugar consumption by 1890, and yet, obesity was about one percent, coronary heart disease ending in myocardial infarction (MI, "heart attack") was virtually unknown (e.g., see Sir Wm. Osler's *Principles and Practice of Medicine*, 1892), cancer was unusual (1 in 17 deaths in 1900, vs. 1 in 3 today), Alzheimer's disease had never been diagnosed, diabetes was exceedingly rare (about 1 in 36,000 people in 1890 vs. 1 in 8 today), and finally, age-related macular degeneration (AMD) was extraordinarily rare (less than 50 cases in all of the world's literature between 1851 and 1930).[10]

By 1930, sugar consumption had elevated to 499 cal/day, or 25% of caloric consumption, yet obesity was only about 4.09% (interpolated data, as there are no documented obesity prevalence studies between 1900 and 1961). If one were to review the data and graph and conclude that I picked a high point for sugar consumption, please observe that the approximate average sugar consumption between 1922 and 1987 is around 475 calories. In short, there is very little difference in sugar consumption for nearly all of those 65 years.

By 1961, obesity had risen to 13.4%, an approximately 3.3-fold increase from the 1930 prevalence. However, sugar consumption was virtually flat during that same time period.

If we jump to 2018, we observe that obesity had risen to 42.5%. Sugar consumption in 2018 was 526 cal/day or 24% of calories, exactly 27 calories higher than the 1930 level. Yet, obesity had climbed from around 4% in 1930 to 42.5% by 2018.

Are we to blame this massive increase in obesity – on an extra 27 calories worth of sugar per day? Literally, just over one teaspoon of sugar?

Sure, there is an 11-year period between 1988 and 1999, where sugar consumption increased by about 100 calories per day, up to just over 600 calories per day. But after 1999, sugar has been on the decline while obesity has taken perhaps the sharpest rise in history. We'll explore this evidence in more detail subsequently.

Are We Including All Forms of Added Sugar? Yes, Of Course!

This might be a good time to define "sugar" as used in the present data and graphs. We have and will frequently use the Food and Agriculture Organization of the United Nations (FAO) data, which is available from FAOSTAT.[11] The FAOSTAT data is presented in the form of "Food Balance Sheets," which are available by country as "food supply (kcal/capita/day), "Sugar and Sweeteners (Total)," and for any given set of years, dating only as far back as 1961. The "sugar and sweeteners (total)" includes the following: "sugar (raw equivalent), sweeteners, other, and honey." This includes all forms of added sugar and, of course, does not include naturally occurring sugars found in milk, fruit, and vegetables. The FAO data is presented as the food quantity that "reaches the consumer."

We also present data from the USDA Economic Research Service (ERS), which defines "added sugars" as follows: "Added sugars include sugars that are added during the processing of foods (such as sucrose and dextrose), foods packaged as sweeteners (such as table sugar), sugars from syrups and honey, and sugars from concentrated fruit or vegetable juices. They do not include naturally occurring sugars that are found in milk, fruits, and vegetables."[12]

For data extraction methods and results on sugar and vegetable oil consumption before 1961 dating back into the 19th century, please see the methodology and references in our 2017 published paper.[4]

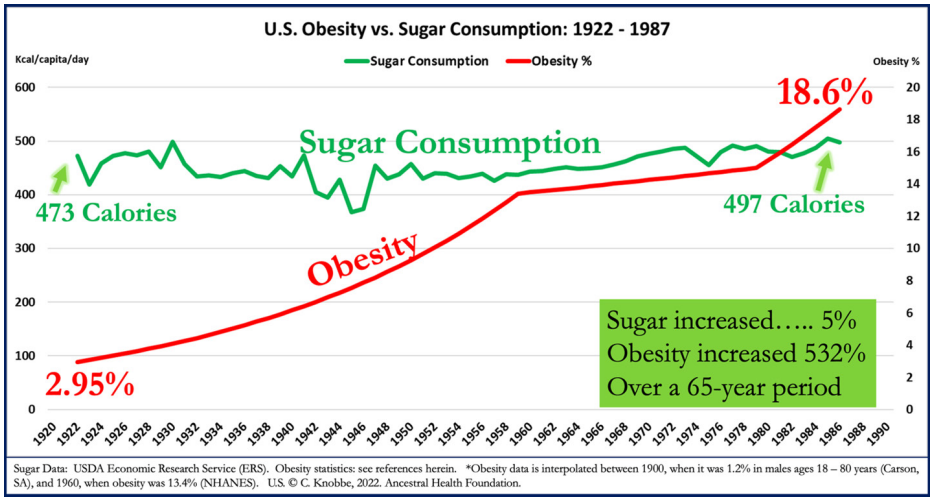
Thus, every form of added sugar is included in this category, and that includes all forms of table sugars, cane and beet sugars, corn syrup, high-fructose corn syrup (HFCS), maple syrup, plus all forms of sugar-sweetened beverages (SSBs), whether in fruit or vegetable juices or soft drinks. No form of added sugar is excluded. Natural sugars, such as those in milk, fruits, vegetables, and naturally squeezed fruit juices, are not "added sugars."

Without fail, it seems that a few detractors of our work invariably question the added sugar data that we have previously published and presented, most commonly with *invalid* assertions that we have not included corn syrup, high-fructose corn syrup, maple syrup, and perhaps other sweeteners in our data on sugar consumption. Such claims are patently false.

All food availability and consumption data from the FAO (FAOSTAT, food balance data sheets) and the USDA Economic Research Service (ERS) are available to researchers and the public. We use both sources, as both are highly valuable and help corroborate consumption evidence.

U.S. Obesity vs. Sugar Consumption: 1922 – 1987

Let's zoom in on the relationship between sugar and obesity during a 65-year period of time in the U.S. between 1922 and 1987. See the following graph, "U.S. Obesity vs. Sugar Consumption: 1922 – 1987."



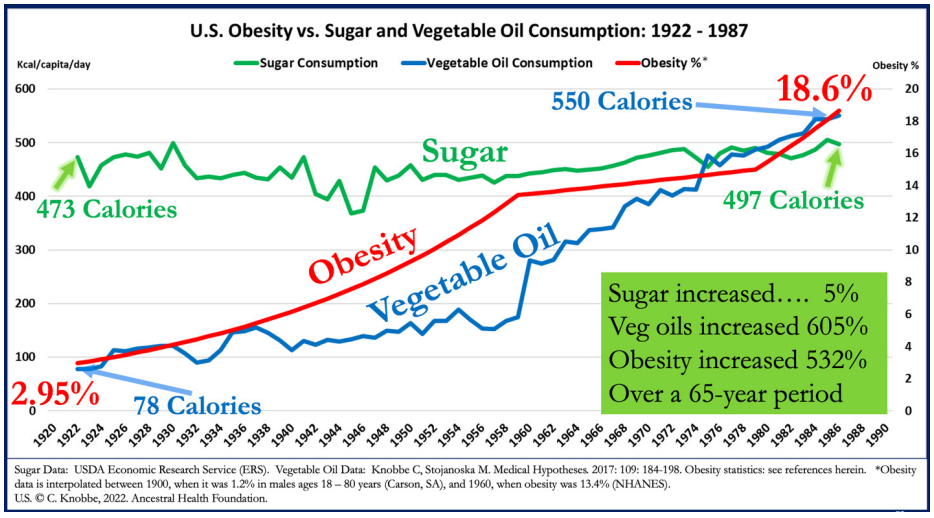
In this 65-year period, note that sugar consumption was 473 cal/day in 1922 and remained almost entirely flat, with consumption at 497 calories in 1987. This is a difference of 24 calories worth of sugar, yet obesity increased 6.32-fold, from 2.95% to 18.6%, a 532% increase.

I repeat. This is a 65-year period of time. If sugar is the cause, or even a cause, of obesity, then why did obesity increase more than 6-fold while sugar consumption remained virtually flat?

"Sugar theorists" (of obesity, diabetes, and other chronic disease) must explain this if this is their position.

Industrial Seed Oils and Sugar vs. Obesity

Next, let's evaluate both vegetable oil and sugar consumption versus obesity.

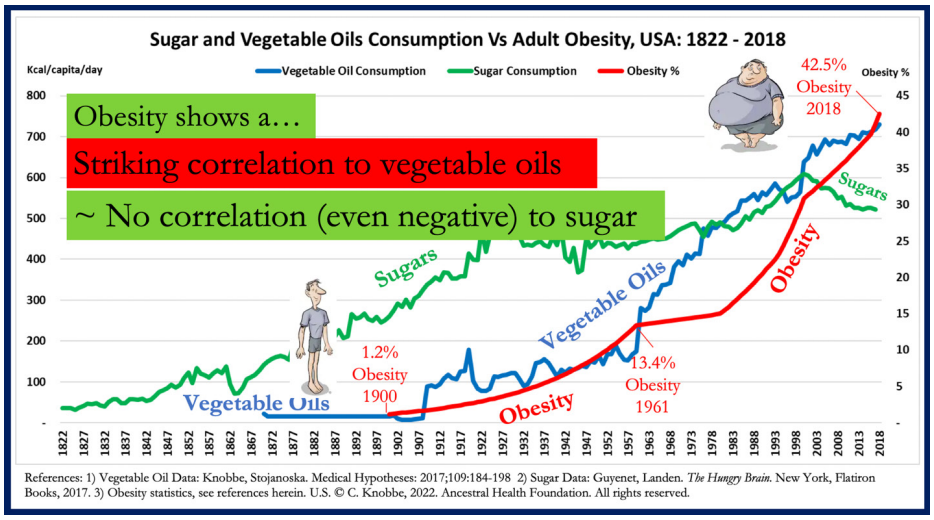


During this time frame previously evaluated (1922-1987), we observe vegetable oil consumption increased from 78 cal/day in 1922 to 550 cal/day in 1987, an increase of 7.05-fold, or 605%.

Thus, while obesity elevated 6.32-fold, from 2.95% to 18.6%, vegetable oils increased 605%, and sugars increased 5%.

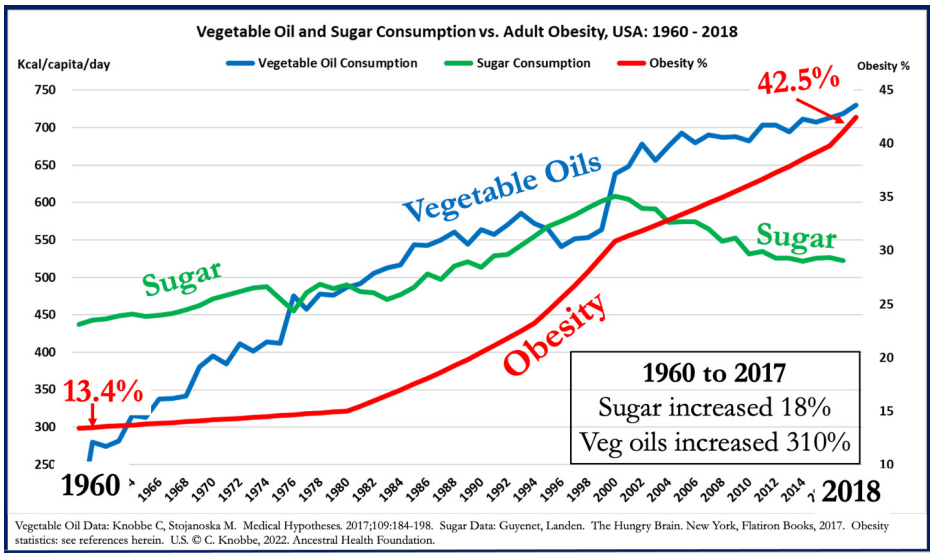
Again, how can we causally link sugar to obesity in this scenario? Sugar consumption was virtually flat, while obesity elevated exponentially. Once again, for "sugar theorists," this must be explained, and, quite frankly, most all of the other data presented herein regarding sugar as a cause of obesity should require an explanation.

Note the following graph, "Sugar and Vegetable Oil Consumption vs Adult Obesity, USA: 1822-1918":



Even to the untrained eye, one may observe a striking relationship between obesity and vegetable oils but little or no correlation – even a negative correlation – between sugar and obesity. Again, note the steady decline in sugar consumption after 1999, for example, while obesity continues a steep vertical ascent.

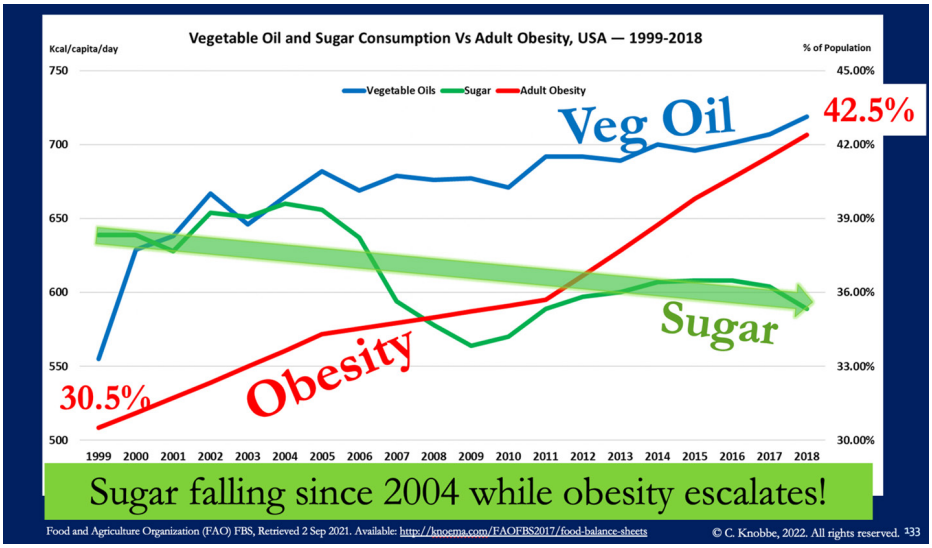
Next, we will zoom in on the data further, examining the period between 1960 and 2018.



