foreword by **TIMOTHY NOAKES**

Why your body's own insulin is the key to controlling your weight

THE OBESITY CODE

UNLOCKING THE SECRETS OF WEIGHT LOSS

JASON FUNG, MD

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This book is dedicated to my beautiful wife, Mina. Thank you for your love and the strength you give me. I could not do it without you, nor would I ever want to.

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FOREWORD

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DR. JASON FUNG is a Toronto physician specializing in the care of patients with kidney diseases. His key responsibility is to oversee the complex management of patients with end-stage kidney disease requiring renal (kidney) dialysis.

His credentials do not obviously explain why he should author a book titled *The Obesity Code* or why he blogs on the intensive dietary management of obesity and type 2 diabetes mellitus. To understand this apparent anomaly, we need first to appreciate who this man is and what makes him so unusual.

In treating patients with end-stage kidney disease, Dr. Fung learned two key lessons. First, that type 2 diabetes is the single commonest cause of kidney failure. Second, that renal dialysis, however sophisticated and even life prolonging, treats only the final symptoms of an underlying disease that has been present for twenty, thirty, forty or perhaps even fifty years. Gradually, it dawned on Dr. Fung that he was practicing medicine exactly as he had been taught: by reactively treating the symptoms of complex diseases without first trying to understand or correct their root causes.

He realized that to make a difference to his patients, he would have to start by acknowledging a bitter truth: that our venerated profession is no longer interested in addressing the causes of disease. Instead, it wastes much of its time and many of its resources attempting to treat symptoms.

He resolved to make a real difference to his patients (and his profession) by striving to understand the true causes that underlie disease.

Before December 2014, I was unaware of Dr. Jason Fung's existence. Then one day I chanced upon his two lectures—"The Two Big Lies of Type 2 Diabetes" and "How to Reverse Type 2 Diabetes Naturally"—on YouTube. As someone with a special interest in type 2 diabetes, not least because I have the condition myself, I was naturally intrigued. Who, I thought, is this bright young man? What gives him the certainty that type 2 diabetes can be reversed "naturally"? And how can he be brave enough to accuse his noble profession of lying? He will need to present a good argument, I thought.

It took only a few minutes to realize that Dr. Fung is not only legitimate, but also more than able to look after himself in any medical scrap. The argument he presented was one that had been bouncing around, unresolved, in my own mind for at least three years. But I had never been able to see it with the same clarity or to explain it with the same emphatic simplicity as had Dr. Fung. By the end of his two lectures, I knew that I had observed a young master at work. Finally, I understood what I had missed.

What Dr. Fung achieved in those two lectures was to utterly destroy the currently popular model for the medical management of type 2 diabetes—the model mandated by all the different diabetes associations around the world. Worse, he explained why this erroneous model of treatment must inevitably harm the health of all patients unfortunate enough to receive it.

According to Dr. Fung, the first big lie in the management of type 2 diabetes is the claim that it is a chronically progressive disease that simply gets worse with time, even in those who comply with the best treatments modern medicine offers. But, Dr. Fung argues, this is simply not true. Fifty percent of the patients on Dr. Fung's Intensive Dietary Management (IDM) program, which combines dietary carbohydrate restriction and fasting, are able to stop using insulin after a few months.

So why are we unable to acknowledge the truth? Dr. Fung's answer is simple: we doctors lie to ourselves. If type 2 diabetes is a curable disease but all our patients are getting worse on the treatments we prescribe, then we must be bad doctors. And since we did not study for so long at such great cost to become bad doctors, this failure cannot be our fault. Instead, we must believe we are doing the best for our patients, who must unfortunately be suffering from a chronically progressive and incurable disease. It is not a deliberate lie, Dr. Fung concludes, but one of cognitive dissonance—the inability to accept a blatant truth because accepting it would be too emotionally devastating.

The second lie, according to Dr. Fung, is our belief that type 2 diabetes is a disease of abnormal blood glucose levels for which the only correct treatment is progressively increasing insulin dosages. He argues, instead, that type 2 diabetes is a disease of insulin resistance with *excessive* insulin secretion—in contrast to type 1 diabetes, a condition of true insulin *lack.* To treat both conditions the same way—by injecting insulin—makes no sense. Why treat a condition of insulin excess with yet more insulin, he asks? That is the equivalent of prescribing alcohol for the treatment of alcoholism.

