

Introduction: The Original Sin

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I'm going to argue that this calories-in/calories-out paradigm of adiposity is nonsensical:

that we don't get fat because we eat too much and move too little,

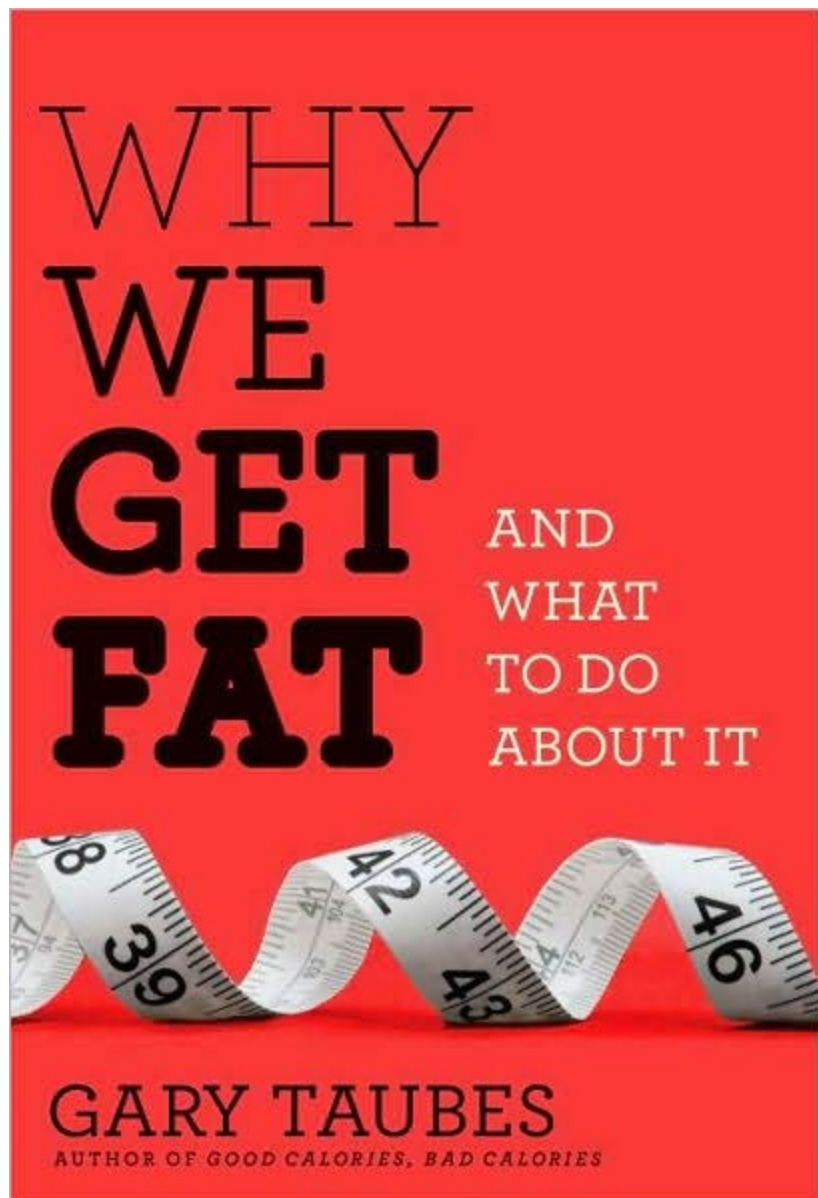
and that we can't solve the problem or prevent it by consciously doing the opposite. This is the original sin, so to speak, and we're never going to solve our own weight problems, let alone the societal problems of obesity and diabetes and the diseases that accompany them, until we understand this and correct it.

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The first part of this book will present the evidence against the calories-in/calories-out hypothesis.

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The second part of this book will present the way of thinking about obesity and excess fat that European medical researchers came to accept just prior to the Second World War. They argued, as I will, that it is absurd to think about obesity as caused by overeating, because anything that makes people grow—whether in height or in weight, in muscle or in fat—will make them overeat. Children, for example, don't grow taller because they eat voraciously and consume more calories than they expend. They eat so much—overeat—because they're growing. They need to take in more calories than they expend. The reason children grow is that they're secreting hormones that make them do so—in this case, growth hormone. And there is every reason to believe that the growth of our fat tissue leading



to overweight and obesity is also driven and controlled by hormones.

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when insulin levels are elevated, we accumulate fat in our fat tissue; when these levels fall, we liberate fat from the fat tissue and burn it for fuel.

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insulin levels are effectively determined by the carbohydrates we eat—not entirely, but for all intents and purposes.

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The more carbohydrates we eat, and the easier they are to digest and the sweeter they are, the more insulin we will ultimately secrete

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we do not get fat because we overeat; we get fat because the carbohydrates in our diet make us fat.

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science tells us that obesity is ultimately the result of a hormonal imbalance, not a caloric one

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These carbohydrates literally make us fat, and by driving us to accumulate fat, they make us hungrier and they make us sedentary. This is the fundamental reality of why we fatten,

Book #1 – Biology, not Physics

Chapter 1 – Why were they fat?

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But then he couldn't explain why the women were typically the fat ones, even though these women did virtually all the hard labor

2 – The Elusive Benefits of Undereating

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The result? After eight years of such undereating, these women lost an average of two pounds each. And their average waist circumference—a measure of abdominal fat—increased.

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Of all the reasons to question the idea that overeating causes obesity, the most obvious has always been the fact that undereating doesn't cure it.

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Prescribing low-calorie diets for obese and overweight patients, according to a 2007 review from Tufts University, leads, at best, to “modest weight losses” that are “transient”—that is, temporary. Typically, nine or ten pounds are lost in the first six months. After a year, much of what was lost has been regained. The Tufts review was an analysis of all the relevant diet trials in the medical journals since 1980. The single largest such trial ever done yields the very same answer.

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undereating isn't a treatment or cure for obesity; it's a way of temporarily reducing the most obvious symptom. And if undereating isn't a treatment or a cure, this certainly suggests that overeating is not a cause.

3 – The Elusive Benefits of Exercise

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The instructions that we're constantly being given to lose weight—eat less (decrease the calories we take in) and exercise more (increase the calories we expend)—are the same things we'll do if our purpose is to make ourselves hungry, to build up an appetite, to eat more.

