Are Dietary Saturated Fats Dangerous?

This is an epic post from my old Advanced Mediterranean Diet blog, originally dated July 6, 2009. That was a watershed year for me because of the ideas in this article. If you or your doctor think low-carb eating is dangerous because it may be higher in saturated fat, this post should convince you otherwise.

I've been thinking a lot lately about saturated fats. Weird, huh?

The American Heart Association recommends that Americans limit the amount of saturated fats they eat to less than 7 percent of total daily calories. If you eat 2,000 calories a day, no more than 140 of them should come from saturated fats. That's about 16 grams of saturated fats.

In over two decades of clinical practice, I've never run across a patient willing to do that calculation. Not many physicians could tell you the "seven percent rule."
One of the two major themes of Gary Taubes’ book, *Good Calories, Bad Calories*, is that dietary saturated fats are not particularly harmful to our health, if at all. From what I’ve been taught, this is sacrilegious. “Saturated fats are a major cause of heart disease and strokes,” I’ve heard and read over and over. In brief, this is the Diet-Heart Hypothesis or the “lipid hypothesis”: Dietary saturated fat, total fat, and cholesterol are directly related to coronary heart disease and other forms of atherosclerosis (aka hardening of the arteries).

In his review of Taubes’ book, Dr. George Bray didn’t even address Taubes’ point about saturated fats, writing instead, “read and decide for yourself.”

That started me thinking either that the Diet-Heart Hypothesis is indefensible or that Dr. Bray is lazy. I don’t think he’s lazy. Dr. Bray is a Grand High Pooh-Bah in the fields of obesity and nutrition.

The American Heart Association in 1957 recommended that polyunsaturated fats replace saturated fats.

U.S. public health recommendations in 1977 were to reduce fat intake to 30% of total calories to lower the risk of coronary heart disease. Slowly, some fats were replaced mostly with carbohydrates, highly refined ones at that. This shift tends to raise triglycerides and lower HDL cholesterol levels, which may themselves contribute to atherosclerosis. Current recommendations are, essentially, to keep saturated fatty acids as low as possible.

One concern about substituting carbohydrates for fats is that blood sugar levels rise, leading to insulin release from the pancreas, in turn promoting growth of fat tissue and potentially leading to weight gain. Some believe that the public health recommendation to reduce total fat (which led to higher carbohydrate intake) is the reason for the dramatic rise in overweight and diabetes we’ve seen over the last 30 years.

Note that if intake of saturated fats is inadequate, our bodies can make the saturated fats it needs from carbohydrates. These are generally the same saturated fats that are present in dietary fats of animal origin. The only exceptions are the two essential fatty acids: alpha-linolenic acid and linoleic acid.

Why would saturated fats be harmful? Apparently because they raise blood levels of cholesterol (including LDL cholesterol – “bad cholesterol”), which is thought to be a cause of atherosclerosis, which increases the risk of coronary heart disease and stroke. I don't recall seeing any mention of a direct toxic effect of saturated fats (or fatty acids) on arterial walls, where the rubber meets the road. (Saturated fats are broken down in the small intestine to glycerol and fatty acids.)

Dietary saturated fats also raise HDL cholesterol – “good cholesterol” – although not to the degree they raise LDL.

Let’s not forget many other factors that cause, contribute to, or predict coronary heart disease and atherosclerosis: smoking, family history, high blood pressure, obesity, diabetes, oxidative stress, homocysteine level, systemic inflammation, high-glycemic index diets, C-reactive protein, lack of exercise, and others. I discussed dietary factors in my April 14, 2009, blog post.

Often overlooked in discussion of dietary fat effects is the great variability of response to fats among individuals. Response can depend on genetics, sex, fitness level, overweight or not, types of carbohydrates eaten, amount of total dietary fat, etc. And not all saturated fats affect cholesterol levels.