Note that, during this same approximate period (1960 to 2017), sugar consumption elevated from 443 cal/day to 522 cal/day, an increase of just 18%. During the exact same period, vegetable oils increased from 175 cal/day to 718 cal/day, an increase of 310% (4.1-fold), and obesity increased in prevalence from 13.4% in 1961 to 42.5% in 2018.

To summarize this period of time, between 1961 and 2018, sugar consumption increased 18%, vegetable oil consumption increased 310%, and obesity elevated 3.17-fold, or 217%. Again, only vegetable oil consumption is tightly correlated to obesity. Clearly, sugar consumption is not.

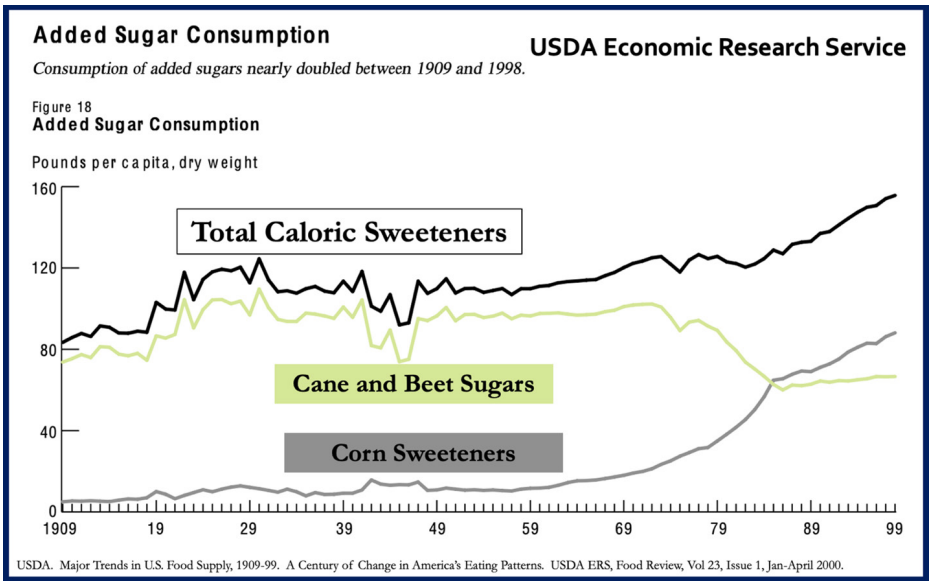
In the next graph below, we further narrow our focus on the period between 1999 and 2018. During this time frame, we observe that obesity climbed from 30.5% to 42.5%, as vegetable oil consumption increased from 564 cal/day in 1999 to 713 cal/day in 2016, while sugar consumption dropped from 602 cal/day in 1999 to 526 cal/day in 2016.



On the graph, we indeed observe a substantial drop in sugar consumption after 2004 while obesity escalates.

However, vegetable oil continues to rise through the end of this time period, and so does obesity.

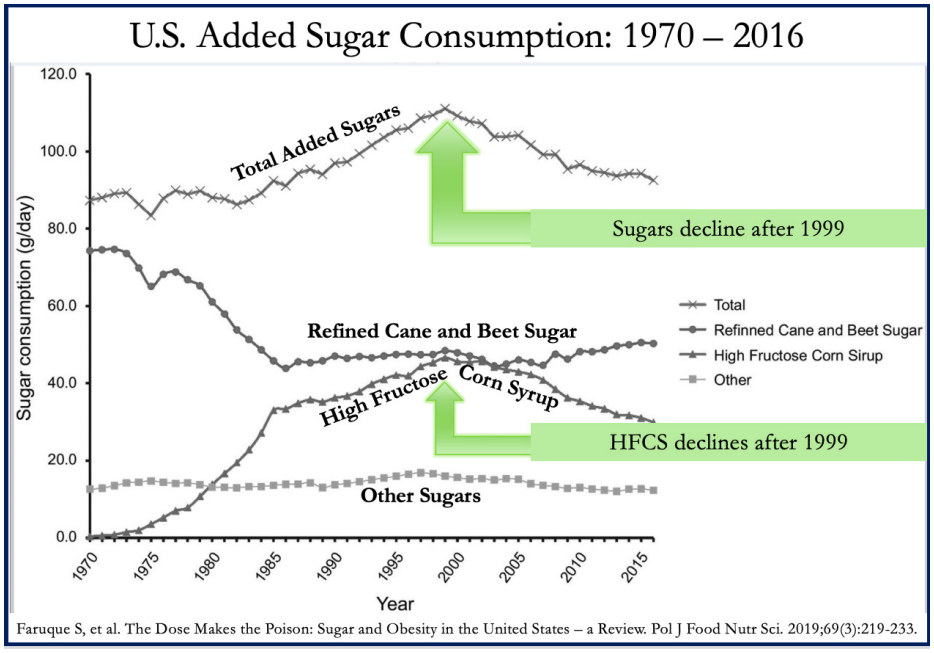
For those who want further confirmation that sugar consumption data accounts for all forms of added sugars, below is a graph from the USDA's Economic Research Service (ERS), showing added sugar consumption in the U.S. between 1909 and 1999:



To estimate final food consumption, the USDA ERS imposes a much larger estimation of losses (historically averaging about 28.8%) than the FAO, which is also corrected for losses but estimates food availability that actually "reaches the consumer."

Again, we're presenting data from both sources. Why? Once again, we observe that both databases are extremely valuable, and both are regarded as very close approximations of food consumption. Furthermore, this data is remarkably accurate in most large and developed countries since national production, imports, and exports are closely tracked via economically motivated traceability systems.[13]

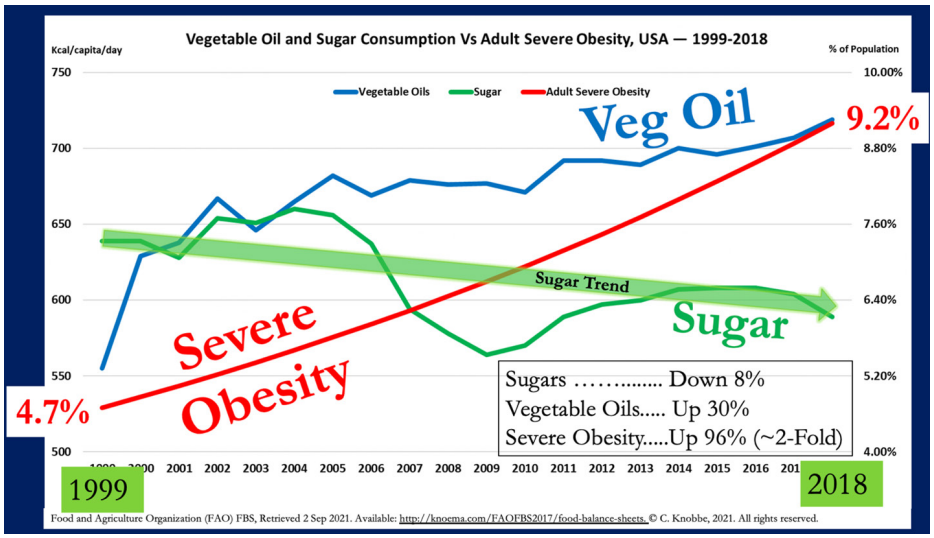
Further evidence of the sugar consumption data we've presented (already representing both the FAO and USDA ERS) is a graph independently published by Faruque et al., who argue that sugar is a significant driver of obesity. They state, for example, "Evidence suggests that diets high in added sugar promote the development of obesity."[14]



Note that the data from Faruque et al. is from the USDA ERS and is entirely consistent with the ERS and FAO data we've presented. However, as reviewed, all consumption data from the ERS shows lower values than the FAO food availability datasets because the ERS estimates greater total losses.

We should observe, precisely as noted before, that the data published by Faruque et al., derived from the USDA ERS, shows what we have observed via the ERS data presented elsewhere. Total added sugars have declined since 1999, and in the words of Faruque et al., "the decline of total sugar consumption after 2002 can be attributed to the considerable decrease in the HFCS consumption [USDA, 2017]."[14]

Next, we'll observe severe obesity versus sugar and vegetable oil consumption. As we observe in the graph below, between 1999 and 2018, severe obesity increased from 4.7% to 9.2%. That is, severe obesity doubled during a period of declining sugar consumption.



Once again, if sugar is the primary culprit in obesity, why is consumption on the decline while severe obesity in the U.S. doubled during this period of time? Of course, total vegetable oil consumption continued to rise during this period.

Clearly, sugar consumption has a very poor correlation to increasing obesity prevalence. We've observed, for example, that, in 1922, sugar consumption was already 473 calories/day, or approximately 24% of total calories, yet obesity was only about 2.95%. Obesity then elevated 6.3-fold by 1987 to 18.6%, or 6.3-fold. However, sugar consumption increased by only 24 calories/day between 1922 and 1987, to 497 calories per day, which is about 22.2% of total calories (NHANES 1988[15]). Vegetable oils, in contrast, increased from 78 cal/day to 550 calories/day, or an increase of 605% during this same period.

To summarize just this time frame, between 1922 and 1987, sugar elevated by 24 calories/day, while vegetable oils increased by 472 calories/day, and obesity increased 6.3-fold. In terms of percentages, during this time frame, sugars increased 5%, vegetable oils increased 605%, and obesity elevated 532%.

Why would it make sense to blame sugar in this scenario? There is a highly variable correlation to increasing obesity, ranging from positive to negative to null. There is no dose-response effect. Furthermore, as we will review subsequently, regression analysis will prove that, when the effect of vegetable oils is taken into account, there is no effect of sugar on obesity whatsoever.

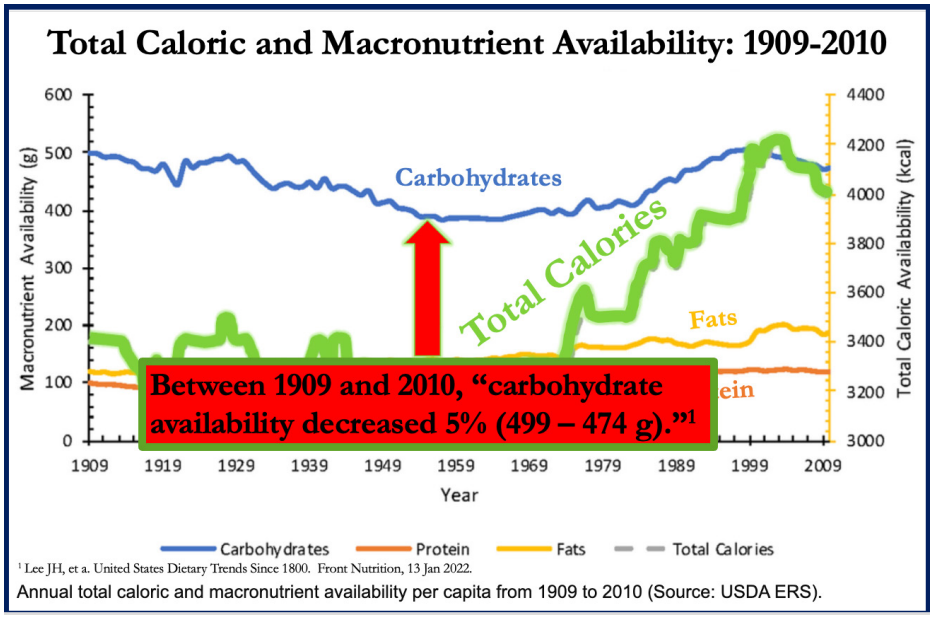
Are Carbohydrates Driving Obesity?

The evidence presented so far is anything but supportive of the hypothesis that sugar is the culprit in obesity and diabetes. But could carbohydrates, in general, be driving obesity? Let's examine the more general theory that carbohydrates per se are the primary culprit, or even a culprit, in obesity development.

The "carbohydrate-insulin model of obesity" appears to be the reigning pathophysiological model to explain why carbohydrates might drive obesity, i.e., carbohydrates increase insulin secretion, and insulin induces fat storage.[16] According to leading proponents of this theory, "the Carbohydrate-Insulin Model of obesity (CIM) proposes that a high-carbohydrate diet – including large amounts of refined starchy foods and sugar, as commonly consumed in the low-fat diet era – produces postprandial hyperinsulinemia, promotes deposition of calories in fat cells instead of oxidation in lean tissues, and thereby predisposes to weight gain through increased hunger, slowing metabolic rate, or both."[16]

If the CIM model is accurate, then we should historically observe increasing carbohydrate consumption, or perhaps at least increased sugar and refined carbohydrate consumption, over long periods of time in correlation to rising obesity prevalence. Could this be a tenable hypothesis?

To examine the epidemiologic evidence for this theory, we'll draw upon food consumption data from a paper previously reviewed by Joyce H. Lee et al., published in *Frontiers in Nutrition*, which documented dietary trends in Americans since 1800.[17] See the graph below from this paper (with labeling modifications to enhance understanding):



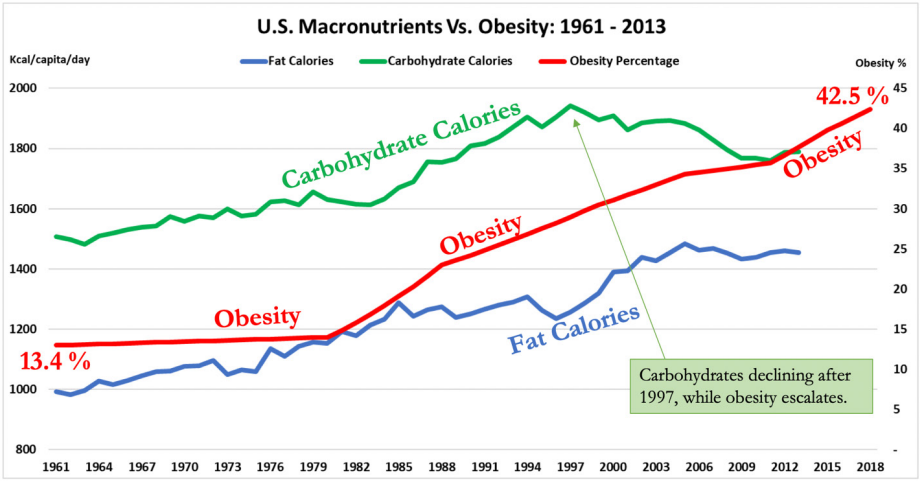
In this paper, Lee et al affirm, "Available daily calories averaged 3,400 kcal in 1909 and increased 18% over the century to 4,000 kcal in 2010. In the same time period, carbohydrate availability decreased 5% (499 – 474 g)… fat availability increased 60% (119 – 190 g)…"[17]

It is clear from this graph that, from 1909 to about 1977, carbohydrate consumption was on the decline, and this is a period in which obesity elevated from about 1.2% (in 1900) to about 14% (in 1980). Carbohydrate consumption then climbed again until 1997 before declining again. Of course, obesity rose from 30.5% in 1999 to 42.5% in 2018, once again, while carbohydrate consumption declined (see further detail below).