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There are indeed excellent reasons to exercise regularly. We can increase our endurance and fitness by doing so; we may live longer, perhaps, as the experts suggest, by reducing our risk of heart disease or diabetes. (Although this has yet to be rigorously tested.) We may simply feel better about ourselves,

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the question I want to explore here is not whether exercise is fun or good for us (whatever that ultimately means) or a necessary adjunct of a healthy lifestyle, as the authorities are constantly telling us, but whether it will help us maintain our weight if we're lean, or lose weight if we're not. The answer appears to be no.

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That the poorer we are the fatter we're likely to be is one very good reason to doubt the assertion that the amount of energy we expend on a day-to-day basis has any relation to whether we get fat. If factory workers can be obese, as I discussed earlier, and oil-field laborers, it's hard to imagine that the day-to-day expenditure of energy makes much of a difference.

Thank you Kindle.

This is the first book I read on my Kindle e-reader. The Kindle allowed me to highlight text and gathered those highlights into a document.

I've marked what I consider to be Taubes' key points with a red box and a light blue background (like the paragraph above). I suggest you read those first.

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The ubiquitous faith in the belief that the more calories we expend, the less we'll weigh is based ultimately on one observation and one assumption. The observation is that people who are lean tend to be more physically active than those of us who aren't. This is undisputed. Marathon runners as a rule are not overweight or obese; the front-runners in marathons often look emaciated.

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We base our belief in the fat-burning properties of exercise on the assumption that we can increase our energy expenditure (calories-out) without being compelled to increase our energy intake (calories-in).

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If you go for a walk or rake some leaves, take a long hike, play two sets of tennis or eighteen holes of golf, you work up an appetite. You get hungry or hungrier.

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When Russell Wilder, an obesity and diabetes specialist at the Mayo Clinic, lectured on obesity in 1932, he said his **fat patients lost more weight with bed rest**

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First, we burn surprisingly few calories doing moderate exercise, and, second, the effort can be easily undone, and probably will be, by mindless changes in diet. A 250-pound man will burn three extra calories climbing one flight of stairs,

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The Danes actually did train sedentary subjects to run marathons (26.2 miles). After eighteen months of training, and after actually running a marathon, the eighteen men in the study had lost an average of five pounds of body fat. As for the nine women subjects, the Danes reported, “no change in body composition was observed.”

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[At the] second ever conference on obesity and weight control the assembled experts concluded that “the importance of exercise in weight control is less than might be believed, because increases in energy expenditure due to exercise also tend to increase food consumption, and it is not possible to predict whether the increased caloric output will be outweighed by the greater food intake.”

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And if reducing calories-in doesn't make us lose weight, and if increasing calories-out doesn't even prevent us from gaining it, maybe we should rethink the whole thing and find out what does.

Chapter 4 — The Significance of Twenty Calories a Day

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If it were true that our adiposity is determined by calories-in/calories-out, then this is one implication: you only need to overeat, on average, by twenty calories a day to gain fifty extra pounds of fat in twenty years.

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You need only to rein yourself in by this amount—undereat by twenty calories a day—to undo it. Twenty calories is less than a single bite of a McDonald’s hamburger or a croissant. It’s less than two ounces of Coke or Pepsi or the typical beer. Less than three potato chips. Maybe three small bites of an apple.

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Ask yourself: How is it possible that anyone stays lean, if all it takes to grow gradually obese is to overshoot this point of energy balance by twenty calories daily? Because quite a few people do stay lean.

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One or two bites or swallows too many (out of the hundred or two we might take to consume a day’s worth of sustenance) and we’re doomed.

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The fact that many people do remain lean for decades (although it’s less common now than in Du Bois’s day), and that even those who are fat don’t continuously get fatter, suggests there is something more going on with this business of weight regulation than can be explained by the notion that it’s all about calories.

Chapter 5 — Why Me? Why there (tummy vs. hips) Why then (age)?

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It’s been known since the 1930s that obesity has a large genetic component. If your parents are fat, it’s far more likely you will be fat than someone whose parents are lean. Another way to say this is that body types run in families.

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Another conspicuous piece of evidence arguing against calories-in/calories-out is that men and women fatten differently. Men typically store fat above the waist—the beer belly—and women below the waist. Women put on fat in puberty, particularly in breasts, hips, butt, and thighs, and men lose fat during puberty and gain muscle. When boys become men, they become taller, more muscular, and leaner. Girls enter puberty with very slightly more body fat than boys (6 percent more, on average), but by the time puberty is over, they have 50 percent more.

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when a girl enters puberty as slender as a boy and leaves it with the shapely figure of a woman, it's not because of overeating or inactivity, even though it's mostly the fat she's acquired that gives her that womanly shape and she had to eat more calories than she expended to accommodate that fat.

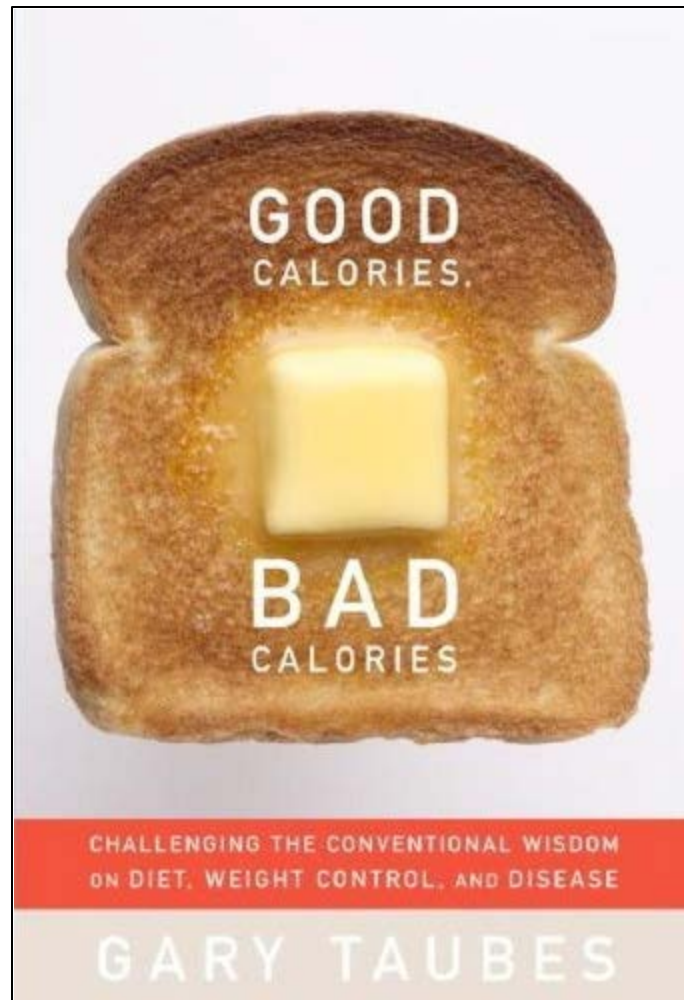
Chapter 6 – Thermodynamics for Dummies, Part #1