Many of the journal articles listed as references below support the idea that the link between dietary saturated fats and coronary heart disease is not strong, and may be nonexistent. Read them and you’ll find that:

- Some studies show no association between dietary saturated fats and coronary heart disease.
- Some studies associate lower rates of coronary heart disease with higher saturated fat intake.
- Higher saturated fat intake was associated with less progression of coronary atherosclerosis in women.
- Lowering saturated fat intake did not reduce total or coronary heart disease mortality.
"Read and decide for yourself," indeed. I think you'll begin to question the reigning dogma.

For example, here's a conclusion from the Hooper article (from 2001):

In this review we have tried to separate out whether changes in individual fatty acid fractions are responsible for any benefits to health (using the technique of meta-regression). The answers are not definitive, the data being too sparse to be convincing. We are left with a suggestion that less total fat or less of any individual fatty acid fraction in the diet is beneficial.

And a conclusion of the J.B. German article:

At this time [2004], research on how specific saturated fatty acids contribute to coronary artery disease and on the role each specific saturated fatty acid play in other health outcomes is not sufficient to make global recommendations for all persons to remove saturated fats from their diet. No randomized clinical trials of low-fat diets or low-saturated fat diets of sufficient duration have been carried out; thus, there is a lack of knowledge of how low saturated fat intake can be without the risk of potentially deleterious health outcomes.

Zarraga and Schwartz (2006) conclude:

Numerous studies have been conducted to help provide dietary recommendations for optimal cardiovascular health. The most compelling data appear to come from trials that tested diets rich in fruits, vegetables, MUFAs [monounsaturated fatty acids], and PUFAs [polyunsaturated fatty acids], particularly the n-3 PUFAs. In addition, some degree of balance among various food groups appears to be a more sustainable behavioral practice than extreme restriction of a particular food group.

Here's another of my favorite quotes on this topic, from the J.B. German article:

If saturated fatty acids were of no value or were harmful to humans, evolution would probably not have established within the mammary gland the means to produce saturated fatty acids . . . that provide a source of nourishment to ensure the growth, development, and survival of mammalian offspring.

Take-Home Points

The connection between dietary saturated fat and coronary heart disease is weak.

I may be excommunicated from the medical community for uttering this. You won't hear it from most physicians or dietitians. They don't have time to spend 80 hours on this topic, so they stick with the party line. And maybe I'm wrong anyway.

The scientific community is slowly moving away from the original Diet-Heart/Lipid Hypothesis. It is being replaced with stronger anti-atherosclerosis theories that promote:

- fruit and vegetable intake
- whole grain intake
- low-glycemic index eating
- increased consumption of plant oils and fish
- moderate intake of nuts
- ? moderate intake of low-fat diary (e.g., DASH diet) (less consensus on this point)

So, saturated fats and dietary cholesterol are being crowded out of the picture, or ignored. In many cases, saturated fats have literally been replaced by poly- and monounsaturated fats (plant oils). Several clinical studies indicate that's a healthy change, but it may be related more to the healthfulness of the plant oils than to detrimental effects of saturated fats.

The original Diet-Heart Hypothesis won't die until the American Heart Association and U.S. public health agencies put a gun to its head and pull the trigger. That will take another 10 years or more.

If you want to hedge your bets, go ahead and limit your saturated fat intake. It probably won’t hurt you. It might help a wee bit. By the same token, I’m not going on an all-meat and cheese, ultra-high-saturated fat diet; I don’t want to miss out on the healthy effects of fruits, vegetables, whole grains, fish, nuts, and low-glycemic index carbohydrates. Some would throw red wine into the mix. This “prudent diet” reflects what I hereby christen The 21st Century Diet-Heart Hypothesis.

If you're worried about coronary heart disease and atherosclerosis, spend less time counting saturated fat grams, and more time on other risk-reducing factors: diet modification as above, get regular exercise, control your blood pressure, achieve a healthy weight, and don’t smoke. More bang for the buck.

What do you think?
Disclaimer: All matters regarding your health require supervision by a personal physician or other appropriate health professional familiar with your current health status. Always consult your personal physician before making any dietary or exercise changes.