Dr. Fung's novel contribution is his insight that treatment in type 2 diabetes focuses on the symptom of the disease—an elevated blood glucose concentration—rather than its root cause, insulin resistance. And the initial treatment for insulin resistance is to limit carbohydrate intake. Understanding this simple biology explains why this disease may be reversible in some cases—and, conversely, why the modern treatment of type 2 diabetes, which does not limit carbohydrate intake, worsens the outcome.

But how did Dr. Fung arrive at these outrageous conclusions? And how did they lead to his authorship of this book?

In addition to his realization, described above, of the long-term nature of disease and the illogic of treating a disease's symptoms rather than

removing its cause, he also, almost by chance, in the early 2000s, became aware of the growing literature on the benefits of low-carbohydrate diets in those with obesity and other conditions of insulin resistance. Taught to believe that a carbohydrate-restricted, high-fat diet kills, he was shocked to discover the opposite: this dietary choice produces a range of highly beneficial metabolic outcomes, especially in those with the worst insulin resistance.

And finally came the cherry on the top—a legion of hidden studies showing that for the reduction of body weight in those with obesity (and insulin resistance), this high-fat diet is at least as effective, and usually much more so, than other more conventional diets.

Eventually, he could bear it no longer. If everyone knows (but won't admit) that the low-fat calorie-restricted diet is utterly ineffective in controlling body weight or in treating obesity, surely it is time to tell the truth: the best hope for treating and preventing obesity, a disease of insulin resistance and excessive insulin production, must surely be the same low-carbohydrate, high-fat diet used for the management of the ultimate disease of insulin resistance, type 2 diabetes. And so this book was born.

In *The Obesity Code,* Dr. Fung has produced perhaps the most important popular book yet published on this topic of obesity.

Its strengths are that it is based on an irrefutable biology, the evidence for which is carefully presented; and it is written with the ease and confidence of a master communicator in an accessible, well-reasoned sequence so that its consecutive chapters systematically develop, layer by layer, an evidence-based biological model of obesity that makes complete sense in its logical simplicity. It includes just enough science to convince the skeptical scientist, but not so much that it confuses those without a background in biology. This feat in itself is a stunning achievement that few science writers ever accomplish.

By the end of the book, the careful reader will understand exactly the causes of the obesity epidemic, why our attempts to prevent both the obesity and diabetes epidemics were bound to fail, and what, more importantly, are the simple steps that those with a weight problem need to take to reverse their obesity.

The solution needed is that which Dr. Fung has now provided: "Obesity is . . . a multifactorial disease. What we need is a framework, a structure, a coherent theory to understand how all its factors fit together. Too often, our current model of obesity assumes that there is only one single true cause, and that all others are pretenders to the throne. Endless debates ensue . . . They are all partially correct."

In providing one such coherent framework that can account for most of what we currently know about the real causes of obesity, Dr. Fung has provided much, much more.

He has provided a blueprint for the reversal of the greatest medical epidemics facing modern society—epidemics that he shows are entirely preventable and potentially reversible, but only if we truly understand their biological causes—not just their symptoms.

The truth he expresses will one day be acknowledged as self-evident.

The sooner that day dawns, the better for us all.

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INTRODUCTION

THE ART OF medicine is quite peculiar. Once in a while, medical treatments become established that don't really work. Through sheer inertia, these treatments get handed down from one generation of doctors to the next and survive for a surprisingly long time, despite their lack of effectiveness. Consider the medicinal use of leeches (bleeding) or, say, routine tonsillectomy.

Unfortunately, the treatment of obesity is also one such example. Obesity is defined in terms of a person's body mass index, calculated as a person's weight in kilograms divided by the square of their height in meters. A body mass index greater than 30 is defined as obese. For more than thirty years, doctors have recommended a low-fat, calorie-reduced diet as the treatment of choice for obesity. Yet the obesity epidemic accelerates. From 1985 to 2011, the prevalence of obesity in Canada tripled, from 6 percent to 18 percent.[1] This phenomenon is not unique to North America, but involves most of the nations of the world.

Virtually every person who has used caloric reduction for weight loss has failed. And, really, who hasn't tried it? By every objective measure, this treatment is completely and utterly ineffective. Yet it remains the treatment of choice, defended vigorously by nutritional authorities.

As a nephrologist, I specialize in kidney disease, the most common cause of which is type 2 diabetes with its associated obesity. I've often

watched patients start insulin treatment for their diabetes, knowing that most will gain weight. Patients are rightly concerned. “Doctor,” they say, “you’ve always told me to lose weight. But the insulin you gave me makes me gain so much weight. How is this helpful?” For a long time, I didn’t have a good answer for them.