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Newton's laws of motion, Einstein's relativity, the electrostatic laws, quantum mechanics—they all describe properties of the universe we no longer question. But they don't tell us why we get fat. They say nothing about it, and this is true of the laws of thermodynamics as well. It is astounding how much bad science—and so bad advice, and a growing obesity problem—has been the result of the experts' failure to understand this one simple fact. The very notion that we get fat because we consume more calories than we expend would not exist without the misapplied belief that the laws of thermodynamics make it true.

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Obesity is not a disorder of energy balance or calories-in/calories-out or overeating, and thermodynamics has nothing to do with it.



Taubes' First Book

Chapter 7 – Thermodynamics for Dummies, Part #2

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People who semi-starve themselves, or who are semi-starved during wars, famines, or scientific experiments, are not only hungry all the time (not to mention cranky and depressed) but lethargic, and they expend less energy. Their body temperatures drop; they tend to be cold all the time. And increasing physical activity does increase hunger; exercise does work up an appetite; lumberjacks do eat more than tailors. Physical activity also makes us tired; it wears us out. We expend less energy when the activity is over. In short, the energy we consume and the energy we expend are dependent on each other. Mathematicians would say they are dependent variables, not independent variables, as they have typically been treated. Change one, and the other changes to compensate. To a great extent, if not entirely, the energy we expend from day to day and week to week will determine how much we consume, while the energy we consume and make available to our cells (a key point, as I will discuss later) will determine how much we expend. The two are that intimately linked.

Chapter 8 – Head Cases

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fat people respond to food restriction just as fat animals do—they reduce their energy expenditure, while experiencing increased hunger

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we've opened up the possibility that the same physiologic mechanism that drives obese individuals to hold on to their **fat in the face of semi-starvation** might have been the cause of their obesity in the first place.

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So long as we believe that people get fat because they overeat, because they take in more calories than they expend, we're putting the ultimate blame on a mental state, a weakness of character, and we're leaving human biology out of the equation entirely.

Book II – Adiposity 101 Chapter 9 – The Laws of Adiposity

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In the early 1970s, a young researcher at the University of Massachusetts named George Wade set out to study the relationship between sex hormones, weight, and appetite by removing the ovaries from rats (females, obviously) and then monitoring their subsequent weight and behavior.* The effects of the surgery were suitably dramatic: the rats would begin to eat voraciously and quickly become obese. If we didn't know any better, we might assume from this that the removal of a rat's ovaries makes it a glutton. The rat eats too much, the excess calories find their way to the fat tissue, and the animal becomes obese. This would confirm our preconception that overeating is responsible for obesity in humans as well. But Wade did a revealing second experiment, removing the ovaries from the rats and putting them on a strict postsurgical diet. Even if these rats were ravenously hungry after the surgery, even if they

desperately wanted to be gluttons, they couldn't satisfy their urge. In the lingo of experimental science, this second experiment controlled for overeating. The rats, post-surgery, were only allowed the same amount of food they would have eaten had they never had the surgery. What happened is not what you'd probably think. The rats got just as fat, just as quickly. But these rats were now completely sedentary. They moved only when movement was required to get food.

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The way Wade explained it to me, the animal doesn't get fat because it overeats, it overeats because it's getting fat.

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The removal of the ovaries literally makes the rat stockpile body fat; the animal either eats more or expends less energy, or both, to compensate.

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(When estrogen was infused back into the rats post-surgery, they did not eat voraciously, become slothful, or grow obese. They acted like perfectly normal rats.)

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The animal has the urge to eat voraciously because it's now losing calories into its fat cells that are needed elsewhere to run its body. The more calories its fat cells sequester, the more it must eat to compensate. The fat cells, in effect, are hogging calories, and there aren't enough to go around for other cells. Now a meal that would previously have satisfied the animal no longer does.

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The only way (short of more surgery) to stop these animals from getting fat—dieting has no effect, and we can be confident that trying to force them to exercise would be futile—is to give them their estrogen back. When that is done, they become lean again, and their appetite and energy levels return to normal. So removing the ovaries from a rat literally makes its fat cells fatten. And this, very likely, is what happens to many women who get fat when they have their ovaries removed or after menopause.

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We have to conclude, as Wade did for his rats, that those who get fat do so because of the way their fat happens to be regulated and that a conspicuous consequence of this regulation is to cause the eating behavior (gluttony) and the physical inactivity (sloth) that we so readily assume are the actual causes.

The First Law

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our bodies, when healthy, are working diligently to maintain a set amount of fat in our fat tissue—not too much and not too little—and that this, in turn, is used to assure a steady supply of fuel to the cells. The implication (our working assumption) is that if someone gets obese it's because this regulation has been thrown out of whack, not that it's ceased to exist.

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When animals do put on significant fat, that fat is always there for a very good reason.*

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Excellent examples of how carefully animals (and so presumably humans, too) regulate their fat accumulation are hibernating rodents—ground squirrels, for example, which double their weight and body fat in just a few weeks of late summer.

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these squirrels will accumulate this fat regardless of how much they eat,

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The fact is, there's very little that researchers can do to keep these animals from gaining and losing fat on schedule.

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A final argument for the careful regulation of body fat is the fact that everything else in our bodies is meticulously regulated. Why would fat be an exception?

The Second Law

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Obesity can be caused by a regulatory defect so small that it would be undetectable by any technique yet invented.

The Third Law

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Whatever makes us both fatter and heavier will also make us overeat.