Thus, for this century-long period of time, we've observed an exponential increase in obesity from 1.2% in 1900 to 42.5% in 2018, a period in which total carbohydrate consumption decreased by about 5%.

Why would this evidence suggest that we should indict carbohydrates per se as a cause of obesity?

Suppose we observe the period between 1961 and 2018 and attempt to draw conclusions regarding carbohydrate and fat consumption (see the following graph, "U.S. Macronutrients vs. Obesity: 1961-2018"). Obesity climbed markedly between 1961 and 1997, while both carbohydrate and fat calories increased.



References: 1) UN Food and Agriculture Organization FAOSTAT food balance sheets: http://www.fao.org/faostat/en/#home. 2) Calculation of Energy Content of Foods, http://www.fao.org/docrep/006/Y5022E/y5022e04.htm. 3) Daily Caloric Supply Derived from Carbohydrates, Protein, and Fat, United States, 1961 to 2013. Our World In Data.org. Retrieved: https://ourworldindata.org/grapher/daily-caloric-supply-derived-from-carbohydrates-protein-and-fat?country=~USA. U.S. © C. Knobbe, 2022. Ancestral Health Foundation. All rights reserved.

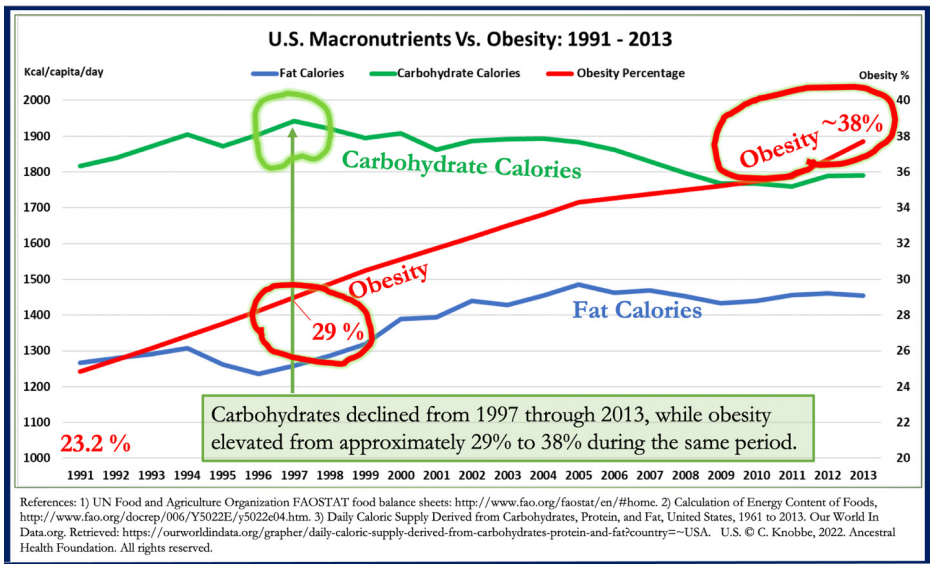
However, then we observe that carbohydrates began to decline after 1997 while fat calories increased as obesity steadily climbed.

Again, if we were to (inappropriately) draw conclusions based solely on these two macronutrient ratios during this time frame, it would make far more sense to blame fat calories for rising obesity than to blame carbohydrate calories.

Again, to be clear, this is not my suggestion. I'm simply trying to make a point.

All of this evidence strongly supports the theory that the type of fat in the diet matters infinitely more, specifically not the amount of fat or carbohydrates or the macronutrient ratios per se.

Let's analyze the period beginning in the late 1990s (see the graph below). We observe that carbohydrate calories declined from 1997 through 2013, while obesity climbed from about 29% to 38% during the same period.



References: 1) UN Food and Agriculture Organization FAOSTAT food balance sheets: http://www.fao.org/faostat/en/#home. 2) Calculation of Energy Content of Foods, http://www.fao.org/docrep/006/Y5022E/y5022e04.htm. 3) Daily Caloric Supply Derived from Carbohydrates, Protein, and Fat, United States, 1961 to 2013. Our World In Data.org. Retrieved: https://ourworldindata.org/grapher/daily-caloric-supply-derived-from-carbohydrates-protein-and-fat?country=~USA. U.S. © C. Knobbe, 2022. Ancestral Health Foundation. All rights reserved.

In review, obesity likely almost steadily elevated from 1900 until about 1983, a period in which carbohydrate consumption was almost entirely on the decline. Then, we observe that carbohydrate consumption again declined after 1997, while obesity again steadily increased.

Total carbohydrate consumption shows no reliable relationship to obesity in the U.S. over 120 years. For the great majority of this period, we have observed declining carbohydrate consumption while obesity increased.

Along with evidence from many other countries and even globally, much of which I review in *The Ancestral Diet Revolution*, I find no evidence to implicate carbohydrates per se causally in obesity.

In *The Ancestral Diet Revolution*, I also make it clear that low-carbohydrate, ketogenic, and zero-carbohydrate diets typically and generally reduce seed oil consumption. The low-carb theory happens to be a classic case of drawing causal inference (blaming carbohydrates) for overweight and obesity because reduction of carbohydrates (the perceived cause) results in weight loss (the effect). However, when reducing carbohydrates, most people unknowingly and unwittingly reduce seed oil consumption (the confounder, in this case), of which they are entirely unaware.

I will assert that this is the true and underlying causal mechanism behind long-term weight loss in reduced carbohydrate diets when they do work, and specifically *not* because the diet is low in carbohydrates. This assertion is unequivocally supported by U.S. food consumption evidence from the NIH because, of the top 15 sources of omega-6 linoleic acid (L.A.) in the diet of Americans, 10 of those are also high-carbohydrate foods.[18]

This association should be (or should become) evident to any shrewd consumer, as seed oils are the clandestine ingredient of processed foods, lying unobserved to most people, hidden in thin films of fat covering portions of pasta, potatoes, rice, grains, and other forms of carbohydrates. It is easy for most people to consume one to three tablespoons (about 13 to 39 grams) in a typical processed food or restaurant meal yet be entirely unaware of the presence of these oils.

Note, for example, in the McDonald's menu items pictured below, that the enormous majority of fat in every single item is made up of vegetable oils. Stated alternatively, the natural animal fat in these items is very low.

It is also important to observe that those consuming a low-carbohydrate diet will most likely, if given some of the above options, eliminate French fries due to the potatoes and Chicken McNuggets due to the breading. Notice, however, that these are enormous sources of vegetable oils, and excluding them, because they are 'high in carbs' will eliminate some of the greatest sources of industrial seed oils.

For example, the McDonald's 20-piece Chicken McNuggets contain 850 calories, 48 g carbohydrates, 50 g fat, and a whopping 20 g of omega-6 polyunsaturated fatty acids (PUFA). The McDonald's Large French Fries contains 480 calories, 65 g carbohydrates, 23 g fat, and 9.2g omega-6 PUFA.[19] [20] If an individual consumed just these two menu items alone, one would consume 29.2 g of omega-6 fat, which is more than two tablespoons of omega-6 linoleic acid (LA) PUFA.

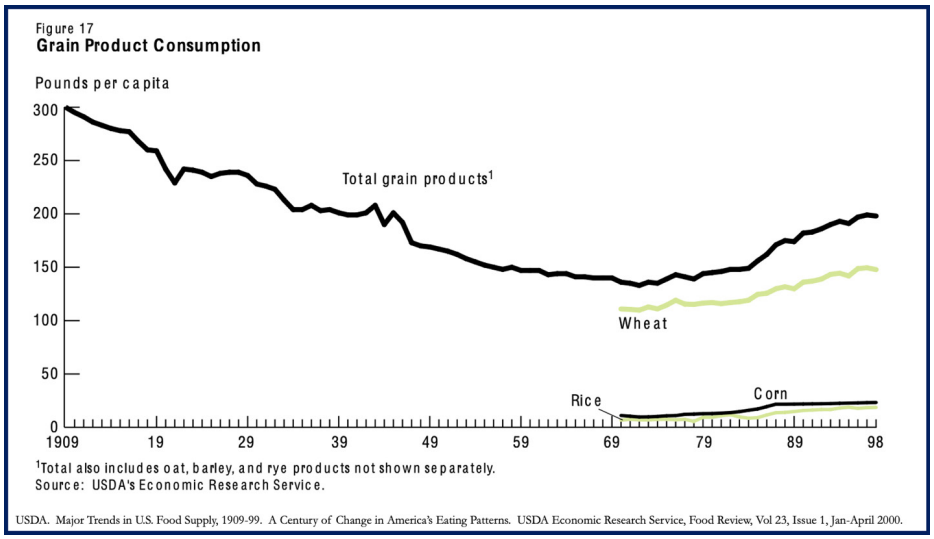
If one chooses to consume the freely provided McDonald's Creamy Ranch Sauce, often used as a "dip" for the McNuggets, this will add another 6.0 g omega-6 PUFA, bringing the total omega-6 PUFA in these three items alone to 35.2 g.

As we will review subsequently, we have calculated that the average omega-6 linoleic acid (L.A., which accounts for ~90% of all omega-6 in the diet) was about 2.2 to 2.6 g/capita/day in the year 1865, before seed oils were available. This is about 1.1% of total calories. By 2008, the average American consumed 29 g omega-6 LA, which is 11.8% of calories. This is a 1,000% increase in omega-6 during this period.

Is it the 'Wheat Belly' Syndrome? Grains & Wheat vs. Obesity

Before we get specifically to the evidence regarding wheat, let's first evaluate total grain consumption. A USDA ERS article entitled "Major Trends in U.S. Food Supply, 1909-99"[21] shows that total grain product consumption dropped precipitously during the 20th century (see graph below). The authors state, "In 1998, Americans consumed 100 pounds less of grain products than in 1909."[21]

Observe the authors' graph, "Grain Product Consumption," below.



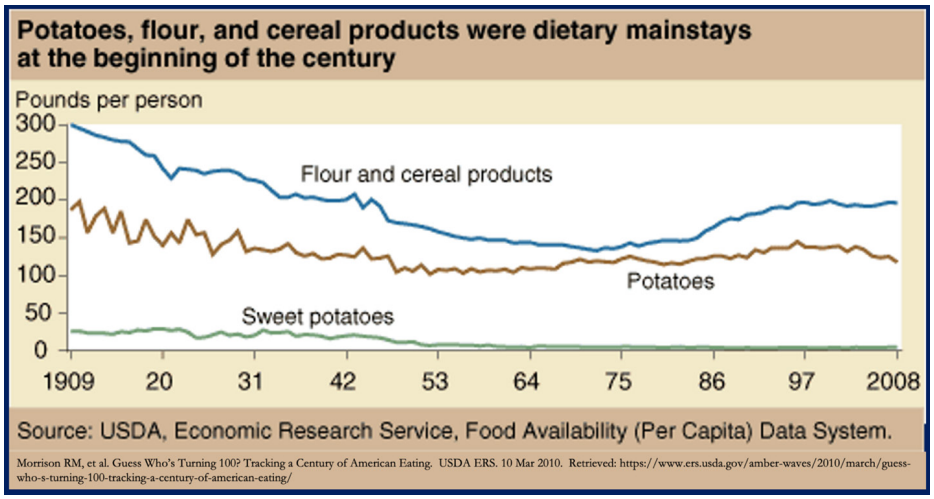
From this graph, we also observe that the enormous bulk of grain consumed in the U.S. is wheat – approximately 70 to 80% between 1970 and 1998 – while corn and rice make up most of the rest, with just trivial amounts of oats, barley, rye, and other grains.

In a USDA article written by R.M. Morrison et al., entitled "Guess Who's Turning 100? Tracking a Century of American Eating,"[22] the authors state,

"During the first half of the 1900s, the most significant changes among food crops were the substantial declines in availability of potatoes, sweet potatoes, and flour and cereal products. Availability of potatoes and sweet potatoes fell from 213.2 pounds per person in 1909 to 114.4 pounds in 1959, while availability of flour and cereal products dropped from 300 to 147 pounds per person. An improved ratio of wages to food prices allowed many to diversify their food spending beyond flour, potatoes, and meats. Greater purchasing power, coupled with increased availability of fresh fruit and vegetables over the winter and growing vitamin consciousness, led Americans to spend a larger portion of their food budgets on milk, cheese, fruit, and vegetables.

"In the second half of the century… Flour and cereal product availability grew as well. Between 1972 and 2008, per capita availability of flour and cereal products increased from a record low 133 pounds per person to 196.5 pounds."[22]

Below is the USDA graph from Morrison et al. showing flour and cereal products, potatoes, and sweet potatoes consumed between 1909 and 2008. It clearly indicates large decreases in all of these carbohydrate-dense categories of foods during this time frame.



Lee et al. (*Frontiers of Nutrition*, *2021*), in their analysis of the ERS data, stated that "From 1909 to 2010, estimated grain (wheat flour, rye flour, rice, corn, oat, and barley products) availability per capita decreased 28% (268 – 194 lbs.)."[17]

Thus, we clearly observe large decreases in grains, including wheat, over the past century, once again, while overweight and obesity have elevated dramatically.