That nagging unease grew. Like many doctors, I believed that weight gain was a caloric imbalance—eating too much and moving too little. But if that were so, why did the medication I prescribed—insulin—cause such relentless weight gain?

Everybody, health professionals and patients alike, understood that the root cause of type 2 diabetes lay in weight gain. There were rare cases of highly motivated patients who had lost significant amounts of weight. Their type 2 diabetes would also reverse course. Logically, since weight was the underlying problem, it deserved significant attention. Still, it seemed that the health profession was not even the least bit interested in treating it. I was guilty as charged. Despite having worked for more than twenty years in medicine, I found that my own nutritional knowledge was rudimentary, at best.

Treatment of this terrible disease—obesity—was left to large corporations like Weight Watchers, as well as various hucksters and charlatans mostly interested in peddling the latest weight-loss “miracle.” Doctors were not even remotely interested in nutrition. Instead, the medical profession seemed obsessed with finding and prescribing the next new drug:

- You have type 2 diabetes? Here, let me give you a pill.
- You have high blood pressure? Here, let me give you a pill.
- You have high cholesterol? Here, let me give you a pill.
- You have kidney disease? Here, let me give you a pill.

But all along, *we needed to treat obesity.* We were trying to treat the problems caused by obesity rather than obesity itself. In trying to

understand the underlying cause of obesity, I eventually established the Intensive Dietary Management Clinic in Toronto, Canada.

The conventional view of obesity as a caloric imbalance did not make sense. Caloric reduction had been prescribed for the last fifty years with startling ineffectiveness.

Reading books on nutrition was no help. That was mostly a game of "he said, she said," with many quoting "authoritative" doctors. For example, Dr. Dean Ornish says that dietary fat is bad and carbohydrates are good. He is a respected doctor, so we should listen to him. But Dr. Robert Atkins said dietary fat is good and carbohydrates are bad. He was also a respected doctor, so we should listen to him. Who is right? Who is wrong? In the science of nutrition, there is rarely any consensus about *anything*:

- Dietary fat is bad. No, dietary fat is good. There are good fats and bad fats.
- Carbohydrates are bad. No, carbohydrates are good. There are good carbs and bad carbs.
- You should eat more meals a day. No, you should eat fewer meals a day.
- Count your calories. No, calories don't count.
- Milk is good for you. No, milk is bad for you.
- Meat is good for you. No, meat is bad for you.

To discover the answers, we need to turn to evidence-based medicine rather than vague opinion.

Literally thousands of books are devoted to dieting and weight loss, usually written by doctors, nutritionists, personal trainers and other "health experts." However, with a few exceptions, rarely is more than a cursory thought spared for the actual *causes* of obesity. What *makes* us gain weight? Why do we get fat?

The major problem is the complete lack of a theoretical framework for understanding obesity. Current theories are ridiculously simplistic, often taking only one factor into account:

- Excess calories cause obesity.
- Excess carbohydrates cause obesity.
- Excess meat consumption causes obesity.
- Excess dietary fat causes obesity.
- Too little exercise causes obesity.

But all chronic diseases are multifactorial, and these factors are not mutually exclusive. They may all contribute to varying degrees. For example, heart disease has numerous contributing factors—family history, gender, smoking, diabetes, high cholesterol, high blood pressure and a lack of physical activity, to name only a few—and that fact is well accepted. But such is not the case in obesity research.

The other major barrier to understanding is the focus on short-term studies. Obesity usually takes decades to fully develop. Yet we often rely on information about it from studies that are only of several weeks' duration. If we study how rust develops, we would need to observe metal over a period of weeks to months, not hours. Obesity, similarly, is a long-term disease. Short-term studies may not be informative.

While I understand that the research is not always conclusive, I hope this book, which draws on what I've learned over twenty years of helping patients with type 2 diabetes lose weight permanently to manage their disease, will provide a structure to build upon.

Evidence-based medicine does not mean taking every piece of low-quality evidence at face value. I often read statements such as "low-fat diets proven to completely reverse heart disease." The reference will be a study of five rats. That hardly qualifies as evidence. I will reference only studies done on humans, and mostly only those that have been published in high-quality, peer-reviewed journals. No animal studies will be discussed in this book. The reason for this decision can be illustrated in "The Parable of the Cow":

Two cows were discussing the latest nutritional research, which had been done on lions. One cow says to the other, “Did you hear that we’ve been wrong these last 200 years? The latest research shows that eating grass is bad for you and eating meat is good.” So the two cows began eating meat. Shortly afterward, they got sick and they died.