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Anything that increases its mass, for whatever reason, will take in more energy than it expends. So, if a regulatory defect makes us both fatter and heavier, it is guaranteed to make us consume more calories

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African elephants are the world's largest land animals. The males typically weigh more than ten thousand pounds, although surprisingly little of this is fat. Blue whales are the largest animals, on or off land. They can weigh three hundred thousand pounds, and much of that is fat. African elephants will eat hundreds of pounds of food a day, and blue whales, thousands,* prodigious amounts, but neither species grow to be enormous because they eat so much. They eat prodigious

Consult your M.D., but first...

Gary Taubes' two books (including his earlier *Good Calories Bad*

Calories) are controversial among

traditional medical folks. This

suggests you may want to be careful

before you dive into any major diet

changes. I'd suggest you read the

book first because your doctor will

probably think its heresy.

amounts because they're enormous animals. With or without large quantities of body fat, body size determines how much they eat.

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we would never even consider the possibility that children grow taller because they eat too much and exercise too little (or that they stunt their growth by exercising too much). So why assume that these are valid explanations for growing fat (or remaining lean)?

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The fat tissue is not reacting to how much these animals are eating but only to the forces making them accumulate fat. And because increasing body fat requires energy and nutrients that are needed elsewhere in their bodies, they will eat more if they can. If they can't—if they are on a strict diet—they will expend less energy, because they have less to expend. They may even compromise their brains, muscles, and other organs. Half-starve these animals and they'll still find a way to stockpile calories as fat, because that's what their fat tissue is now programmed to do.

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This implies that our emaciated marathoners are not lean because they train religiously and burn off thousands of calories doing so; rather, they're driven to expend those calories—and so perhaps to work out for hours a day and become obsessive long-distance runners—because they're wired to burn off calories and be lean.

10 — A Historical Digression on “Lipophilia”

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a predisposition to fatten easily or remain lean is obviously determined in large part by our genes—a heritage, something passed down from generation to generation. If genes determine our height and our hair color and the size of our feet, he said, then “why can't heredity be credited with determining one's shape?” But if genes control our shape, how do they do it?

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Dozens of enzymes and multiple hormones had already been identified that influence fat accumulation, Astwood explained. Some work to liberate fat from the fat tissue; others to put it there.

The Controversy

If you're not sure how the conventional health community feels about Taubes' work, just log on to the web site for the Charlie Rose show and watched [the 2002 show](#) when Dr. Mehmet Oz (of Oprah fame) was guest host and moderated a “spirited” discussion that included Dean Ornish, Barbara Howard (American Heart Association) and Taubes. It did get ugly though it's safe to say all sides agreed that simple carbohydrates and sugar should be avoided.

Ultimately, the amount of fat that would be stored in any single person or at any single location on the human body would be determined by the balance of these competing regulatory forces. “Now just suppose that any one of these ... regulatory processes were to go awry,”

11 — A Primer on the Regulation of Fat

The Basics (Why Anyone Gets Fat)

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The reason nutritionists like to think (and like to tell us) that carbohydrates are somehow the preferred fuel for the body, which is simply wrong, is that your cells will burn carbohydrates before they'll burn fat. They do so because that's how the body keeps blood sugar levels in check after a meal. And if you're eating a carbohydrate-rich diet, as most people do, your cells will have a lot of carbohydrates to burn before they get to the fat.

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You'll start secreting insulin (from the pancreas) even before you start eating—indeed; it's stimulated just by *thinking* about eating.

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When you take your first bites, more insulin will be secreted. And as the glucose from the meal begins flooding the circulation, still more is secreted.

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The insulin then signals cells throughout the body to increase the rate at which they're pumping in glucose from the bloodstream.

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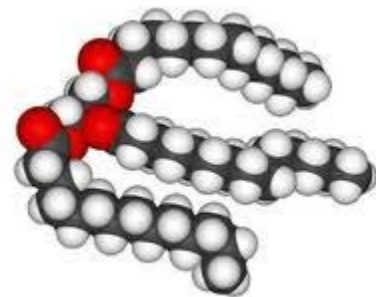
The fat in our bodies exists in two different forms that serve entirely different purposes. Fat flows in and out of cells in the form of molecules called “fatty acids”; this is also the form we burn for fuel. We store fat in the form of molecules called “triglycerides,” which are composed of three fatty acids (“tri-”) bound together by a molecule of glycerol (“glyceride”).

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triglycerides are too big to flow through the membranes that surround every fat cell, whereas fatty acids are small enough to slip through cell membranes with relative ease, and so they do. Flowing back and forth, in and out of fat cells all day long, they can be burned for fuel whenever needed. Triglycerides are the form in which fat is fixed inside fat cells, stashed away for future use.

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As a result, anything that works to promote the flow of fatty acids into your fat cells, where they can be bundled together



Triglyceride:
1 Glycerol (red) + 3 Fatty Acids 1

into triglycerides, works to store fat, to make you fatter. Anything that works to break down those triglycerides into their component fatty acids so the fatty acids can escape from the fat cells works to make you leaner.

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LPL is the enzyme that sticks out from the membranes of different cells and then pulls fat out of the bloodstream and into the cells. If the LPL is on the surface of a muscle cell, then it directs the fat into the muscle to be used for fuel. If it's on a fat cell, then it makes that fat cell fatter. (The LPL breaks down triglycerides in the bloodstream into their component fatty acids, and then the fatty acids flow into the cell.) As I said previously, the female sex hormone estrogen stifles the activity of LPL on fat cells and so works to decrease fat accumulation.

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Insulin activates LPL on fat cells, particularly the fat cells of the abdomen; it “up-regulates” LPL, as researchers say. The more insulin we secrete, the more active the LPL on the fat cells, and the more fat is diverted from the bloodstream into the fat cells to be stored. Insulin also happens to suppress LPL activity on the muscle cells, assuring that they won't have many fatty acids to burn. (Insulin also tells muscle cells and others in the body not to burn fatty acids but to continue burning up blood sugar instead.) This means that when fatty acids do escape from a fat cell, if insulin levels happen to be high, these fatty acids won't be taken up by the muscle cells and used for fuel. They'll end up back in the fat tissue.

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Insulin also influences an enzyme that we haven't discussed, hormone-sensitive lipase, or HSL for short.