Selected References Contradicting or Questioning the Diet-Heart Hypothesis (updated February 19, 2012):

Astrup, A., et al (including Ronald Krause, Frank Hu, and Walter Willett). The role of reducing intakes of saturated fat in the prevention of cardiovascular disease: where does the evidence stand in 2010. American Journal of Clinical Nutrition, 93 (2011): 684-688. (The authors believe that replacing saturated fats with polyunsaturated fats (but not carbohydrates) can reduce the risk of coronary heart disease (CHD). For the last four decades, low-fat diets replaced fat with carbohydrates. So they believe saturated fatty acids cause CHD or polyunsaturated fatty acids prevent it. I see no mention of total fat intake in this article written by major names in nutritional epidemiology and lipid metabolism. "In countries following a Western diet, replacing 1% of energy intake from saturated fats with polyunsaturated fats has been associated with a 2–3% reduction in the incidence of CHD." Furthermore, the effect of particular foods on CHD cannot be predicted solely by their content of total saturated fatty acids because individual saturated fatty acids may have different cardiovascular effects and major saturated fatty acid food sources contain other constituents that could influence coronary heart disease risk.”)


Food Reward versus Carbohydrate/Insulin Theory of Obesity

A few months ago, several of the bloggers/writers I follow were involved in an online debate about two competing theories that attempt to explain the current epidemic of overweight and obesity. The theories:

1. Carbohydrate/Insulin (as argued by Gary Taubes)
2. Food Reward (as argued by Stephan Guyenet)

The whole dustup was about as interesting to me as debating how many angels can dance on the head of a pin.

Regular readers here know I’m an advocate of the Carbohydrate/Insulin theory. I cite it in Conquer Diabetes and Prediabetes: The Low-Carb Mediterranean Diet and The Advanced Mediterranean Diet: Lose Weight, Feel Better, Live Longer (2nd edition). But the Food Reward theory also has validity. They’re both right, to an extent. They’re not mutually exclusive. The Food Reward theory isn’t as well publicized as Carbohydrate/Insulin.

Dr. Guyenet lays out a masterful defense of the Food Reward theory at his blog. Mr. Taubes presents his side here, here, here, here, and here. If you have a couple hours to wade through this, I’d start with Taubes’ posts in the order I list them. Finish with Guyenet.

You’d think I’d be more interested in this. I’m still not.

Moving from theory to real world practicality, I do see that limiting consumption of concentrated refined sugars and starches helps with loss of excess body fat and prevention of weight regain. Not for everybody, but many. Whether that’s mediated through lower insulin action or through lower food reward, I don’t care so much.

Any thoughts?

Steve Parker, M.D.

h/t Dr. Emily Deans
Book Review: Why We Get Fat

Gary Taubes's new book, *Why We Get Fat: And What To Do About It*, comes on the market later this month. I give it five stars per Amazon.com's ranking system (I love it).

At the start of my medical career over two decades ago, many of my overweight patients were convinced they had a hormone problem causing it. I carefully explained that's rarely the case. As it turns out, I may have been wrong. And the hormone is insulin.

Mr. Taubes wrote this long-awaited book for two reasons: 1) to make the ideas in his 2007 masterpiece (*Good Calories, Bad Calories*) more accessible to the public, and 2) to speed up the process of changing conventional wisdom on overweight. GCBC was the equivalent of a college-level course on nutrition, genetics, history, politics, science, physiology, and biochemistry. Many nutrition science geeks loved it while recognizing it was too difficult for the average person to digest.

Paradigm Shift

The author hopes to convince us that "We don't get fat because we overeat; we overeat because we're getting fat." We need to think of obesity as a disorder of excess fat accumulation, then ask why the fat tissue isn't regulated properly. A limited number of hormones and enzymes regulate fat storage; what's the problem with them?