One year later, two lions were discussing the latest nutritional research, which was done on cows. One lion said to the other that the latest research showed that eating meat kills you and eating grass is good. So, the two lions started eating grass, and they died.

What’s the moral of the story? We are not mice. We are not rats. We are not chimpanzees or spider monkeys. We are human beings, and therefore we should consider only human studies. I am interested in obesity in humans, not obesity in mice. As much as possible, I try to focus on causal factors rather than association studies. It is dangerous to assume that because two factors are associated, one is the cause of the other. Witness the hormone replacement therapy disaster in post-menopausal women. Hormone replacement therapy was *associated* with lower heart disease, but that did not mean that it was the *cause* of lower heart disease. However, in nutritional research, it is not always possible to avoid association studies, as they are often the best available evidence.

Part 1 of this book, “The Epidemic,” explores the timeline of the obesity epidemic and the contribution of the patient’s family history, and shows how both shed light on the underlying causes.

Part 2, “The Calorie Deception,” reviews the current caloric theory in depth, including exercise and overfeeding studies. The shortcomings of the current understanding of obesity are highlighted.

Part 3, “A New Model of Obesity,” introduces the hormonal theory of obesity, a robust explanation of obesity as a medical problem. These chapters explain the central role of insulin in regulating body weight and describe the vitally important role of insulin resistance.

Part 4, “The Social Phenomenon of Obesity,” considers how hormonal obesity theory explains some of the associations of obesity. Why is obesity associated with poverty? What can we do about childhood obesity?

Part 5, “What’s Wrong with Our Diet?,” explores the role of fat, protein and carbohydrates, the three macronutrients, in weight gain. In addition, we examine one of the main culprits in weight gain—fructose—and the effects of artificial sweeteners.

Part 6, “The Solution,” provides guidelines for lasting treatment of obesity by addressing the hormonal imbalance of high blood insulin. Dietary guidelines for reducing insulin levels include reducing added sugar and refined grains, keeping protein consumption moderate, and adding healthy fat and fiber. Intermittent fasting is an effective way to treat insulin resistance without incurring the negative effects of calorie reduction diets. Stress management and sleep improvement can reduce cortisol levels and control insulin.

The Obesity Code will set forth a framework for understanding the condition of human obesity. While obesity shares many important similarities and differences with type 2 diabetes, this is primarily a book about obesity.

The process of challenging current nutritional dogma is, at times, unsettling, but the health consequences are too important to ignore. What actually causes weight gain and what can we do about it? This question is the overall theme of this book. A fresh framework for the understanding and treatment of obesity represents a new hope for a healthier future.

JASON FUNG, MD

PART ONE

The Epidemic

(1)

HOW OBESITY BECAME AN EPIDEMIC

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Of all the parasites that affect humanity, I do not know of, nor can I imagine, any more distressing than that of Obesity.

WILLIAM BANTING

HERE'S THE QUESTION that has always bothered me: Why are there doctors who are fat? Accepted as authorities in human physiology, doctors should be true experts on the causes and treatments of obesity. Most doctors are also very hardworking and self-disciplined. Since nobody wants to be fat, doctors in particular should have both the knowledge and the dedication to stay thin and healthy.

So why are there fat doctors?

The standard prescription for weight loss is "Eat Less, Move More." It *sounds* perfectly reasonable. But why doesn't it work? Perhaps people wanting to lose weight are not following this advice. The mind is willing, but the flesh is weak. Yet consider the self-discipline and dedication needed to complete an undergraduate degree, medical school, internship, residency

and fellowship. It is hardly conceivable that overweight doctors simply lack the willpower to follow their own advice.

This leaves the possibility that the conventional advice is simply wrong. And if it is, then our entire understanding of obesity is fundamentally flawed. Given the current epidemic of obesity, I suspect that such is the most likely scenario. So we need to start at the very beginning, with a thorough understanding of the disease that is human obesity.

We must start with the single most important question regarding obesity or any disease: "What causes it?" We spend no time considering this crucial question because we think we already know the answer. It seems so obvious: it's a matter of Calories In versus Calories Out.

A calorie is a unit of food energy used by the body for various functions such as breathing, building new muscle and bone, pumping blood and other metabolic tasks. Some food energy is stored as fat. Calories In is the food energy that we eat. Calories Out is the energy expended for all of these various metabolic functions.