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HSL works to make fat cells (and us) leaner. It does so by working inside the fat cells to break down triglycerides into their component fatty acids, so that those fatty acids can then escape into the circulation. The more active this HSL, the more fat we liberate and can burn for fuel and the less, obviously, we store. Insulin also suppresses this enzyme HSL, and so it prevents triglycerides from being broken down inside the fat cells and keeps the outward flow of fatty acids from the fat cells to a minimum. And it takes just a little bit of insulin to accomplish this feat of shutting down HSL and trapping fat in our fat cells. When insulin levels are elevated, even a little, fat accumulates in the fat cells.

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insulin signals liver cells not to burn fatty acids but to repackage them into triglycerides and ship them back to the fat tissue. It even triggers the conversion of carbohydrates directly into fatty acids in the liver and in the fat tissue,

Addicted to carbs?

One big thing I got from this book is that some people are extra sensitive to simple carbohydrates and are thus prone to gaining fat. If that includes you, I recommend you buy the book and read it.

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In short, everything insulin does in this context works to increase the fat we store and decrease the fat we burn. Insulin works to make us fatter.

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The one thing we absolutely have to do if we want to get leaner—if we want to get fat out of our fat tissue and burn it—is to lower our insulin levels and to secrete less insulin to begin with.

The Implications

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So, as we fatten, our energy demand increases, and our appetite will increase for this reason as well—particularly our appetite for carbohydrates, because this is the only nutrient our cells will burn for fuel when insulin is elevated. This is a vicious cycle, and it’s precisely what we’d like to avoid. If we’re predisposed to get fat, we’ll be driven to crave precisely those carbohydrate-rich foods that make us fat.

Chapter 12 — Why I Get Fat and You Don’t (or Vice Versa)

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One way to think about insulin in this context is as a hormone that determines how fuels are “partitioned” around the body. After a meal, insulin and the various enzymes it influences, such as LPL, determine what proportion of the different nutrients will be sent to which tissues, how much will be burned, how much will be stored, and how this will change with need and with time. Since I’m concerned here with whether fuels will be used for energy or stored, imagine insulin and these enzymes as determining which way the needle points on what I’m going to call a fuel-partitioning gauge. Imagine it looking like the fuel gauge in your car, but instead of the “F” standing for “full” on the right, it stands for “fat,” and the “E” on the left doesn’t stand for “empty,” but for “energy.”

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[If your needle naturally] points to the right—toward the “F”—it means that insulin partitions a disproportionate amount of the calories you consume into storage as fat, rather than use for energy by the muscles. **In this case, you’ll have a tendency to fatten, and you’ll have less energy available for physical activity, so you’ll also tend to be sedentary.** The farther the needle points toward fat storage, the more calories will be stored, the fatter you’ll be. If you don’t want to be sedentary, of course, then you have to eat more to compensate for this loss of calories into fat.

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[If your needle naturally] points in the other direction—toward the “E”—you’ll be burning as fuel a disproportionate share of the calories you consume. You’ll have plenty of energy for physical activity, but little will be stored as fat. You’ll be lean and active (just as you’re supposed to be), and you’ll eat in moderation. The farther out you go in this direction, the more energy you’ll have for physical activity and the less will be stored—the leaner you’ll be.

Emaciated-looking marathoners can be found down here.

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But another important factor is just how sensitive to insulin your cells happen to be and how quickly they become insensitive—the property called “insulin resistance”—in response to the insulin you secrete. This idea of being resistant to insulin is absolutely critical to understanding the reasons we get fat and also many of the diseases associated with it. I’ll return to it frequently. The more insulin you secrete, the more likely it is that your cells and tissues will become resistant to that insulin. That means it will take more insulin to do the same glucose-disposal job, keeping blood sugar under control.

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If these cells become resistant to insulin, more insulin is required to keep blood sugar levels in check, so now you secrete more insulin, which prompts more insulin resistance. And all the while, that insulin is working to make you fatter (to store calories as fat), unless your fat cells are also resistant to it. So secreting more insulin will move the needle on the fuel-partitioning gauge toward storage.

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The more sensitive a particular tissue is to insulin, the more glucose it will take up when insulin is secreted. If it’s muscle, it will store more glucose as glycogen and burn more for fuel. If it’s fat, it will store more fat and release less.

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These cells now generate less energy, and this is what we mean when we say that our metabolism slows down. Our “metabolic rate” decreases. Once again, what appears to be a cause of fattening—the slowing of our metabolism—is really an effect. You don’t get fat because your metabolism slows; your metabolism slows because you’re getting fat.

Chapter 13 – What We Can Do

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Not all of us get fat when we eat carbohydrates, but for those of us who do get fat, the carbohydrates are to blame; the fewer carbohydrates we eat, the leaner we will be.

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The carbohydrates in leafy green vegetables like spinach and kale, on the other hand, are bound up with indigestible fiber and take much longer to be digested and enter our bloodstream. These vegetables contain more water and fewer digestible carbohydrates for their weight than starches

like potatoes. We have to eat far more to get the same load of carbohydrates, and those carbohydrates take longer to digest. As a result, blood sugar levels remain relatively low when we eat these vegetables; they initiate a far more modest insulin response and are therefore less fattening. It is possible, though, that some people may be so sensitive to the carbohydrates in their diet that even these green vegetables may be a problem.

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But if we're predisposed to put on fat, it's a good bet that most fruit will make the problem worse, not better.

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The very worst foods for us, almost assuredly, are indeed sugars—sucrose (table sugar) and high-fructose corn syrup in particular.

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The human body, and particularly the liver, never evolved to handle the kind of fructose load we get in modern diets.

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fructose as the most “lipogenic” carbohydrate—it's the one we convert to fat most readily.

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Our “pattern of fructose metabolism” changes with time, as the British biochemist and fructose expert Peter Mayes says. Not only will this cause us to accumulate fat directly in the liver—a condition known as “fatty liver disease”—but it apparently causes our muscle tissue to become resistant to insulin through a kind of domino effect that is triggered by the liver cells' resistance. So, even though fructose has no immediate effect on blood sugar and insulin, over time—maybe a few years—it is a likely cause of insulin resistance and thus the increased storage of calories as fat. The needle on our fuel-partitioning gauge will point toward fat storage, even if it didn't start out that way. It's quite possible that if we never ate these sugars we might never become fat or diabetic, even if the bulk of our diet were still starchy carbohydrates and flour. This would explain why some of the world's poorest populations live on carbohydrate-rich diets and don't get fat or diabetic, while others aren't so lucky.