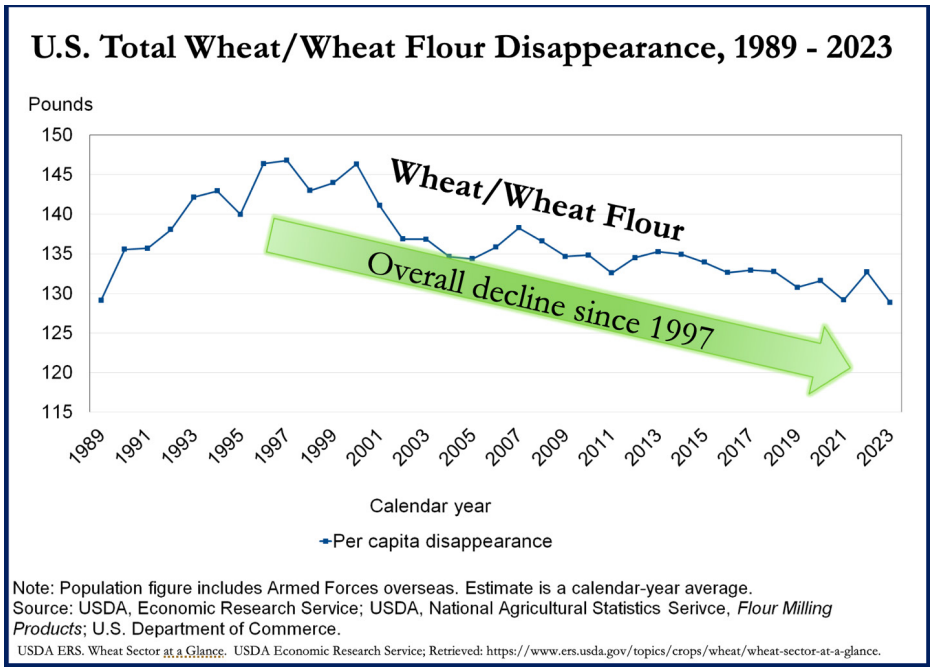
Wheat vs. Obesity: Wheat on the Decline Since 1879 While Obesity Soars

The USDA Economic Research Service, in an article entitled "Wheat Sector at a Glance," reported an enormous decrease in wheat consumption since 1879:[23]

> "For nearly 100 years, per capita wheat consumption declined in the United States as diets diversified to include substitute carbohydrates such as potatoes and rice. Wheat consumption dropped from more than 225 pounds per person in 1879 to 180 pounds in 1925 before bottoming out at 110 pounds in 1972. Per capita consumption began to steadily rise and had rebounded to 147 pounds per capita by 1997.
>
> "The decades-long growth in per capita consumption ended 1997 as changing consumer preferences, led by the adoption of low-carbohydrate diets, again reduced per capita wheat consumption…By 2005, per capita flour consumption had fallen to 134.4 pounds. As interest in low-carbohydrate diets abated, per capita use grew modestly for the next 3 years. Starting in 2008, rising consumer interest in gluten-free foods and renewed promotion of low-carbohydrate diets again put downward pressure on the per capita wheat food-use figure. USDA's Economic Research Service (ERS) estimates per capita wheat flour use at 128.8 pounds in 2023."[23]

From the 1879 consumption value of 225 pounds per person to the 2023 value of 128.8 pounds, we observe that wheat consumption has dropped 43% in this nearly 150-year period.

The graph below, from the USDA Economic Research Service, entitled "U.S. Total Wheat/Wheat Flour Disappearance, 1989-2023," shows that wheat and wheat products, including wheat flour, after a rally of increase from about 1970 through 1997, have been in overall decline since 1997.[23]



Therefore, we have observed large increases in obesity during the 20th century, a period marked by large decreases in consumption of total grains, wheat, wheat flour, cereal products, potatoes, and sweet potatoes, all of which are the primary sources of carbohydrates in the American diet. Hence, the marked decline in carbohydrate consumption until the late 1980s, as observed in the graph, "U.S. Total Caloric and Macronutrient Availability: 1909-2010."

The 'wheat belly' theory is, in part, based upon a purported theory that, mainly during the mid-to-late 20th century, wheat has been markedly hybridized (bred) into a species that barely resembles the ancient version, thereby inducing marked metabolic derangements in consumers. According to the leading authority of this movement, modern wheat has been called "the transformed product of genetic research conducted during the mid-twentieth century."[24]

However, to be clear regarding modernized wheat being a 'transformed product of genetic research,' first of all, there is no genetically modified (G.M.) wheat available in the U.S. for planting or sale,[25] and there is no G.M. wheat approved for cultivation or consumption anywhere in the world.[26]

Furthermore, Levy and Feldman have shown that wheat naturally hybridized itself over about 7 million years, finally deriving its current hexaploid (42 chromosome) variety, known as *Triticum aestivum*, about 9,000 years ago.[27] These researchers also assert that "All of the major evolutionary processes required to produce domesticated tetraploid [28 chromosome] and hexaploid [42 chromosome] wheat were completed by the end of the Pre-Pottery Neolithic period… about 7,500" years ago.[27]

The National Wheat Improvement Committee (NWIC) affirms that all plants, whether wild or cultivated, have undergone natural hybridization over the millennia and that none are the same as they were millions of years ago.[28] They also affirm that the past 70 years of wheat breeding have produced relatively minor changes in the genetic structure of wheat and that there are no new proteins in hybridized wheat that weren't in the parent wheat plants (the "Wheat Belly" author claims that fourteen new gluten proteins were present in a single hybridization experiment. However, this technique was the result of somatic cell fusion hybridization, which is not a process used in any conventional wheat breeding).

Researchers Brouns, van Buul, and Shewry, who have no conflicts of interest, in response to the "Wheat Belly" author's claims regarding "genetically modified" wheat and the induction of "new proteins," published a paper in the journal *Cereal Science*, in which they asserted, "In reality, the presence of such new components is not supported either by comparative studies of old and recently bred types of wheat (Ware et al, 2008) or by analyses of genomic sequences (Brenchley et al., 2012)."[29] They continued, "… there are no grounds to advise the general public not to consume this common dietary staple. Only individuals with a genetic predisposition for celiac disease, or suffering from allergy or other forms of sensitivity to gluten and other wheat proteins, will benefit from excluding wheat and related cereals from their diet."[29]

Furthermore, the NWIC has reviewed the following facts: 1) Gluten and gliadin have always been significant components of wheat, B) increasing celiac disease over the past 50 years has paralleled the increase in all autoimmune diseases during that period, and 3) there is no clinical evidence that gliadin stimulates appetite or increases consumption.[28] Also, according to the WHO, in 2010, there was no history of correlation between a country's wheat consumption and obesity prevalence.[28]

Overall, wheat consumption has been negatively correlated with obesity. Since the late 19th century, wheat consumption has mostly been on the decline, while obesity continues to break records of increasing prevalence.

Wheat consumption, at least at the population level, appears to have no effect on obesity development. I wholeheartedly agree with researchers Brouns, van Buul, and Shewry, who wrote, "No data justifies a negative opinion about whole-wheat products in a healthy population."[29]

Are Processed Carbohydrates Driving Obesity?

Many in the field of Nutrition assert that, while carbohydrates, wheat, and grains per se are not the culprits in obesity and diabetes, processed carbohydrates are. Is this a tenable hypothesis? Let's examine it further.

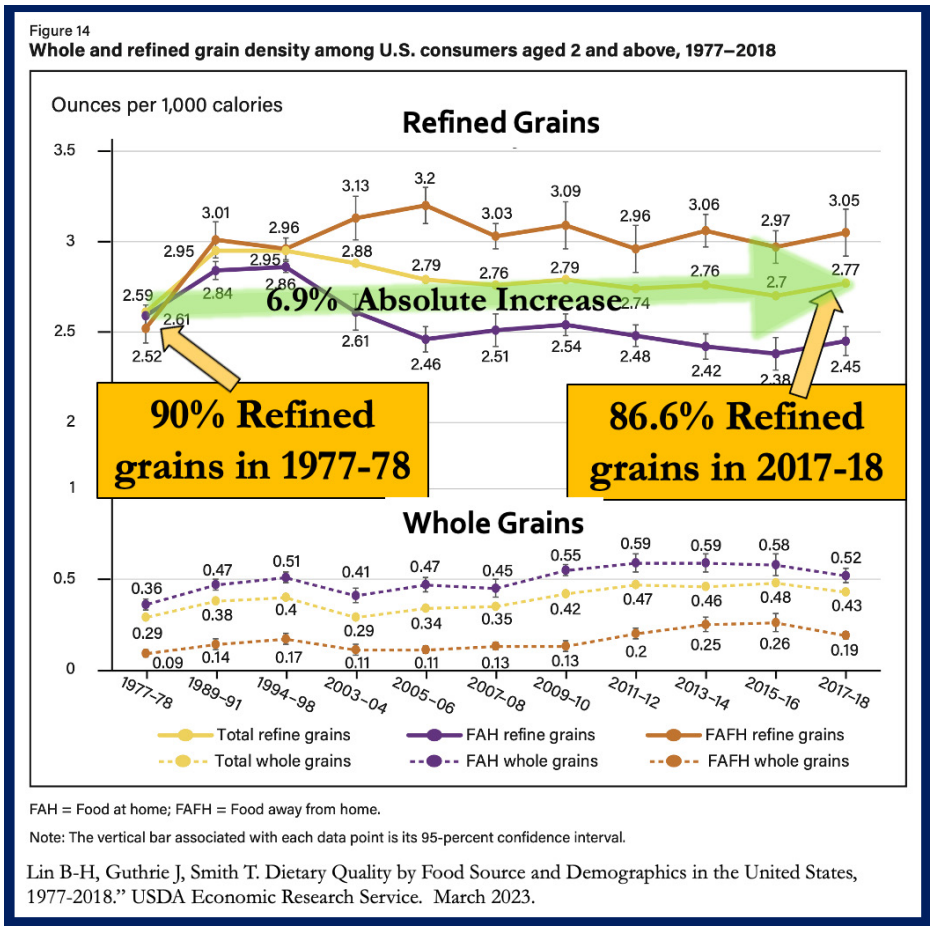
Processed carbohydrates consist almost exclusively of two primary components: 1) Refined (added) sugars and 2) refined grain flour (approximately 70% to 80% wheat flour in the U.S. in recent decades).

We've already observed that added sugars have a very poor correlation to obesity (more on this subsequently), and we've noted that obesity elevated markedly after 1999 when total sugar consumption, which includes high-fructose corn syrup (HFCS), was on the decline.

So what about refined flours? First, we know that roller-mill technology, which may provide refined white wheat flour, was introduced for the first time in 1880. To my knowledge, the proportion of flour that was refined between 1880 and 1977 is not precisely known. In 1977, however, the USDA ERS began accurately tracking refined flours versus whole grains.

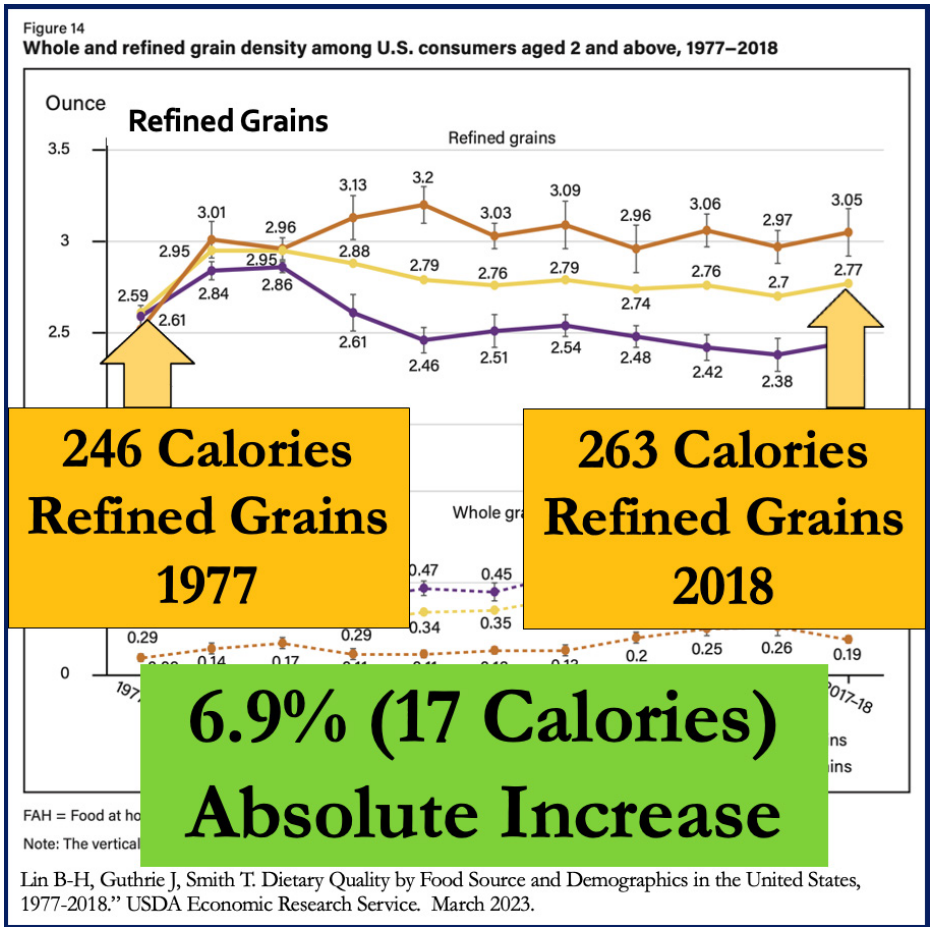
Because obesity increased vastly and rapidly, from 14% to 42.5%, between 1980 and 2018, it is critically important to further examine processed carbohydrates during this time frame.

The USDA ERS published a review entitled "Dietary Quality by Food Source and Demographics in the United States, 1977-2018,"[30] which included the following graph (graphic inserts added):



From the graph above, notice that refined grain consumption was practically identical between 1977-78 and 2017-18. It was 2.59 ounces per 1,000 calories in 1977 and 2.77 ounces per 1,000 calories in 2017. The increase in refined grains consumed between 1977-78 and 2017-18 is 0.18 ounces per 1,000 calories, or an increase of just 6.9%.