When the number of calories we take in exceeds the number of calories we burn, weight gain results, we say. Eating too much and exercising too little causes weight gain, we say. Eating too many *calories* causes weight gain, we say. These "truths" seem so self-evident that we do not question whether they are actually true. But are they?

PROXIMATE VERSUS ULTIMATE CAUSE

EXCESS CALORIES MAY certainly be the *proximate* cause of weight gain, but not its *ultimate* cause.

What's the difference between proximate and ultimate? The proximate cause is *immediately* responsible, whereas the ultimate cause is what started the chain of events.

Consider alcoholism. What causes alcoholism? The proximate cause is "drinking too much alcohol"—which is undeniably true, but not particularly

useful. The question and the cause here are one and the same, since alcoholism *means* "drinking too much alcohol." Treatment advice directed against the proximate cause—"Stop drinking so much alcohol"—is not useful.

The crucial question, the one that we are really interested in, is: What is the *ultimate* cause of *why* alcoholism occurs. The ultimate cause includes

- the addictive nature of alcohol,
- any family history of alcoholism,
- excessive stress in the home situation and/or
- an addictive personality.

There we have the real disease, and treatment must be directed against the ultimate, rather than the proximate cause. Understanding the ultimate cause leads to effective treatments such as (in this case) rehabilitation and social support networks.

Let's take another example. Why does a plane crash? The proximate cause is, "there was not enough lift to overcome gravity"—again, absolutely true, but not in any way useful. The ultimate cause might be

- human error,
- mechanical fault and/or
- inclement weather.

Understanding the ultimate cause leads to effective solutions such as better pilot training or tighter maintenance schedules. Advice to "generate more lift than gravity" (larger wings, more powerful engines) will not reduce plane crashes.

This understanding applies to everything. For instance, why is it so hot in this room?

PROXIMATE CAUSE: Heat energy coming in is greater than heat energy leaving.

SOLUTION: Turn on the fans to increase the amount of heat leaving.

ULTIMATE CAUSE: The thermostat is set too high.

SOLUTION: Turn down the thermostat.

Why is the boat sinking?

PROXIMATE CAUSE: Gravity is stronger than buoyancy.

SOLUTION: Reduce gravity by lightening the boat.

ULTIMATE CAUSE: The boat has a large hole in the hull.

SOLUTION: Patch the hole.

In each case, the solution to the proximate cause of the problem is neither lasting nor meaningful. By contrast, treatment of the ultimate cause is far more successful.

The same applies to obesity: What causes weight gain?

Proximate cause: Consuming more calories than you expend.

If more calories in than out is the proximate cause, the unspoken answer to that last question is that the ultimate cause is "personal choice." We *choose* to eat chips instead of broccoli. We *choose* to watch TV instead of exercise. Through this reasoning, obesity is transformed from a disease that needs to be investigated and understood into a personal failing, a character defect. Instead of searching for the ultimate cause of obesity, we transform the problem into

- eating too much (gluttony) and/or
- exercising too little (sloth).

Gluttony and sloth are two of the seven deadly sins. So we say of the obese that they "brought it on themselves." They "let themselves go." It gives us the comforting illusion that we understand ultimate cause of the problem. In a 2012 online poll,[1] 61 percent of U.S. adults believed that "personal choices about eating and exercise" were responsible for the obesity epidemic. So we discriminate against people who are obese. We both pity and loathe them.

However, on simple reflection, this idea simply cannot be true. Prior to puberty, boys and girls average the same body-fat percentage. After puberty, women on average carry close to 50 percent more body fat than men. This

change occurs despite the fact that men consume more calories on average than women. But why is this true?

What is the ultimate cause? It has nothing to do with personal choices. It is not a character defect. Women are not more gluttonous or lazier than men. The hormonal cocktail that differentiates men and women must make it more likely that women will accumulate excess calories as fat as opposed to burning them off.

Pregnancy also induces significant weight gain. What is the ultimate cause? Again, it is obviously the hormonal changes resulting from the pregnancy—*not* personal choice—that encourages weight gain.

Having erred in understanding the proximate and ultimate causes, we believe the solution to obesity is to eat fewer calories.