Chapter 14 — Injustice Collecting

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The message of Adiposity 101 is simple enough: if you're predisposed to get fat and want to be as lean as you can be without compromising your health, you have to restrict carbohydrates and so keep your blood sugar and insulin levels low. The point to keep in mind is that you don't lose fat because you cut calories; you lose fat because you cut out the foods that make you fat—the carbohydrates.

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That only some people get fat from eating carbohydrates (just as only some get lung cancer from smoking cigarettes) doesn't change the fact that if you're one of those who do, you'll only lose fat and keep it off if you avoid these foods.

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The longer we go between meals and the more energy we've expended, the hungrier we'll be. And the hungrier we are, the better foods will taste:

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Remember, we begin secreting insulin just by thinking about eating (and particularly eating carbohydrate-rich foods and sweets), and this insulin secretion then increases within seconds of taking our first bite.

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This palatability-by-blood-sugar-and-insulin response is almost assuredly exaggerated in people who are fat or predisposed to get that way. And the fatter they get, the more they'll crave carbohydrate-rich foods, because their insulin will be more effective at stashing fat and protein in their muscle and fat tissue, where they can't be used for fuel.

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Once we get resistant to insulin, which will happen eventually, we'll have more insulin coursing through our veins during much, if not all, of the day.

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But sugar seems to hijack the signal to an unnatural degree, just as cocaine and nicotine do. If we believe the animal research, then sugar and high-fructose corn syrup are addictive in the same way that drugs are and for much the same biochemical reasons.

Chapter 15 – Why Diets Succeed and Fail

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The more likely explanation is that any diet that succeeds does so because the dieter restricts fattening carbohydrates, whether by explicit instruction or not. To put it simply, those who lose fat on a diet do so because of what they are not eating—the fattening carbohydrates—not because of what they are eating.

Chapter 16 – History of the Fatty Carbohydrate (I highlighted nothing in chapter 16)

Chapter 17 – Meat or Plants?

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our genes were effectively shaped by the two and a half million years during which our ancestors lived as hunters and gatherers prior to the introduction of agriculture twelve thousand years ago.

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It constitutes more than 99.5 percent of human history—more than a hundred thousand generations of humanity living as hunter-gatherers, compared with the six hundred succeeding generations of farmers or the ten generations that have lived in the industrial age.

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First, “whenever and wherever it was ecologically possible,” hunter-gatherers consumed “high amounts” of animal food. In fact, one in every five of these 229 populations subsisted almost entirely by hunting or fishing. These populations got more than 85 percent of their calories from meat or fish; some got 100 percent.

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Their diets were high to very high in protein compared with today (19 to 35 percent of calories), and high to very high in fat (28 to 58 percent of calories). And some of these populations consumed as much as 80 percent of their calories from fat,

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Hunter-gatherers, as these researchers explained, preferentially ate the fattest animals they could hunt; they preferentially ate the fattest parts of the animal, including organs, tongue, and bone marrow, and they would eat “virtually all” of the fat on the animal.

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Lions, for instance, will eat the fat organ meat of their kills and leave the “lean muscle meat” for scavengers.

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All of the plant foods that these populations gathered (seeds, nuts, roots, tubers, bulbs, “miscellaneous plant parts,” and fruits) would have what nutritionists today call a low glycemic index: they would be very slow to raise blood sugar, which would dictate an equally slow and measured insulin response. Not only would these hunter-gatherers eat relatively few carbohydrates, but what digestible carbohydrates they did eat would be bound up tightly with indigestible fiber,

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the machine refining of flour and sugar dates only to the late nineteenth century. Just two hundred years ago, we ate less than a fifth of the sugar we eat today.

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it has become popular to suggest that one problem with Western diets is the relative absence of fruit, it's worth remembering that we've been cultivating fruit trees for only the past few thousand years, and that the kinds of fruit we eat today—Fuji apples, Bartlett pears, navel oranges—have been bred to be far juicier and sweeter than the wild varieties and so, in effect, to be far more fattening.

Chapter 18 — The Nature of a Healthy Diet

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Since carbohydrates make us fat, it follows that the best and perhaps only way to avoid becoming fat is to avoid the carbohydrate-rich foods that are responsible. For those who are already fat, this implies that the best and perhaps only way to become lean again is to do the same.

The Con Job Argument (no notes) The Unbalanced Diet Argument

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If you cut back on all calories equally, or preferentially restrict fat calories, as we're often counseled, you'll be eating less fat and protein, which are not fattening, and more of the carbohydrates that are. Not only won't this diet work as well, if it works at all, but hunger will be a constant companion.

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The argument that a diet that restricts fattening carbohydrates will be lacking in essential nutrients—including vitamins, minerals, amino acids—does not hold up. First, the foods that you would be avoiding are the fattening ones, not leafy green vegetables and salads. This alone should take care of any superficial anxieties about vitamin or mineral deficiencies. Moreover, the fattening carbohydrates that are restricted—starches, refined carbohydrates, and sugars—are virtually absent essential nutrients in any case.

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Ever since the 1960s, when it was first argued that animal products could be bad for our health because they contain saturated fat, nutritionists have typically refrained from pointing out that meat contains all the amino acids necessary for life,† all the essential fats, and twelve of the thirteen essential vitamins in surprisingly large quantities. It's true nonetheless. Meat is a particularly concentrated source of vitamins A and E, and the entire complex of B vitamins. Vitamins B12 and D are found only in animal products (although we can get sufficient vitamin D from regular exposure to sunlight). Vitamin C is the one vitamin that is relatively scarce in animal products. But it appears to be the case, as it certainly is for the B vitamins, that the more fattening carbohydrates we consume, the more of these vitamins we need. We use B vitamins to metabolize glucose in our cells. So, the more carbohydrates we consume, the more glucose we burn (instead of fatty acids), and the more B vitamins we need from our diets. Vitamin C uses the same mechanism to get into cells (where it's needed) that glucose does, so the higher our blood

sugar level, the more glucose enters the cells and the less vitamin C. Insulin also inhibits what's called the uptake of vitamin C by the kidney, which means that when we eat carbohydrates we excrete vitamin C with our urine rather than retaining it, as we should, and using it. Without carbohydrates in the diet, there's every indication that we would get all the vitamin C we ever needed from animal products.