Mr. Taubes makes a great effort convince you the old "energy balance equation" doesn't apply to fat storage. You remember the equation: eat too many calories and you get fat, or fail to burn up enough calories with metabolism and exercise, and you get fat. To lose fat, eat less and exercise more. He prefers to call it the "calories-in/calories-out" theory. He admits it has at least a little validity. Problem is, the theory seems to have an awfully high failure rate when applied to weight management over the long run. We've operated under that theory for the last half century, but keep getting fatter and fatter. So the theory must be wrong on the face of it, right? Is there a better one?

So, Why DO We Get Fat?

Here is Taubes's explanation. The hormone in charge of fat storage is insulin; it works to make us fatter, building fat tissue. If you've got too much fat, you must have too much insulin action. And what drives insulin secretion from your pancreas? Dietary carbohydrates, especially refined carbs such as sugars, flour, cereal grains, starchy vegetables (e.g., corn, beans, rice, potatoes), liquid carbs. These are the "fattening carbs." Dozens of enzymes and hormones are at play either depositing fat into tissue, or mobilizing the fat to be used as energy. It's an active process going on continuously. Any regulatory derangement that favors fat accumulation will CAUSE gluttony (overeating) or sloth (inactivity). So it's not your fault.

What To Do About It

Cut back on carb consumption to lower your fat-producing insulin levels, and you turn fat accumulation into fat mobilization.

Before you write off Taubes as a fly-by-night crackpot, be aware that he's received three Science-in-Society Journalism Awards from the National Association of Science Writers. He's a respected, professional science writer. Having read two of his books, it's clear to me he's very intelligent. If he's got a hidden agenda, it's well hidden.

One example illustrates how hormones control growth of tissues, including fat tissue. Consider the transformation of a skinny 11-year-old girl into a voluptuous woman of 18. Various hormones make her grow and accumulate fat in the places we now see curves. The hormones make her eat more, and they control the final product. The girl has no choice. Same with our adult fat tissue, but with different hormones. If some derangement is making us grow fatter, it's going to make us more sedentary (so more energy can be diverted to fat tissue) or make us overeat, or both. We can't fight it. At not least very well, as you can readily appreciate if look at the people around you at any American shopping mall.

This'N'That

Taubes's writing is clear and persuasive. He doesn't beat you over the head with his conclusions. He lays out a logical series of facts and potential connections and explanations, helping you eventually see things his way. If insulin controls fat storage by building and maintaining fat tissue, and if carbohydrates drive insulin secretion, then the way to reduce overweight and obesity is carbohydrate-restricted eating, especially avoiding the
fattening carbohydrates. I'm sure that's true for many folks, perhaps even a majority.

If you're overweight and skeptical about this approach, you could try out a very-low-carb diet for a couple weeks or a month at little expense and risk (but not zero risk). If Mr. Taubes and I are right, there's a good chance you'll lose weight. At the back of the book is a university-affiliated low-carb eating plan.

If cutting carb consumption is so critical for long-term weight control, why is it that so many different diets—with no focus on carb restriction—seem to work, if only for the short run? Taubes suggests it's because nearly all diets reduce carb consumption to some degree, including the fattening carbs. If you reduce your total daily calories by 500, for example, many of those calories will be from carbs. Simply deciding to “eat healthy” works for some people: stopping soda pop, candy bars, cookies, desserts, beer, etc. That cuts a lot of fattening carbs right there.

Losing excess weight or controlling weight by avoiding carbohydrates was the conventional wisdom prior to 1960, as documented by Mr. Taubes. Low-carb diets for obesity date back almost 200 years. The author attributes many of his ideas to German internist Gustav von Bergmann (1908).

Taubes discusses the Paleolithic diet, mentioning that the average paleo diet derived about a third of total calories from carbohydrates (compared to the standard American diet's 55% of calories from carb). My prior literature review found 40-45% of paleo diet calories from carbohydrate. I'm not sure who's right.

**Minor Bone of Contention RE: Coronary Heart Disease**

Mr. Taubes provides numerous scientific references to back his assertions. I checked out one in particular because it didn't sound right. Some background first.