Simple calculations show that, in 1977, Americans consumed 246 calories worth of refined grains, which increased to 263 calories in 2018, an increase of just 17 calories (or 6.9%). See the following graph for details.



The consumption of whole grains increased by 48% between 1977-78 and 2017-18. However, the total consumption of whole grains at all points during this 40-year time frame is very low and never exceeds 0.48 ounces per 1,000 calories per day. Thus, over these 40 years (1977-2017), Americans have consumed only about 0.60 to 1.0 ounces of whole grains (about 60 to 100 calories) per capita per day.

Even as recently as 2015-2016, NHANES data showed that ready-to-eat (RTE) cereal contributed just 10% of total energy intake (214 cal/cap/day) in adults aged 18 years or more who consume cereal.[17] However, when examining the entire population, i.e., both cereal and non-cereal consumers, the total energy intake for RTE cereal is only 2% of caloric energy (42 cal/cap/day out of 2,102 cal/cap/day). Thus, the contribution of RTE cereal grains to the overall diet is very, very small.

The evidence that grains, including wheat, whether processed or not, *are not* a significant driver of the obesity and diabetes epidemics may be summarized as follows:

A) Total grain consumption dropped 28% between 1909 and 2010, while obesity elevated from about 1.2% in 1900 to about 38% in 2010.

B) The percentage of refined grains was practically identical between 1977-78 and 2017-18, at 90% for the former and 86.6% for the latter.

C) The absolute consumption of refined grains was also practically identical between 1977-78 and 2017-18, being 2.59 ounces per 1000 calories for the former and 2.77 ounces per 1000 calories for the latter, an increase of just 6.9%.

D) The above numbers indicate that refined grains consumption was 246 calories/day in 1977 and 263 calories/day in 2018, an increase of 17 calories (6.9%).

E) Ready-to-eat (RTE) cereal consumption accounted for only 2% (42 cal/capita/day) of total calorie consumption in the U.S. in 2015-2016, thus making it a very minor contributor to the overall diet.

F) Total grain consumption (both refined and whole grains) was 2.88 ounces per 1000 calories in 1977-78 and 3.2 ounces per 1000 calories in 2017-18, which is a difference of 0.32 ounces per 1000 calories. This amounts to an increase of about 18 grams (~61 calories) of grains per day during this 40-year period.

G) Wheat consumption between 1997 and 2023 declined about 12 percent, while obesity increased from 29% to 42.5%.

H) Finally, we observe that obesity steadily climbed from about 14% in 1977 to 42.5% in 2018, a period in which we've documented only a small increase (about 61 calories/day) in total grain consumption and a trivial increase (17 calories, or 6.9%) in refined grains.

Finally, to answer the question as to whether processed carbohydrates might be the driver of obesity, we find that total added sugar consumption was 480 cal/day in 1977, which increased to 522 cal/day by 2017, a total increase of 42 calories/day worth of sugar. We already reviewed that processed (refined) grains increased from 246 to 263 calories between 1977 and 2017, an absolute increase of just 17 calories/day.

Thus, total processed (refined) carbohydrate consumption was 726 calories in 1977 and 785 calories in 2017.

The total increase in refined carbohydrates between 1977 and 2017-18 is 59 calories, which is an 8% increase (see table below, "Processed Carbohydrates: 1977 vs. 2017."

Processed Carbohydrates: 1977 vs. 2017

Year	1977	2017
Sugar Consumption (Kcal)	480	522
Refined Grains (Kcal)	246	263
Total Processed Carbohydrates (Kcal)	726	785
Increase in Processed Carbs Relative to 1977 (Kcal) (% in Parentheses)	N/A	59 Calories (8% Increase)

During this same time frame (1977 – 2018), vegetable oils increased by 272 calories, or 59%.

Refined sugar and grains are processed, nutrient-deficient foods that have changed little (8%) over the past 40 years.

Vegetable oils are chronic metabolic biological poisons, which increased markedly (59%) – 7-fold more than refined sugar and grains combined – during the past 40 years.

Neither of these food groups (refined carbohydrates or vegetable oils) increased total caloric consumption after 2004, while obesity elevated markedly.

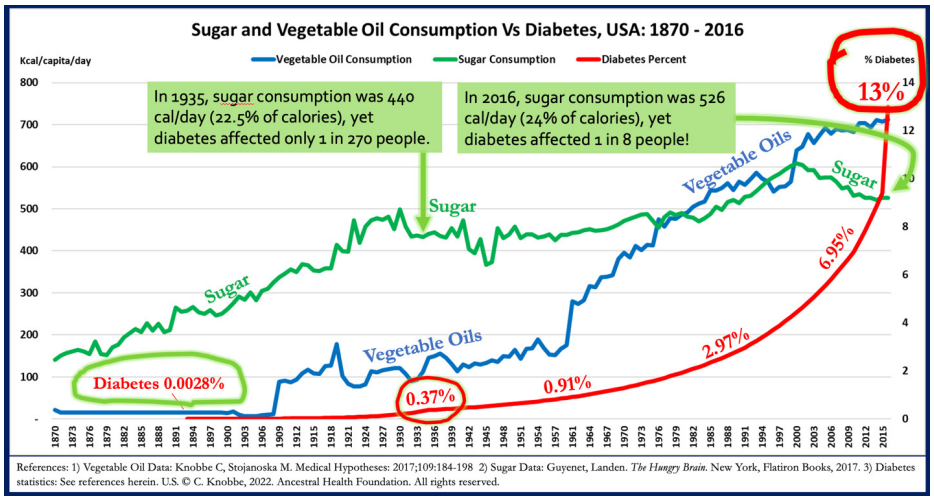
Thus, we cannot assert that we consumed an *additional* 59 calories per day of refined carbohydrates on top of our usual diet, nor did we consume an *additional* 272 calories worth of vegetable oils on top of our usual diet.

The changes in our diet that are far and away the most important are qualitative, not quantitative.

The evidence of culpability seems obvious, doesn't it?

Sugar and Vegetable Oils vs. Diabetes

Next, observe the graph below, "Sugar and Vegetable Oil Consumption vs. Diabetes, USA: 1870-2016."



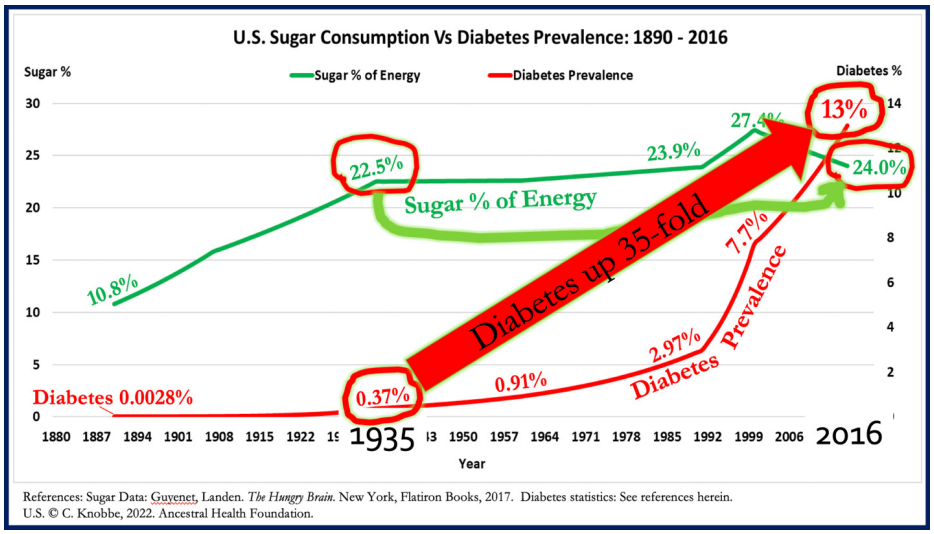
First, note that, as published by Sir William Osler in his classic medical textbook, *Principles and Practice of Medicine* (1892), in 1890, diabetes affected only 2.8 per 100,000 people in the U.S. That is 0.0028% of the population, or approximately 1 in 36,000 people. At that time, vegetable oil consumption in the U.S. was only about 16 calories (1.5 grams) per person per day.

Diabetes prevalence then elevated to 0.37% by 1935, which is an increase of 132-fold from the 1890 prevalence. However, vegetable oils had risen to 146 calories (16 grams) per day. This trend continued, with diabetes prevalence rising to 0.91% by 1961, 2.97% by 1991, 7.7% by 2000, and finally, to 13.0% by 2016.

By 2016, vegetable oil consumption was 713 calories (79 grams) per day.

In 1935, sugar consumption had already risen to 440 cal/day, which was 22.5% of total calories, yet diabetes affected only 0.37% of the population, or 1 in 270 people.

By 2016, sugar had risen only mildly from the 1935 level to 526 calories/day, which was 24% of calories. This is just 86 calories more than the level of consumption in 1935. Yet, diabetes affected 13.0% of the population, or 1 in 8 people. In brief, diabetes had risen 3,400% (35-fold) from the 1935 prevalence, while absolute sugar consumption had increased just 86 cal/day and just 1.5% higher as an absolute percentage of calories consumed. See the following graph, "U.S. Sugar Consumption vs Diabetes Prevalence: 1890 – 2016."

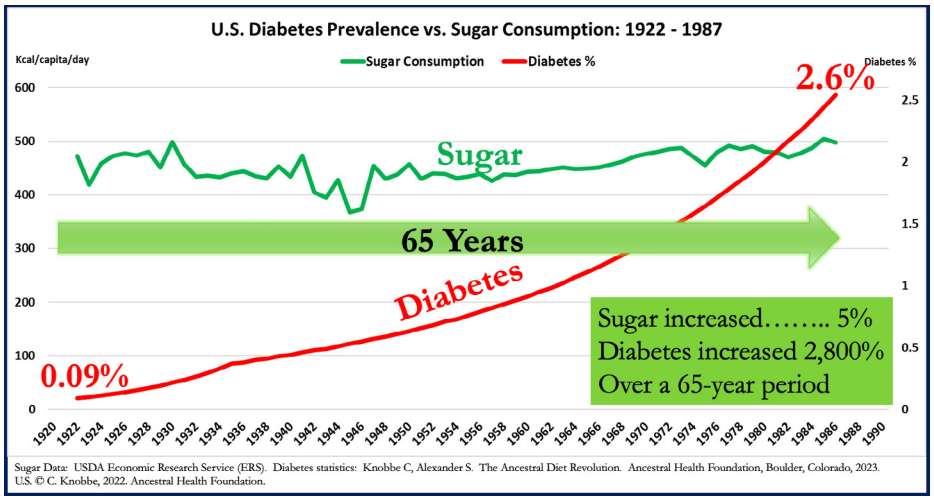


Need I ask? Are we to blame sugar for the cause of diabetes when diabetes prevalence increased 35-fold in response to an absolute sugar consumption increase of just 1.5%? This would be an absurdly illogical conclusion.

However, during this same time frame (1935 to 2016), vegetable oil consumption elevated from 146 cal/day (16.2 g) to 713 cal/day (79.2 g), which is a 4.9-fold increase in vegetable oils, whether measured by caloric content or mass. Furthermore, vegetable oils accounted for 7.5% of total calories consumed in 1935, but that number increased to 29.0% of calories consumed in 2016, which is a 21.5% absolute percentage of energy increase.

Put another way, between 1935 and 2016 alone, more than one-fifth of all calories consumed in the American diet were supplanted and replaced by vegetable oils.

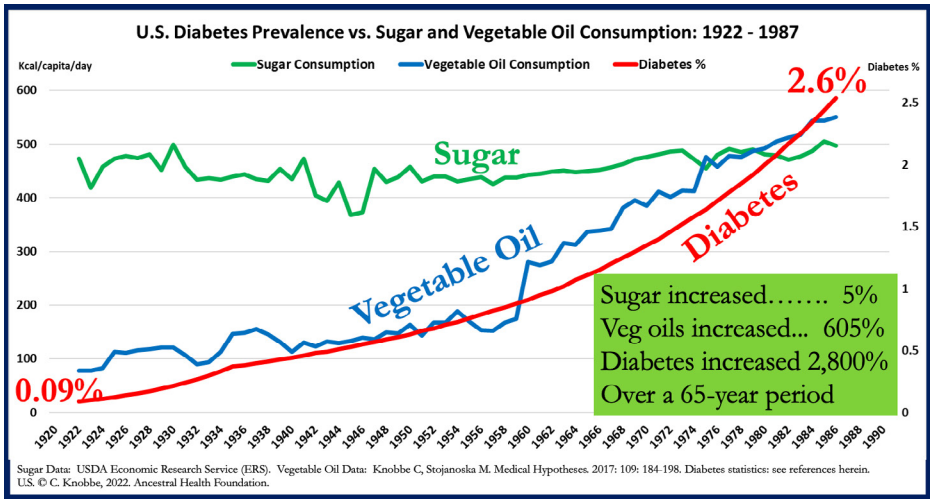
Next, we will focus on the period between 1922 and 1987, a 65-year period in which sugar consumption was virtually flat.



While observing the graph above, "U.S. Diabetes Prevalence vs. Sugar Consumption: 1922-1987," note that sugar consumption in this 65-year period elevated by just 5%, but diabetes prevalence increased from 0.09% in 1922 to 2.6% in 1987, which is a 29-fold or 2,800% increase.

Once again, how can we postulate that sugar is to blame as the cause of diabetes? Such a conclusion seems absurdly illogical and ignores virtually all of the Bradford Hill criteria in establishing causal inference. There is no strength of association (correlation), temporality, biological gradient (dose-response effect), coherence, etc.