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Carbohydrates are not required in a healthy human diet. Another way to say this (as proponents of carbohydrate restriction have) is that there is no such thing as an essential carbohydrate.

The Heart Disease Argument

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Nutritionists initially got angry about carbohydrate restriction because they believed that the claims made for these diets were impossible, but this is the one that kept them angry and keeps their minds resolutely closed to any contrary evidence. They believe that if we buy into the logic of these diets, we'll replace what they consider “heart-healthy” carbohydrates—broccoli, whole-wheat bread, and potatoes, for instance—with meat, butter, eggs, and maybe cheese, which we very well might. Since the latter are all sources of saturated fat, the diets will raise our cholesterol, according to this logic, specifically the cholesterol in LDL (low-density lipoproteins), commonly known as the “bad” cholesterol, and we will have increased our risk of heart attack and premature death.... It's why most physicians and medical organizations still believe—or say they do—that the restricted carbohydrate diets are foolhardy.

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there's a well-documented relationship between obesity and heart disease.

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Fat, at least above the waist, and heart disease go hand in hand. (The fatter we are, or at least the more obese we are, the more likely we are to get virtually every major chronic disease.)

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As a nation, we were told to eat less fat and less saturated fat, which we did, or at least tried to do—saturated-fat consumption steadily declined over the years that followed, according to U.S. Department of Agriculture statistics—and yet, rather than getting leaner, we got fatter. What's more, the incidence of heart disease has not even diminished, which goes against expectations, if eating less fat or saturated fat makes a difference.

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That the official embrace of low-fat, high-carbohydrate diets coincided not with a national decline in weight and heart disease but with epidemics of both obesity and diabetes (both of which increase heart disease risk), should make any reasonable person question the underlying assumptions of the advice.

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“Despite decades of effort and many thousands of people randomized,” the Cochrane Collaboration authors concluded, “there is still only limited and inconclusive evidence of the effects of modification of total, saturated, monounsaturated, or polyunsaturated fats on cardiovascular morbidity [i.e., sickness] and mortality [death].”

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After six years on the diet, these women had cut both their total fat consumption and their saturated-fat consumption by a quarter, lowering their total cholesterol and their LDL cholesterol below (albeit only very slightly below) that of the other twenty-nine thousand women, who were eating whatever they wanted and yet their low-fat diet, as the final reports stated, had no beneficial effect on heart disease, stroke, breast cancer, colon cancer, or, for that matter, fat accumulation. Eating less total fat and saturated fat, and replacing the fatty foods with fruits and vegetables and whole grains, had no observable beneficial effect at all.*

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Let's start with triglycerides. These are also a risk factor for heart disease. The higher your level of circulating triglycerides (which are transported in the same lipoprotein particles that carry cholesterol), the greater the likelihood that you'll have a heart attack. This is not controversial. But it's the carbohydrates we eat that elevate triglyceride levels; fat, saturated or not, has nothing to do with them. If you replace the saturated fat in your diet with carbohydrates—replace eggs and bacon for breakfast, say, with cornflakes, skim milk, and bananas—your LDL cholesterol may go down, but your triglycerides will go up. What might be a good thing, lowering LDL cholesterol (and I'll explain the “might” shortly), will be compensated for by a bad thing, raising triglycerides. This has been recognized since the early 1960s.



Tensions were high when Taubes appeared on Dr. Oz's show

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Those of us with low HDL cholesterol are at far greater risk of having a heart attack than those of us with high total or LDL cholesterol. For women, HDL levels are so good at predicting future heart disease that they are, effectively, the only predictors of risk that matter.

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When you replace fat in your diet, even saturated fat, with carbohydrates, you lower your HDL, which means you make it more likely that you'll have a heart attack, at least by this predictor of risk. Once again, if you give up scrambled eggs and bacon for breakfast and replace them with cornflakes, skim milk, and bananas, your HDL cholesterol, your “good” cholesterol, will go down, and your heart-attack risk will go up. If you're currently eating cereal, skim milk, and bananas and switch instead to eggs and bacon, your HDL cholesterol will go up, and your heart-attack risk will go down. This has been known since the 1970s.

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In other words, if your HDL is high, it's a good bet that you're eating few carbohydrates. If your HDL cholesterol is low, then you're very likely eating a lot of carbohydrates.

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In total, **more than 70 percent of the fat in lard will improve your cholesterol profile** compared with what would happen if you replaced that lard with carbohydrates. The remaining 30 percent will raise LDL cholesterol (bad) but also raise HDL (good). In other words, and hard as this may be to believe, if you replace the carbohydrates in your diet with an equal quantity of lard, it will actually reduce your risk of having a heart attack.

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This is what happened to those who ate mostly fat and protein:

- They lost at least as much weight, if not considerably more.
- Their HDL cholesterol went up.
- Their triglycerides went way down.
- Their blood pressure went down.
- Their total cholesterol remained about the same.
- Their LDL cholesterol went up slightly.
- Their risk of having a heart attack decreased significantly.

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Gardner presented the results of the trial in a lecture that's now viewable on YouTube — [“The Battle of Weight Loss Diets: Is Anyone Winning \(at Losing\)?”](#)

The Bad Cholesterol Problem — Updating the LDL Connection



Researchers who study these things now say that it's not the cholesterol carried by the LDL that is to blame for heart disease but, rather, the LDL particle itself and other similar particles. The cholesterol seems to be an innocent bystander.

To complicate matters, not all LDL particles appear to be equally harmful...Some of the LDL in our circulation is large and buoyant, some is small and dense and there are gradations in between. The small and dense LDL particles appear to be the atherogenic ones, the ones we want to avoid. They work themselves into the walls of our arteries and begin the process of forming plaques. The large, buoyant LDL particles appear to be harmless.

This is important because carbohydrate-rich diets not only lower HDL and raise triglycerides; they also make LDL small and dense. These three effects all increase our risk of heart disease. When we eat high-fat diets and avoid carbohydrates, the opposite happens: HDL goes up, triglycerides go down, and the LDL in the circulation becomes larger and fluffier. Individually and together, these changes decrease our risk of having a heart attack.