Reducing our total fat and saturated fat consumption over the last 40 years was supposed to lower our LDL cholesterol, thereby reducing the burden of coronary heart disease, which causes heart attacks. Instead, we've experienced the obesity epidemic as those fats were replaced by carbohydrates. Taubes mentions a 2009 medical journal article by Kuklina et al, in which Taubes says Kuklina points out the number of heart attacks has not decreased as we've made these diet changes. Kuklina et al don't say that. In fact, age-standardized heart attack rates have decreased in the U.S. during the last decade.

Furthermore, autopsy data document a reduced prevalence of anatomic coronary heart disease in people aged 20-59 from 1979 to 1994, but no change in prevalence for those over 60. The incidence of coronary heart disease decreased in the U.S. from 1971 to 1998 (the latest reliable data). Death rates from heart disease and stroke have been decreasing steadily over the last 40 years in the U.S.; coronary heart disease death rates are down by 50%. I do agree with Taubes that we shouldn't credit those improvements to reduced total and saturated fat consumption. [Reduced trans fat consumption may play a role, but that's off-topic.]

I think Mr. Taubes would like to believe that coronary artery disease is either more severe or unchanged in the last few decades because of low-fat, high-carb eating. That would fit nicely with some of his theories, but it's not the case. Coronary artery disease is better now thanks to a variety of factors, but probably not diet (setting aside the trans-fat issue).

**Going Forward**

Low-carb dieting was vilified over the last half century partly out of concern that the accompanying high fat consumption would cause premature heart attacks, strokes, and death. We know now that total dietary fat and saturated fat have little to do with coronary heart disease and atherosclerosis (hardening of the arteries), which sets the stage for a resurgence of low-carb eating.

I advocate Mediterranean-style eating as the healthiest, in general. It's linked with prolonged life and lower risk of heart disease, stroke, dementia, diabetes, and cancer. On the other hand, obesity is a strong risk factor for premature death and development of heart disease, stroke, diabetes, and cancer. If consistent low-carb eating cures the obesity, is it healthier than the Mediterranean diet? Maybe so. Would a combination of low-carb and Mediterranean be better? Maybe so. I'm certain Mr. Taubes would welcome a decades-long interventional study comparing low-carb with the Mediterranean diet. But that's probably not going to happen in our lifetimes.

Gary Taubes rejects the calories-in/calories-out theory of overweight that hasn't done a very good job for us over the last 40 years. Taubes's alternative ideas deserve serious consideration.

Steve Parker, M.D.

**Update December 18, 2010: I found Mr. Taubes's reference for stating that Paleolithic diets provide about a third of calories from carbohydrate (22-40%), based on modern hunter-gatherer societies). See References below.**

**References:**


Myth Busted: Alzheimer Dementia NOT Caused By Diabetes

Contrary to popular belief among the experts, type 2 diabetes is not one of the causes of Alzheimer dementia. They may indeed be associated with each other, but that's not causation.

An oft-repeated theory from Gary Taubes' 2007 masterpiece, *Good Calories, Bad Calories*, is that many of the chronic diseases of modern civilization, including Alzheimer disease, are caused by abnormal blood sugar and insulin metabolism. Especially high insulin levels induced by a diet rich in refined carbohydrates. If that's the case, you'd expect to see a high prevalence of Alzheimer disease in older type 2 diabetics.

Dr. Emily Deans (psychiatrist) has been considering this issue recently at her *Evolutionary Psychiatry* blog.

The brains of Alzheimer patients, under a microscope, are characterized by many senile plaques (aka neuritic plaques) and neurofibrillary tangles. That's the gold standard for diagnosis. Nevertheless, brain biopsies are rarely done to diagnose Alzheimer disease in living patients, and even autopsies after death are rare. The diagnosis usually is clinical, based on ruling out other illnesses, etc.

Nearly all the studies associating diabetes with Alzheimer's disease (and other dementias) are observational or epidemiologic. [The exception is the Honolulu-Asia Aging Study.] Establishing an association is helpful in generating theories, but establishing causation is the goal. At least five studies confirm an association.