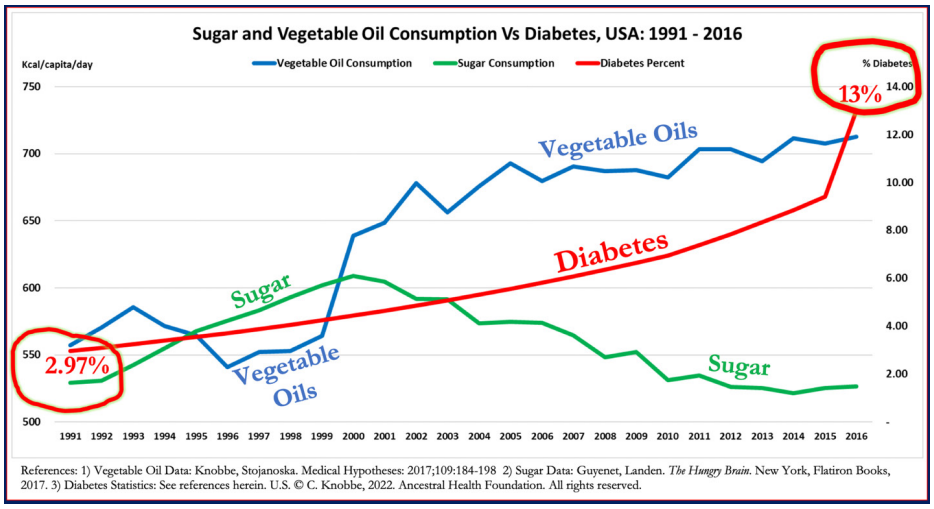
However, let's examine both sugar and vegetable oil in the same graph. We observe a striking correlation between vegetable oil consumption and diabetes prevalence (see the graph below, "U.S. Diabetes Prevalence vs. Sugar and Vegetable Oil Consumption: 1922-1987").



Note from the above graph that from 1922 to 1987, diabetes prevalence elevated 29-fold (2,800%), from 0.09% to 2.6%. During this period, sugar consumption increased 5%, vegetable oils increased 605%, and diabetes increased 2,800%.

Is there any question as to where the correlation is?

In the next graph, we've narrowed the timeline to the very recent period of 1991 to 2016 (see the graph entitled "Sugar and Vegetable Oil Consumption vs Diabetes, USA: 1991 – 2016").



In this graph, we observe that diabetes increased in prevalence from 2.97% in 1991 to 13.0% in 2016, which is an increase of 4.4-fold. During this same time frame, sugar consumption was 529 cal/day in 1991, peaked at 609 cal/day in 2000, and then dropped down to 526 cal/day by 2016. Total vegetable oil consumption was 557 cal/day in 1991 and increased to 713 cal/day by 2016.

Just as with obesity, diabetes continued an unmitigated ascent after 2000 while sugar consumption was on the decline. In this case, we have a steady decline and a total 14% drop in sugar consumption over a 16-year period (2000 to 2016) while diabetes steadily increased. This, of course, is a negative correlation between diabetes prevalence and sugar consumption.

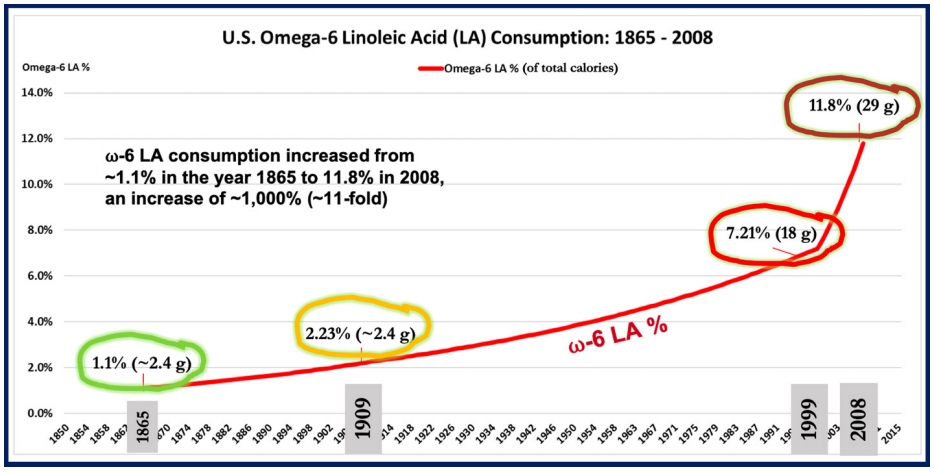
Once again, if sugar consumption is on the decline while diabetes prevalence rises, shouldn't that strongly suggest that sugar is not the driving cause?

Omega-6 Apocalypse:

The True Underlying Cause of Obesity and Diabetes

We modeled the typical diet of Americans in 1865, before seed oils were available, without any nuts or seeds (typical of ancestral diets) and based upon consumption of animals (and animal products, such as eggs) that were also consuming ancestral diets themselves (e.g., no corn or soy feed which raises omega-6 levels markedly in monogastric animals such as chickens and pigs). We determined that the average omega-6 L.A. consumption at that time in history was approximately 2.2 to 2.6 g/capita/day (average 2.4 g L.A./day), which is about 1.1% of calories (data pending publication).

In 2008, the average American consumed 29 g of omega-6 L.A. per day, which was 11.8% of calories. This is an 11-fold or 1,000% increase in omega-6 L.A. consumption between 1865 and 2008. See the following graph, "U.S. Omega-6 Linoleic Acid (L.A.) Consumption: 1865-2008."



A full review of the biological mechanisms via which seed oils and high omega-6 diets drive obesity, diabetes, and chronic disease is beyond the scope of this review. However, this is covered in great detail in my latest book, *The Ancestral Diet Revolution*, along with much other evidence on how and why ancestral diets prevent and treat obesity and chronic disease.

Regression Analysis:

Another "Nail in the Coffin" of Sugar Causality Theorists

Perhaps, even after all of this evidence, you're still not quite convinced that industrial seed oils could be such a prominent driver of obesity and diabetes. Maybe graph reading is just not your forte, or you just cannot accept my argument that there is an undeniable and striking correlation between obesity, diabetes, and vegetable oils and an obviously very poor correlation (even negative) between sugar consumption, obesity, and diabetes.

Not to worry. We've dug for even more convincing evidence. We took all of our previously published data (as observed in the above graphs) and consulted an outside data analyst—that's right—a professor of data analytics, whom we consulted for an independent review.

Eugene Antipov, PhD, of Dubai, United Arab Emirates, is a University Professor of Statistics and Data Analytics, holding a PhD and an MSc in Quantitative Research Methods, and he has published many research papers on statistics, marketing analytics, and data analysis in peer-reviewed journals.

We provided Professor Antipov with our previously published data on vegetable oil and sugar consumption, as well as obesity and diabetes prevalence data in the U.S., and asked him one fundamental statistical question:

"Using the most appropriate statistical analyses for data in the U.S., if we assume that one or both factors, that is, sugar and vegetable oils, are the primary etiologic agents in obesity and diabetes, what is the effect of each of these two factors on these disease conditions?"

Following a complete statistical analysis and multiple regression analyses, Professor Antipov's review unequivocally states:

> **" It turned out that in such a two-factor linear regression model, there is no significant effect of sugar consumption on the prevalence of obesity and diabetes at all.**
>
> **Therefore, in a model assuming that two factors (vegetable oil and sugar) are the primary causal factors, sugar consumption does not add significantly to explaining the variance in diabetes or obesity prevalence (P>0.01).**
>
> **At the same time, vegetable oil consumption's positive effect is significant at any imaginable significance level commonly used in scientific literature (P<0.0001)."**

This is a striking analysis and statement. What was so blatantly obvious to me and many other researchers in our group had now been put to the scientific rigors of a data scientist. Now, the obvious answer was also backed by mathematical precision.

And given that most of us are not data scientists, I'll spare us all from Professor Antipov's extensive review and numerous graphical regression analyses scatterplots, which clearly show strongly positive, curvilinear, and systematic correlations between increasing vegetable oil consumption and the prevalence of obesity and diabetes.

On the other hand, there were persistently non-linear, non-systematic, and non-monotonous relationships between sugar consumption and the prevalence of obesity and diabetes. For example, in the U.S., we observe sugar consumption climbing markedly before 1922 with little change in obesity, almost level consumption for 65 years with exponential increases in obesity, and finally, large decreases after 1999 while obesity continued to increase.

Does this leave any question regarding causality, at least with regard to seed oils versus sugar?

But We're All Just Exercising Less, Correct?

No matter how much evidence I present in country after country and population after population, someone always claims, 'You can't prove it's the seed oils. The problem is we're just lazy and don't exercise as much as we used to.'

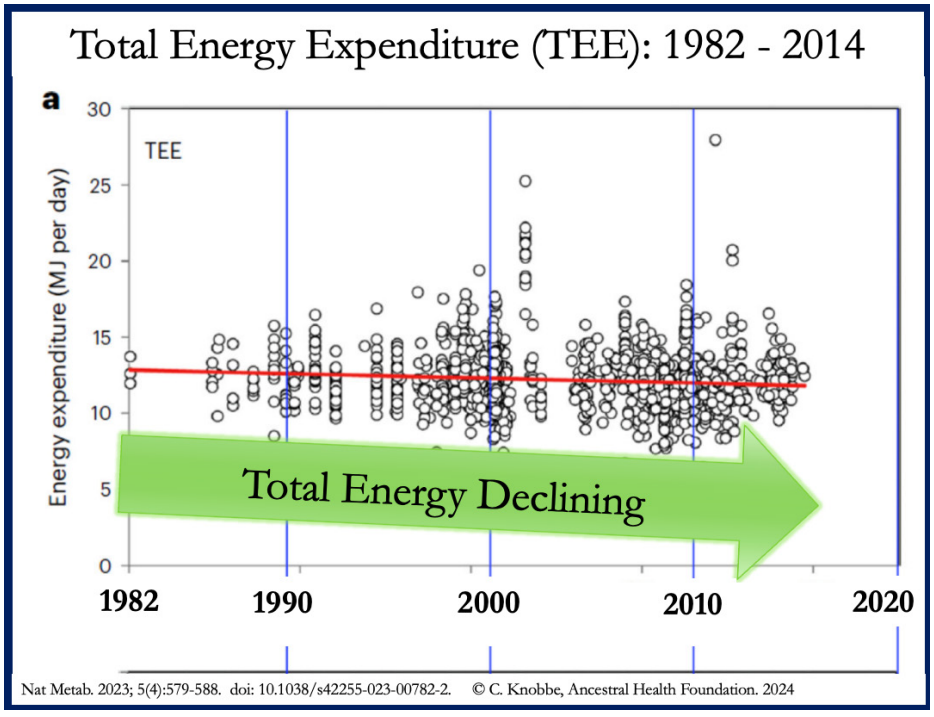
Can we answer this claim with analytical precision and real-world data?

Due to the brilliant work of Professor John Speakman and numerous colleagues, we finally have an answer to this age-old question. Speakman and colleagues published a paper last year (2023) in the journal *Nature Metabolism* entitled, "Total daily energy expenditure has declined over the past three decades due to declining basal expenditure, not reduced activity expenditure."[31]

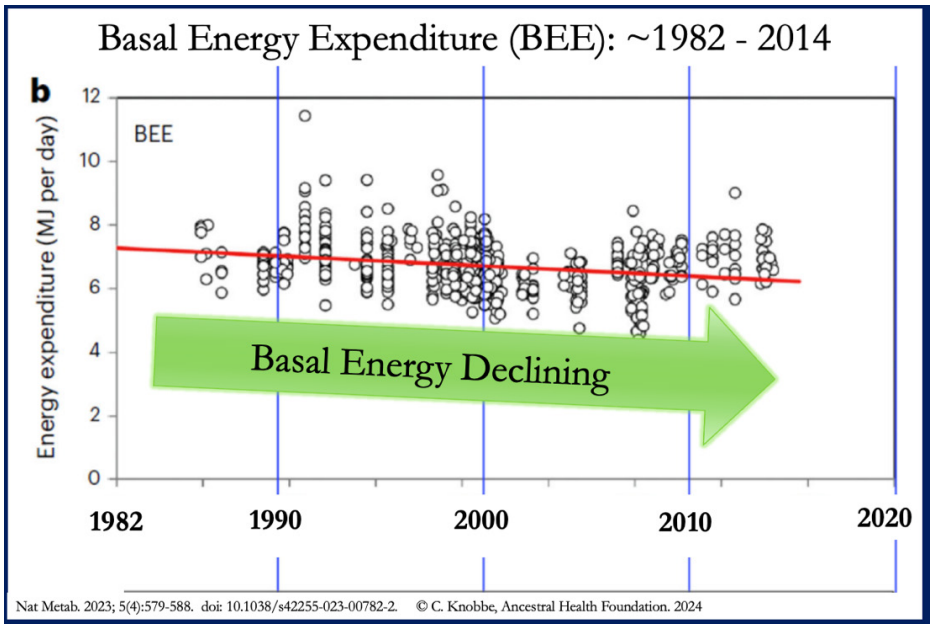
The title of this paper says it all. Speakman et al. analyzed data on total energy expenditure (TEE), basal energy expenditure (BEE), and physical activity energy expenditure (AEE) from 1981 until about 2017. They also examined a larger dataset of basal metabolic rate (BMR, equivalent to BEE) of nearly 10,000 subjects across 163 studies spanning a century-long period from about 1919 to 2020.

In this study, for the TEE, BEE, and AEE analyses between 1981 and 2017, the researchers only included data from U.S. and European studies using the gold standard technique of doubly labeled water (DLW).[32] All data was drawn only from the International Atomic Energy Agency (IAEA) database, assuring data integrity and analyses of the absolute highest degree.