Metabolic Syndrome



Earlier I discussed what happens when we become what's technically known as insulin-resistant — when muscle and liver cells, in particular, become resistant to the effect of the hormone insulin. Not only do we secrete more insulin in response, and so tend to get fatter, especially around the waist (where the fat cells are most sensitive to insulin), but we begin to manifest a host of other, related metabolic disturbances as well:

- blood pressure goes up
- triglycerides go up
- HDL cholesterol goes down.

What I didn't mention earlier is that LDL particles become small and dense. We become glucose-intolerant, which means we have trouble controlling our blood sugar. We might even develop type 2 diabetes, which happens when the pancreas can no longer secrete enough insulin to compensate for the insulin resistance.

This combination of heart-disease risk factors is now known as “metabolic syndrome,” and it is, in effect, the intermediate step on the way to heart disease.

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We know now that carbohydrates make us fat and it's been demonstrated in numerous clinical trials that low-carbohydrate, high-fat diets improve each and every one of the metabolic and hormonal abnormalities of metabolic syndrome — the low HDL, the high triglycerides, the small, dense LDL, the high blood pressure and the insulin resistance and chronically elevated levels of insulin. This suggests the obvious: that the same carbohydrates that make us fat are the ones that cause metabolic syndrome. And it tells us that the best and perhaps only way to treat the condition, as with obesity and overweight, is to avoid carbohydrate-rich foods, particularly the ones we digest easily and sugars.

Metabolic Syndrome — Redux

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As it turns out, both Alzheimer's disease and most cancers — including breast cancer and colon cancer — are associated with metabolic syndrome, obesity and diabetes.

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(some researchers have even taken to referring to Alzheimer's as “type 3 diabetes”)

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What makes us fat — the quality and quantity of carbohydrates we consume — also makes us sick.

Chapter 19 — Following Through

Moderation or Renounce Them Entirely? Part I

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And for some, weight will be lost only on a diet of virtually zero carbohydrates, and even this may not be sufficient to eliminate all our accumulated fat, or even most of it.

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Identify and avoid other foods that might stimulate significant insulin secretion — diet sodas, dairy products (cream, for instance), coffee, and nuts among others — and have more patience. (Anecdotal evidence suggests that occasional or intermittent fasting for eighteen or twenty-four hours might work to break through the plateaus of weight loss, but this, too, has not been adequately tested.)

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But the fact is that we are trying to counteract a regulatory disorder of fat metabolism, one that may have been years or decades in the making. Reversing the process might take more than a few months or even a few years as well.

Moderation or Renounce Them Entirely? Part II

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If you begin to gain weight, say, because you're now eating an apple a day and you don't want to be any heavier, then don't eat the apple. If you don't gain weight that means your body can tolerate an apple a day, and you can experiment with other carbohydrates.

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If a diet requires that you semi-starve yourself, it will fail, because (1) your body adjusts to the caloric deficit by expending less energy, (2) you get hungry and stay hungry, and (3) a product of both of these, you get depressed, irritable, and chronically tired. Eventually you go back to eating what you always did — or become a binge eater — because you can't abide semi-starvation and *its* side effects indefinitely.

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The biggest challenge is the craving for carbohydrates. The hunger that accompanies our attempts to eat fewer calories is an unavoidable physiological phenomenon; the craving for carbohydrates is more like an addiction.

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As I discussed earlier, sugar appears to be addictive in the brain in the same way in which cocaine, nicotine and heroin are. This suggests that the relatively intense craving for sugar — a sweet tooth — may be explained by the intensity of the dopamine secretion in the brain when we consume sugar.

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“After a year to 18 months,” he wrote, “the craving for sweets is lost,” and the children often pinpointed when this happened to “within a specific one to two-week period.”

What It Means to Eat as Much as We Would Like

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Another effect, though, of restricting carbohydrates is that your energy expenditure should increase. You're no longer diverting fuel into your fat tissue, where you can't use it, and so you literally have more energy to burn. By avoiding the fattening carbohydrates, you remove the force that diverts calories into your fat cells. Your body should then find its own balance between energy consumed (appetite and hunger) and energy expended (physical activity and metabolic rate). This process could take time, but it should happen without conscious thought.

Fat or Protein?

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There's good reason to question the benefits of diets that abnormally elevate the protein content. Populations that ate mostly meat or exclusively meat tried to maximize the fat they ate, and one reason seems to be that high-protein diets — without significant fat or carbohydrates — can be toxic.

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The short-term symptoms of these high-protein, low-fat, low-carbohydrate diets, these protein-metabolism experts point out, are weakness, nausea and diarrhea. These symptoms will disappear when the protein content is reduced to a more moderate 20 to 25 percent of calories and fat content is increased to compensate.

On Side Effects and Doctors

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[The shift of replacing carbohydrates with fat] can come with side effects. These can include weakness, fatigue, nausea, dehydration, diarrhea, constipation, a condition known as postural or orthostatic hypotension — if you stand up too quickly, your blood pressure drops precipitously, and you can get dizzy or even pass out...



Gary Taubes

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These symptoms have nothing to do with the high fat content of the diet. Rather, they appear to be a consequence of either eating too much protein and too little fat, of attempting strenuous exercise without taking the time to adapt to the diet, or, in most cases, of the body's failure to compensate fully for the restriction of carbohydrates and the dramatic lowering of insulin levels.

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When insulin levels drop, as they do when we restrict carbohydrates, our kidneys will excrete the sodium they've been retaining and with it water. For most people this is beneficial, and it's the reason why blood pressure comes down with carbohydrate restriction.

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For some the body will perceive the water loss as something to be prevented. It does so through a web of responses that can lead to water retention and what are called electrolyte imbalances (the kidneys excrete potassium to save sodium), and the result is the side effects cited. The reaction can be countered, as Phinney has noted, by adding sodium back into the diet: taking a gram or two of sodium a day (a half to one teaspoon of salt) or drinking chicken or beef broth

Appendix

Lifestyle Medicine Clinic Duke University Medical Center

“No Sugar, No Starch” Diet: Getting Started

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For most effective weight loss, you will need to keep the total number of carbohydrate grams to fewer than 20 grams per day.