*Neurology* this year reported findings of Japanese researchers who examined the brains of 135 people who died between 1998 and 2003. They lived in Hisayama, a town with an incredibly high autopsy rate of 74%. These people before death had undergone an oral glucose tolerance test. Their insulin resistance was calculated on the basis of fasting glucose and fasting insulin (HOMA-IR). None of them showed signs of dementia at the time of study enrollment in 1988.

**What Did They Find?**

Twenty-one of the 135 subjects developed Alzheimer-type dementia. The investigators don't say if the diagnosis was based on the brain examination, or just a clinical diagnosis without a brain biopsy. How this got beyond the article reviewers is beyond me. [If I'm missing something, let me know in the comments section below.] It must be a clinical diagnosis because if you don't act demented, it doesn't matter how many senile plaques and neurofibrillary tangles you have in your brain.

Senile plaques, but not neurofibrillary tangles, were more common in those with higher levels of blood sugar (as measured two hours after the 75 g oral glucose dose), higher fasting insulin, and higher insulin resistance. People with the APOE epsilon-4 gene were at even higher risk for developing senile plaques.

The researchers did not report whether the subjects in this study had been previously during life with diabetes or not. One can only hope those data will be published in another paper. Why make us wait?

Average fasting glucose of all subjects was 106 mg/dl (5.9 mmol/l); average two-hour glucose after the oral glucose load was 149 mg/dl (8.3
mmol/l). By American Association of Clinical Endocrinologists criteria, these are prediabetic levels. Mysteriously, the authors fail to mention or discuss this. [I don't know if AACE criteria apply to Japanese.] Some of these Japanese subjects probably had diabetes, some had prediabetes, others had normal glucose and insulin metabolism.

As with all good research papers, the authors compare their findings with similar published studies. They found one autopsy study that tended to agree with their findings (Honolulu) and three others that don't (see references below). In fact, one of the three indicated that diabetes seems to protect against the abnormal brain tissue characteristic of Alzheimer disease.

**Botton Line**

Type 2 diabetes doesn't seem to be a cause of Alzheimer disease, if autopsy findings and clinical features are the diagnostic criteria for the disease.

If we assume that type 2 diabetics have higher than normal blood sugar levels and higher insulin levels for several years, then hyperglycemia and hyperinsulinemia don't cause or contribute to Alzheimer dementia. Myth busted. [I hope that's not copyrighted by the “Myth Busters” TV show.]

Type 2 diabetes is, however, linked with impaired cognitive performance, at least according to many of the scientific articles I read in preparation for this post. So type 2 diabetics aren't in the clear yet. It's entirely possible that high blood sugar and/or insulin levels cause or contribute to that. [Any volunteers to do the literature review? Best search term may be “mild cognitive impairment.”]

Type 2 diabetes is associated with Alzheimer disease, but we have no proof that diabetes is a cause of Alzheimers. Nor do we have evidence that high blood sugar and insulin levels cause Alzheimer disease.

Alzheimer disease is a major scourge on our society. I'd love to think that carbohydrate-restricted eating would help keep blood sugar and insulin levels lower and thereby lessen the devastation of the disease. Maybe it does, but I'd like to see more convincing evidence. It'll be years before we have a definitive answer.

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**Steve Parker, M.D.**

References:


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**2002 Atkins Diet at a Glance**

**Dr. Robert C. Atkins** is the modern popularizer of low-carb dieting. He was neither the first nor only low-carb advocate of the 20th century, but certainly the most influential in modern history in terms of followers. His *Dr. Atkins Diet Revolution* was published in 1972 and sold millions of copies.

**Sir Isaac Newton** wrote in 1676: “If I have seen further, it is by standing on the shoulders of giants.” Twentieth century giants for Dr. Atkins include Frank Evans, Blake Donaldson (the original paleo diet guru?), Per Hansen, Alfred Pennington, and John Yudkin. Most of these were physicians, by the way. **William Banting** preceded them, in the 19th century.