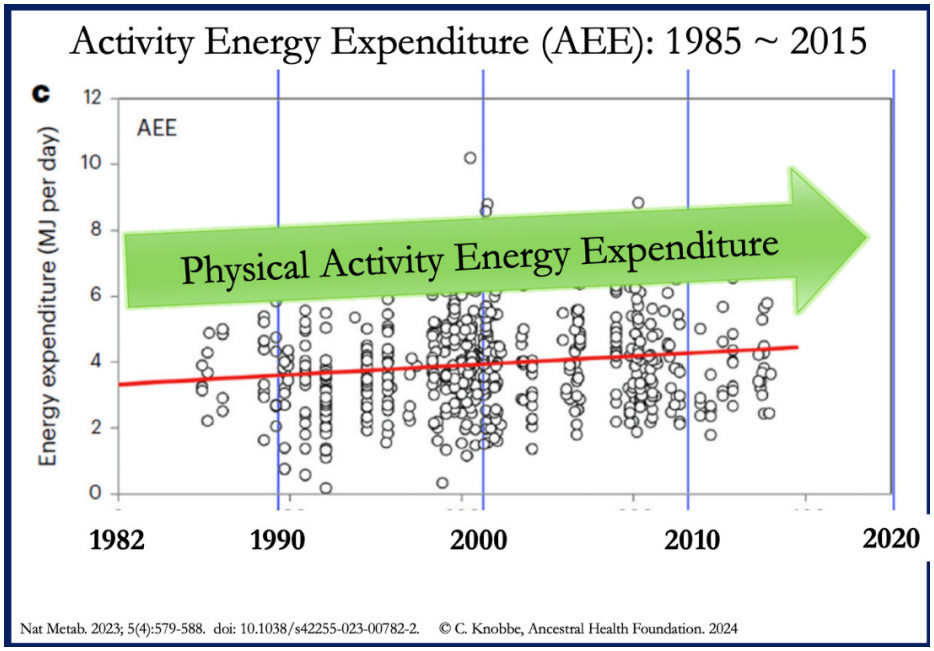
The findings? First, the researchers found that TEE did indeed decline between about 1982 and 2014 (see the graph, "Total Energy Expenditure (TEE): 1982 – 2014"). The TEE is a combination of BEE and AEE. Therefore, without knowing the individual components specifically, we don't know whether our basal metabolism is down, our activity energy expenditure is down, or both.



Speakman et al. then determined that Basal Energy Expenditure (BEE) from 1982 to 2014 also declined. This evidence indicates that we're burning fewer calories at rest for some reason (see the following graph, "Basal Energy Expenditure (BEE): ~1982-2014").



The above analysis indicates that our total energy expenditure (TEE) has decreased over the past 30 years. Also, we're burning fewer calories at rest, i.e., our basal energy expenditure (BEE) is down. But are we burning fewer calories because our activity is also down? Please examine the next graph, "Activity Energy Expenditure (AEE): 1985 – 2015."

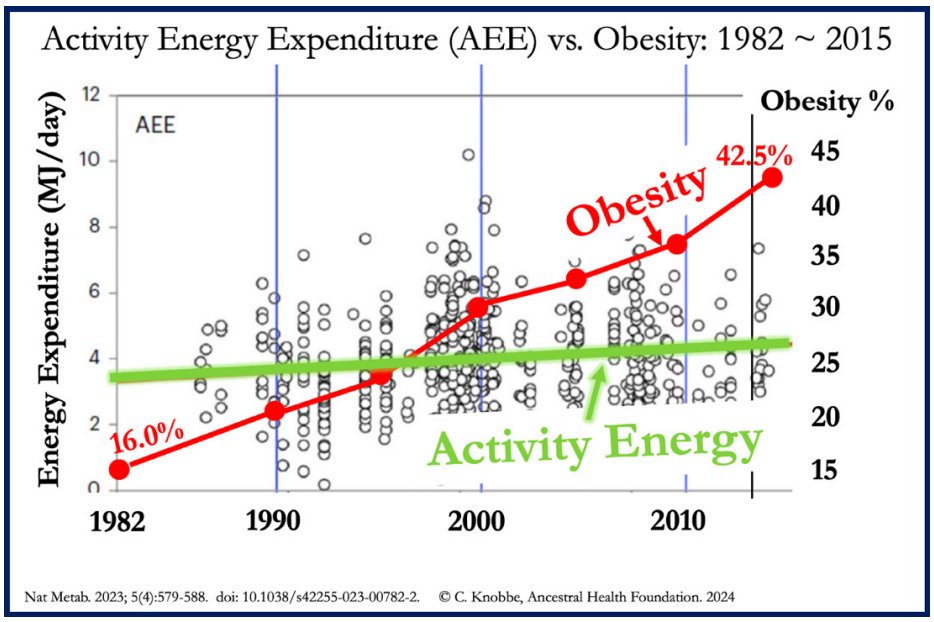


The above graph and evidence came as a surprise to Professor John Speakman and many of his colleagues. The evidence, however, was undeniable and irrefutable. In their own words, "Overall our data show that there has been a significant reduction in adjusted TEE over the last three decades, which can be traced to a decline in BEE rather than any reduction in AEE linked to declining physical activity levels. Indeed, our data show that AEE has significantly increased over time."[31]

Based on their own mouse studies and broad dietary evidence, Speakman and colleagues proffered only one possible causal explanation in the conclusion segment of their paper: "…one of the many possible explanations is decreases in the intake of saturated relative to unsaturated fatty acids."[31]

Speakman and colleagues suggested that an increase in polyunsaturated fatty acids, a decrease in saturated fatty acids, or both might be the most likely cause. Clearly, this suggestion is related almost entirely to increased seed oil consumption.

I've plotted the AEE graph from the paper vs. obesity in the U.S. between about 1982 and 2015 (note: the final obesity data is from 2018). See the following graph, "Activity Energy Expenditure (AEE) vs. Obesity: 1982 ~ 2015."

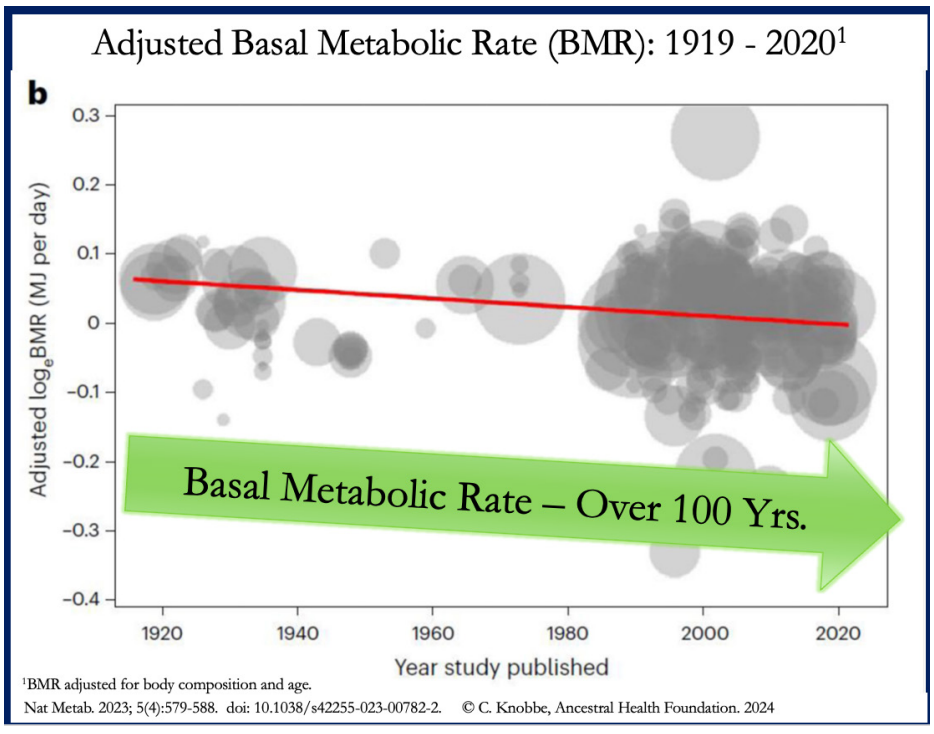


The greatest increase in obesity in American history, rising from about 16.0% to 42.5% over a period of about 36 years, occurred precisely while activity energy expenditure (AEE) progressively *increased*.

The fact that increasing activity levels fail to prevent overweight and obesity comes as no surprise to me. If you carefully observe the world around you, it is blatantly obvious. For example, I see the hardest working physical laborers, such as landscapers and carpenters, who work at physically exhaustive occupations, yet many of them are overweight or even obese.

Jack LaLanne opened the first fitness club in the U.S. in 1936 in Oakland, California.[33] In 2023, 113,326 gyms, health clubs, and fitness clubs were operating in the U.S.[34] People are exercising more and more while generally gaining more undesirable weight.

Professor Speakman et al. provided yet another analysis that speaks volumes on this subject. They plotted the adjusted basal metabolic rate (BMR), adjusted for body mass, age, and sex, over the last 100 years (~1919 to 2020). See the graph below, entitled "Adjusted Basal Metabolic Rate (BMR): 1919 – 2020." The BMR is equivalent to the BEE.



Speakman et al. determined that, even after adjusting for body mass, age, and sex, the BMR has declined by approximately 0.34 MJ/d over the last 100 years.

This is indicative of our metabolic inefficiency, i.e., our declining basal metabolic rate, which generally cannot overcome increased physical activity.

As I have declared since 2016, seed oils drive metabolic destruction, partly through mitochondrial dysfunction.

Detractors of the Seed Oil Hypothesis of Obesity and Chronic Disease:

"Correlation is Not Causation!"

Detractors of the "seed oil hypothesis" of obesity and chronic disease are often quick to dismissively quip, " Correlation is not causation," as though, with such Statistics 101 rhetoric, they've won the argument.

Such a statement, albeit true, might indicate a rudimentary, incomplete, and inchoate understanding of the immense significance of correlation in establishing causation. On the other hand, it might also suggest that one is chafed by statistical correlational evidence that does not fit their belief systems, agenda, rhetoric, or even non-science-based propaganda.

Tobacco industry executives argued for decades that cigarette smoking is not a cause of lung cancer. What was their primary argument? "Correlation is not causation."[35]

In general, the approximate relative risk of lung cancer development is more than 20-fold higher for smokers than for lifelong non-smokers.[36] In congressional hearings, tobacco industry executives argued that the increase in lung cancer in smokers as compared to non-smokers was "just correlation" and didn't prove causation.[35]

In the written words of Robert L. Rabin, Professor of Law at Stanford University, the "correlation is not causation" argument upheld by Big Tobacco "became an albatross, not just because of the voluminous epidemiological findings, but also because of the hypocrisy revealed in the documents."[35]

In scientific data analytics, correlation can be argued to be the single most crucial element in establishing causal inference.[37] If it weren't, why would the greater part of an entire branch of science — statistics — be based largely on this single mathematical concept? Indeed, correlational analyses are one of the most, if not the most, commonly utilized data analytic techniques in health and epidemiological research today.[38]

In 1965, Sir Austin Bradford Hill (1897 – 1991) proposed nine criteria that remain the fundamental tenets of causal inference in observational epidemiology today.[39] The first of these nine criteria is "strength of association," which is measured by the correlation coefficient in the science of statistics. That's correct. Correlation was the first and perhaps most important fundamental measure in Bradford Hill's criteria.

In Hill's 1965 paper, in regard to establishing causation, he wrote:

"Disregarding then any such problem in semantics we have this situation. Our observations reveal an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance. What aspects of that association should we especially consider before deciding that the most likely interpretation of it is causation? ... First upon my list I would put the strength of the association."[39]

"Strength of the association" is an alternate way of describing the strength of correlation, or, mathematically, the correlation coefficient. Of the nine criteria that Bradford Hill presented, he appears to have considered the strength of correlation to be the most important.

In this seminal paper, Hill went on to describe not only the 200-fold increased mortality of chimney sweeps in the eighteenth century but an 8 to 32-fold increased risk of lung cancer in smokers versus non-smokers and a 14-fold greater death rate from cholera for those supplied with sewage-contaminated water as compared to sewage-free water.[39]

Today, many authors and researchers attempt to convince us by arguing first that the metabolic mechanism of their theory is established, e.g., 'carbohydrates drive insulin and insulin drives fat,' the so-called "carbohydrate-insulin model" (CIM) of obesity.[16] The CIM model or theory then leads to their conclusion, for example, 'carbohydrates are dangerous,' and, of course, the prescribed treatment – a low carbohydrate diet. Sugar, of course, is a prime suspect and key driver for those who ascribe to this CIM model of obesity.[40]

Proposing a physiological or pathophysiological mechanism for a hypothesis is what Bradford Hill listed as his sixth criterion, and he called this "plausibility" or "biological plausibility."[39]

While establishing a biologically plausible mechanism helps to further support a proposed hypothesis, it is not a necessary criterion to infer causality. Percival Pott couldn't possibly have developed a deeply honed biological theory of the mechanism by which chimney sweeps had 200-fold greater mortality from scrotal cancer. But this didn't dissuade him from drawing conclusions.

To Pott, the strength of the correlation spoke volumes, which is only logical.

However, while correlation does not imply causation, one must fully understand that causation *always* implies correlation.[41]

Always.

This does not mean that if causation in a relationship between two variables is established, the correlation is perfect, e.g., a coefficient of +1.0 (a perfect positive correlation) or -1.0 (a perfect negative correlation). For example, if 100 people are sufficiently exposed to a cold virus by the same dose of inoculant in the nasal passages, perhaps only 20% develop a cold. The cause – in this case, the cold virus – had to be present 100% of the time to induce the disease, even though the disease was only induced 20% of the time.

According to mathematician and data analyst Mathias Jesussek, PhD, and his colleague, quantitative and qualitative research analyst Hannah Volk-Jessusek, PhD, there are multiple prerequisites for establishing causal inference. First, "there must be a significant relationship, i.e., a significant correlation," second, a temporally ordered sequence of the variables (cause must precede effect), and a "theoretically justified and plausible theory" regarding which direction the causal relationship must take.[42] Note that both affirm that the first and foremost prerequisite to establishing causal inference is "a significant correlation."

Dismissing a correlation between a potential cause and effect, particularly one that occurs commonly and repeatedly, dismisses a staggeringly large body of critical scientific evidence. Most scientific theory is based, first and foremost, on the correlation of two or more variables. The fact that two variables tend to occur together and move in the same or opposite directions is often the first determinant of developing a hypothesis.

Causal inference is the methodology that attempts to answer causal research questions using a combination of data and theoretical assumptions. A fair amount of data and correlations between variables have been presented in this writing. Many, if not most, scientific papers and texts are supported by data and correlations. But none of us are interested in correlation for its own sake. We're interested in establishing causation. Causation is established if one can determine that one variable (the independent variable) has a clear effect on a second variable (the dependent variable).