*Dr. Atkins New Diet Revolution*, published in 2002, was a huge seller then and maintains a respectable sales volume even now. My impression is that Atkins Nutritional, Inc., has replaced it with *The New Atkins for a New You*, which I reviewed last spring. Enough people still follow DANDR that I
need to stay familiar with it. Here’s my brief summary of the phases.

**Induction or Phase 1**

- Limit carbs to 20 g of “net carbs” daily for a minimum of two weeks.
- “Net carbs” is the total carb count in grams, minus the fiber grams.
- 3 cups of salad greens daily with olive oil/vingar or lemon juice OR 2 cups of salad greens and one cup of non-starchy cooked vegetables (e.g., broccoli or zucchini).
- May also eat 3–4 ounces of aged cheese, a handful of olives, and half an avocado daily.

**Ongoing Weight Loss (OWL) or Phase 2**

- Deliberate slowing of weight loss.
- Gradually add back nutrient-rich carbs.
- Increase net carbs weekly by just 5 g, by eating more veggies, nuts, seeds, even berries (this is where the “carb ladder” comes into play, adding carb groups in a specific order).
- Some dieters can even add small amounts of beans and fruits other than berries, until weight loss stalls. At that point, you drop back 5 g net carbs, to your Critical Carbohydrate Level for Losing (CCLL).

**Pre-maintenance or Phase 3**

- Begins 5 or 10 pounds before reaching your weight goal.
- Weight loss slows even more, taking at least 2 months to lose that last 10 pounds.
- Can now add some starchy veggies like sweet potatoes, peas, whole grains.
- If weight loss stops before goal, drop back down by 5-10 g net carbs, to your revised CCLL.

**Lifetime Maintenance or Phase 4**

- Starts when you’ve been at goal weight for one month.
- No more junk food, ever.
- Stay vigilant for excessive carbs. You may never be able to go back to whole grains or higher-carb fruits and vegetables.

Steve Parker, M.D.

PS: Gary Taubes reviews the history of low-carb diets in his masterpiece, *Good Calories, Bad Calories*.

**Sir Isaac Newton**

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Comments Off
Book Review: Good Calories, Bad Calories

Here’s my review of good Calories, Bad Calories: Challenging the Conventional Wisdom on Diet, Weight Control, and Disease, by Gary Taubes, 2007. I give it five stars on Amazon.com's five-star system (“I love it”).

♦ ♦ ♦ ♦

This brilliant book deserves much wider currency among physicians, dietitians, nutritionists, and obesity researchers. The epidemic of overweight and obesity over the last 30 years should make us question the reigning theories of obesity treatment and prevention. Taubes questioned those theories and pursued answers wherever the evidence led. He shares in GCBC his eye-opening, even radical, well-reasoned findings.

Ultimately, this tome is an indictment of the reigning scientific community and public nutrition policy-makers of the last four decades. That explains why, two years after publication, this serious, scholarly work has not been reviewed by the New England Journal of Medicine, the Journal of the American Medical Association, the American Journal of Clinical Nutrition, and the Journal of the American Dietetic Association (as of August, 2009).

In Part 1, Taubes examines the scientific evidence for what he calls the fat-cholesterol hypothesis. More commonly known as the diet-heart hypothesis, it’s the idea that dietary fat (especially saturated fat) and cholesterol clog heart arteries, causing heart attacks. Taubes finds the evidence unconvincing. He's probably right.

Part 2, The Carbohydrate Hypothesis, revives and older theory from the mid-twentieth century that is elsewhere called the Cleave-Yudkin carbohydrate theory of dental and chronic systemic disease. In the carbohydrate theory, high intake of sugary foods, starches, and refined carbohydrates leads first to dental disease (cavities, gum inflammation, periodontal disease) then, later, to obesity and type 2 diabetes, coronary heart disease, perhaps even cancer and Alzheimer's Disease. These are, collectively, the “diseases of civilization.”