Isn't this what we've observed with vegetable oils, obesity, and diabetes?

Daniel Dvorkin, PhD, Bioinformatics and Biostatistics consultant, University of Colorado School of Medicine, states, "Correlation is almost always the first clue to causality: if it seems like most of the time when A happens, B happens too, then that's a good reason to suspect that A might cause B, or vice versa. Making an argument for causality without observing correlation first isn't quite impossible, but it's a lot more difficult."[43]

Unfortunately, causation cannot be calculated by any equation, statistical analysis, or even artificial intelligence. Instead, we must infer it from the correlations and other bodies of evidence, both observational and experimental.[37] Thus, correlation *is almost always necessary*, albeit insufficient, to establish causation.

Computers, statistical analyses, machine learning, and even artificial intelligence (AI) cannot reliably establish causation.[43] Only the human brain can do that. In the words of Judea Pearl, PhD, UCLA Samueli School of Engineering professor and author of *The Book of Why*, "…you are smarter than your data. Data do not understand causes and effects; humans do."[44]

The point of this discussion is that correlation is exceedingly important. It should generally be considered the single most critical factor in establishing causal inference.

Discussion

Arthur Conan Doyle's famous fictional detective, Sherlock Holmes, aptly said, "When you have eliminated the impossible, whatever remains, however improbable, must be the truth."[45] Holmes would induce hypotheses based on the details of a crime and then eliminate them one by one until only a single hypothesis remained, which was generally the correct one. Holmes deduced the correct hypothesis through a process of elimination.

When a homicide is committed, the first thing that a highly trained and thorough investigator does is develop a list of suspects based on any and all criteria, including motive, opportunity, means, weapon availability, witnesses, crime scene evidence, and so on. This is known as "criminal profiling." Suppose a "person of interest" becomes a suspect, for example, because of a potential motive. In that case, a keen investigator will first determine if the suspect was present or could have been present at the crime scene.

The investigator, in this case, is establishing a correlation. Anyone knows that the murderer must have been present to have committed the crime. If a suspect is positively confirmed to have been three states away at the time that the homicide was committed, they are ruled out as a suspect (ignoring the hiring of a "hit man" hypothesis in this example).

On the other hand, a suspect will be further investigated if they are known to have been present at the scene of the crime. The correlation, if you will, is positive.

In the present analysis, I've developed and analyzed a list of "suspects" for the potential cause of obesity and diabetes. These are the "usual suspects," the primary food components that have been slandered, maligned, demonized, and vilified by journalists, nutritionists, physicians, and, yes, even nutrition researchers. These food "suspects" include excessive total calories (overeating), sugars, carbohydrates, refined carbohydrates, grains, wheat, vegetable oils, and lack of exercise.

Out of all of these, only one single component has been almost steadily increasing, that is, rising in parallel with increasing obesity and diabetes prevalence (and most all other chronic diseases) over the past 145 years, and that is industrial seed oils.

None of the other major food components share such an extremely high degree of correlation to the increasing prevalence of obesity and diabetes. Not increasing calories. Not sugar. Not carbohydrates. Not processed (refined) carbohydrates. Not grains. Not wheat. Not saturated fat or meat (although these weren't discussed herein). And not lack of exercise.

As of 2018, 42.5% of the nation over the age of 20 is obese, and another 31.1% are overweight; thus, 73.6% of adult Americans are either overweight or obese. It is essential to recognize that, with nearly three-fourths of Americans now either overweight or obese, the data in these graphs is representative of the nation at large.

In brief, how can we blame sugar consumption for escalating obesity rates when in the U.S.:

A) The consumption of sugar was already exceedingly high – 22.5% of calories – in 1935, yet obesity was relatively very low (about 4%);

B) Sugar consumption barely changed —an increase of 5% — between 1922 and 1987, while obesity climbed from about 2.95% to 18.6%, or 6.32-fold, and

C) Finally, obesity climbed from 30.5% in 1999 to 42.5% by 2018, precisely while sugar consumption was on the decline.

While obesity, diabetes, and sugar consumption have all increased dramatically over the past 150 years, the relationship between the independent variable – sugar consumption – and the dependent variables – obesity and diabetes – are generally stochastic, that is, not predictable.

Do we observe sugar consumption moving in direct correlation to obesity? No, we do not. In the U.S., for example, whether sugar is rising, falling, or flat, we have observed obesity steadily rising.

Do we observe sugar consumption moving in direct correlation to diabetes? Once again, no we do not. Very similar to obesity, whether sugar is rising, falling, or flat, we've observed diabetes steadily rising.

This is why the regression analysis completed by Professor Antipov turned out as it did: In his own words, "…there is no significant effect of sugar consumption on the prevalence of obesity and diabetes at all."

And even if "sugar theorists" should claim that sugar consumption surpassed a lower limit threshold beyond which obesity, diabetes, and chronic disease are induced in an all-or-none fashion, like cancer, then what is the explanation for the fact that obesity and diabetes prevalence have continued to rise?

Let's further entertain the hypothesis that sugar is causal in obesity and diabetes. If so, how could sugar consumption have averaged a very substantial 10.8% of calories in 1890, indicating many were consuming much more, yet obesity was 1.2%, and diabetes was extraordinarily rare at 0.0028%?

Again, the data does not align with such a theory.

Furthermore, how can we blame carbohydrates, per se, when we clearly observe that carbohydrates have slightly declined (about 5%) during a period (1909 to 2010) in which obesity has skyrocketed more than 30-fold?

There is also no substantial correlative evidence to suggest that refined grains are the primary culprit in obesity since we've clearly observed nothing more than minor increases (17 calories, 6.9%) during the 40-year period from 1978 to 2018. Total processed carbohydrates – added sugars and refined grains – only increased by 59 calories (8%) during this period.

On the other hand, there is overwhelming correlative evidence to indict vegetable oils as primary drivers of overweight and obesity. In fact, even without examining all of the other evidence in many nations and without the corroborating evidence in animal studies, as well as the many studies implicating seed oils as biologically plausible drivers of oxidation, inflammation, toxicity, nutrient deficiency, insulin resistance, and chronic disease, the evidence in the U.S. alone should practically convict seed oils as the culprit.

At this point, I would ask any casual or scientific observer, if it is not seed oils, what is it?

Again, correlation does not imply causation, but causation always implies correlation. And there is no shortage of strikingly positive correlations between industrial seed oils, obesity, and diabetes.

In my new book, *The Ancestral Diet Revolution*, I show many examples of declining sugar consumption in a given population while obesity and diabetes are simultaneously rising. This would include the United States (as reviewed herein), the United Kingdom, Australia, Israel, and Japan.

In some cases, such as the United States and Japan, we observe examples where total calories, carbohydrates, and sugar are all on the decline while obesity and diabetes (and other chronic diseases) are on the rise (the U.S. data, of course, is reviewed in this monograph). But what is the one food that is increasing?

Industrial seed oils.

Conclusion

In short, if we consider only two possible factors – sugar and vegetable oils – as causative of obesity and diabetes, and fully consider the effect of vegetable oils, the probability that sugar is the culprit in obesity and diabetes is negligible. In fact, it is virtually zero.

We can also state with statistical precision that, again, if only these two factors (sugar and vegetable oils) are considered causative of obesity and diabetes, the probability that vegetable oils do not have a positive effect on the induction and progression of obesity and diabetes is less than 1 in 10,000.

Alternatively stated, the statistical probability that industrial seed oils are indeed a major driver of obesity and diabetes is astronomically large.

I would suggest that the totality of evidence supporting the hypothesis that vegetable oils are the primary drivers of obesity and diabetes is enormous, incontrovertible, and irrefutable.

As shown repeatedly in *The Ancestral Diet Revolution*, industrial, highly polyunsaturated seed oils, when consumed to almost any degree (any amount is excess), accumulate in our body fat and produce a biological milieu that is concurrently pro-oxidative, pro-inflammatory, toxic, and nutrient deficient. I consider these to be the four pillars of metabolic hazard, ultimately causing severe mitochondrial dysfunction, metabolic syndrome, and physical degeneration.

In short, the highly polyunsaturated industrial seed oils, particularly including soybean, corn, canola, cottonseed, rapeseed, grapeseed, sunflower, safflower, and rice bran oils, are nothing short of chronic metabolic biological poisons.

These oils are poisons, plain and simple.

Could consumption of these oils in significant doses over prolonged periods end in catastrophic failure? Certainly, heart attacks, strokes, heart failure, major cancers, diabetic amputations, dementia, blindness, and premature death are all examples of catastrophic failure.

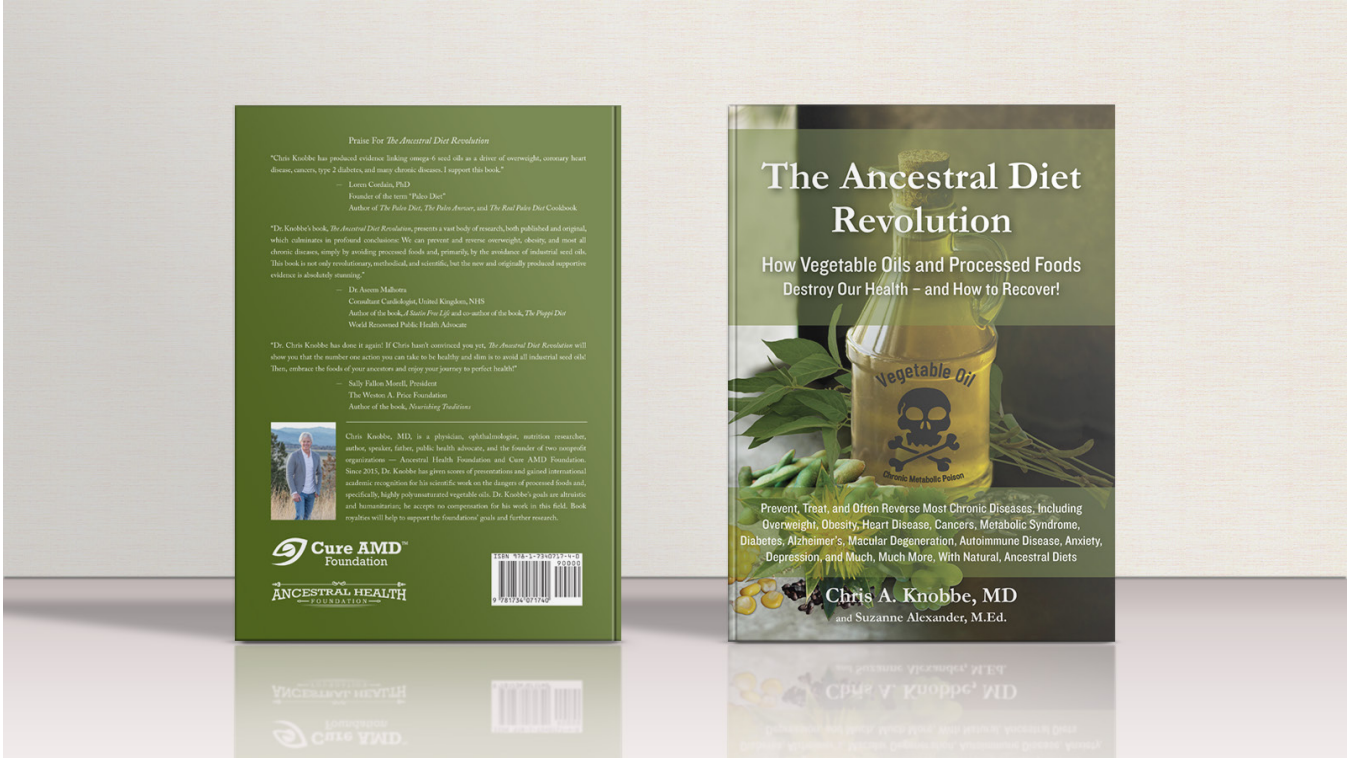
Could all of these diseases be driven primarily by industrial seed oils?

The evidence I have prepared will shock you.

I invite you to read my latest book, *The Ancestral Diet Revolution*, to learn more and get all the details.

To get the book on Amazon, click here.

To get the book at Barnes & Noble, click here.



A Message from the Author:

Will You Help Us to Prevent Suffering and Save Lives?

To conclude this not-so-brief introduction to my newest book, ***The Ancestral Diet Revolution***, we ask that you consider purchasing this book, reading it, and providing a review on Amazon. This will help spread this message of health and hope.

I left practice nine years ago, at the time of this writing, to pursue this research full-time. I've funded much of this work and these nonprofit foundations with my own hard-earned monies. I haven't earned a single dime in more than 9 years, since February of 2015. But I've never looked back. I have no regrets.

Some will consider me to be an iconoclast or a zealot. My lectures and presentations might be considered passionate diatribes against industrial seed oils, but no one has accused my work as being non-scientific.

We've reached millions of people with this message. Most of those people have been reached through presentations I've given in eight countries, some of which have been published on YouTube. I've also been interviewed on scores of podcasts, and we've reached many thousands through books and written pieces like this one.

I believe this work is my calling. And I like to think about how many lives my work has positively impacted. How many people will not suffer heart attacks, strokes, blindness, dementia, and premature death because of my work? How many more will not have to endure cancer?

Obesity and diabetes are the primary topics of this monograph. But I'm in this business to prevent suffering and to save lives. And I hope to accomplish this on a massive scale.

I accept no compensation for my work in Nutrition. Book royalties from both of my books (in Nutrition) support the Ancestral Health Foundation and Cure AMD Foundation. I have no financial relationship with any industry.

One hundred percent of philanthropic contributions to Ancestral Health Foundation and Cure AMD Foundation support the operations of these nonprofit 501(c)(3) organizations, current and forthcoming research, and scientific expeditions.

Please consider supporting these foundations. Your generous gifts will support research that will change allopathic Medicine, ultimately preventing suffering and saving lives.

Sincerely,

Chris A. Knobbe, MD

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ANCESTRAL HEALTH FOUNDATION

Cure AMD™ Foundation

CHRIS KNOBBE, MD

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