Part 3 tackles obesity and weight regulation. Taubes writes that “…fattening and obesity are caused by an imbalance—a dysequilibrium—in the hormonal regulation of adipose [fat] tissue and fat metabolism.” Think of the transformation of a skinny 10-year-old girl into a voluptuous young woman. It’s not over-eating that leads to curvaceous fat deposits, growth of mammary tissue, and increase in height; it’s hormonal changes beyond her control.

The primary hormonal regulator of fat storage is insulin, per Taubes. Elevated insulin levels lead to storage of food energy as fat. Carbohydrates stimulate insulin secretion and make us fat.

Although it’s a brilliant book, by no means do I agree with all Taubes’ conclusions. For instance, if carbohydrates cause heart disease, why is glycemic index only very weakly associated with coronary heart disease in men? It’s way too early to blame cancer and Alzheimers on carbohydrates. Primitive cultures may not exhibit many of the diseases of civilization because their members die too young. Taubes is clearly an advocate of low-carb eating. Why didn't he directly address the evidence that fruits, vegetables, and whole grains in the right amounts are healthy?

I have to give Taubes credit for thinking “outside the box.” His search for answers included reviews of esoteric literature and interviews with scientists in the fields of genetics, anthropology, public policy, physiologic psychology, and paleontology, to name a few.

Towards the end of the book, Taubes describes a Mediterranean-style or “prudent” diet that is popular these days. After five years of research for his book, he says that whether a very low-carb meat diet is healthier than a prudent diet “… is still anybody's guess.” It’s hard for me to put aside numerous observational studies associating health benefits with legumes, fruits, vegetables, and wholegrains. So my “guess” is that the Mediterranean-style diet is healthier. Perhaps the answer is different for each individual. Heck, maybe the answer is low-carb Mediterranean. Both Taubes and I are prepared to accept either result when we have proof-positive data.

Taubes doesn't base his opinions on late-breaking scientific results. Instead, his research findings mostly span from 1930 to 1980, especially 1940-1960. Once the fat-cholesterol (diet-heart) hypothesis took root around 1960 and blossomed in the 1970s, these data were ignored by the entrenched academics and policy-makers of the day.

To be fair, I've got to mention this is not light reading. A majority of people never read another book after they graduate high school. Of those who do, many (like me) will have to look up the definition of “tautology,” “solecism,” etc.

I was taught in medical school years ago that “a calorie is a calorie is a calorie.” Meaning: if you want to lose excess weight, it doesn't matter if you cut calories from fat, protein, or carbohydrates. I really wonder about that now.

Steve Parker, M.D

Additional Reading


Quote of the Day

The urge to simplify a complex scientific situation so that physicians can apply it to their patients and the public embrace it has taken precedence over the scientific obligation of presenting the evidence with relentless honesty.

—Gary Taubes, in Good Calories, Bad Calories. (2007)
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- dLife
- Gab
- Goodbye Diabetes
- InfoGalactic: The Planetary Knowledge Core
- Mendosa.com
- Metabolism Society
- My Son's Art

Bible Verse of the Day

- Psalm 14:1
  “For the director of music. Of David. The fool says in his heart, “There is no God.” They are corrupt, their deeds are vile; there is no one who does good.”
Tag Archives: Gary Taubes. Zero Carb Interview: Sergey Yakunin. Posted on August 17, 2018 by esmeelafleur. 4. What books or people were most influential in guiding you to this way of eating? First of all you, Esmee, then Gary Taubes, Weston Price, Stefansson and the like. 5. Do you eat only meat, or do you include eggs, cheese, and cream in your diet? I only eat chicken, chicken liver, chicken eggs at present. Tag Archives for "Gary Taubes". 14. fructose... victim or villain? Why we really get fat (response to the Stephan Guyenet vs Gary Taubes debate on Joe Rogan Experience). Harnessing the power of the food insulin index. related. Guess what happened to body fat, lean mass and waist when a hundred people tried the Nutrient Optimiser? Why we really get fat (response to the Stephan Guyenet vs Gary Taubes debate on Joe Rogan Experience).