The "authorities" all agree. The U.S. Department of Agriculture's *Dietary Guidelines for Americans,* updated in 2010, forcefully proclaims its key recommendation: "Control total calorie intake to manage body weight." The Centers for Disease Control[2] exhort patients to balance their calories. The advice from the National Institutes of Health's pamphlet "Aim for a Healthy Weight" is "to cut down on the number of calories . . . they get from food and beverages and increase their physical activity."[3]

All this advice forms the famous "Eat Less, Move More" strategy so beloved by obesity "experts." But here's a peculiar thought: If we already understand what causes obesity, how to treat it, and we've spent millions of dollars on education and obesity programs, *why are we getting fatter?*

ANATOMY OF AN EPIDEMIC

WE WEREN'T ALWAYS so obsessed with calories. Throughout most of human history, obesity has been rare. Individuals in traditional societies eating traditional diets seldom became obese, even in times of abundant food. As civilizations developed, obesity followed. Speculating on the cause, many identified the refined carbohydrates of sugar and starches. Sometimes

considered the father of the low-carbohydrate diet, Jean Anthelme Brillat-Savarin (1755–1826) wrote the influential textbook *The Physiology of Taste* in 1825. There he wrote: "The second of the chief causes of obesity is the *floury and starchy substances* which man makes the prime ingredients of his daily nourishment. As we have said already, all animals that live on farinaceous food grow fat willy-nilly; and man is no exception to the universal law."[4]

All foods can be divided into three different macronutrient groups: fat, protein and carbohydrates. The "macro" in "macronutrients" refers to the fact that the bulk of the food we eat is made up of these three groups. Micronutrients, which make up a very small proportion of the food, include vitamins and minerals such as vitamins A, B, C, D, E and K, as well as minerals such as iron and calcium. Starchy foods and sugars are all carbohydrates.

Several decades later, William Banting (1796–1878), an English undertaker, rediscovered the fattening properties of the refined carbohydrate. In 1863, he published the pamphlet *Letter on Corpulence, Addressed to the Public,* which is often considered the world's first diet book. His story is rather unremarkable. He was not an obese child, nor did he have a family history of obesity. In his mid-thirties, however, he started to gain weight. Not much; perhaps a pound or two per year. By age sixty-two, he stood five foot five and weighed 202 pounds (92 kilograms). Perhaps unremarkable by modern standards, he was considered quite portly at the time. Distressed, he sought advice on weight loss from his physicians.

First, he tried to eat less, but that only left him hungry. Worse, he failed to lose weight. Next, he increased his exercise by rowing along the River Thames, near his home in London. While his physical fitness improved, he developed a "prodigious appetite, which I was compelled to indulge."[5] Still, he failed to lose weight.

Finally, on the advice of his surgeon, Banting tried a new approach. With the idea that sugary and starchy foods were fattening, he strenuously avoided all breads, milk, beer, sweets and potatoes that had previously made up a large portion of his diet. (Today we would call this diet low in refined carbohydrates.) William Banting not only lost the weight and kept it off, but he also felt so well that he was compelled to write his famous pamphlet. Weight gain, he believed, resulted from eating too many "fattening carbohydrates."

For most of the next century, diets low in refined carbohydrates were accepted as the standard treatment for obesity. By the 1950s, it was fairly standard advice. If you were to ask your grandparents what caused obesity, they would not talk about calories. Instead, they would tell you to stop eating sugary and starchy foods. Common sense and empiric observation served to confirm the truth. Nutritional "experts" and government opinion were not needed.

Calorie counting had begun in the early 1900s with the book *Eat Your Way to Health,* written by Dr. Robert Hugh Rose as a "scientific system of weight control." That book was followed up in 1918 with the bestseller *Diet and Health, with Key to the Calories,* written by Dr. Lulu Hunt Peters, an American doctor and newspaper columnist. Herbert Hoover, then the head of the U.S. Food Administration, converted to calorie counting. Dr. Peters advised patients to start with a fast, one to two days abstaining from all foods, and then stick strictly to 1200 calories per day. While the advice to fast was quickly forgotten, modern calorie-counting schedules are not very different.

By the 1950s, a perceived "great epidemic" of heart disease was becoming an increasing public concern. Seemingly healthy Americans were developing heart attacks with growing regularity. In hindsight, it should have been obvious that there was really no such epidemic.

The discovery of vaccines and antibiotics, combined with increased public sanitation, had reshaped the medical landscape. Formerly lethal infections, such as pneumonia, tuberculosis and gastrointestinal infections, became curable. Heart disease and cancer now caused a relatively greater percentage of deaths, giving rise to some of the public misperception of an epidemic. (See Figure 1.1.[6])

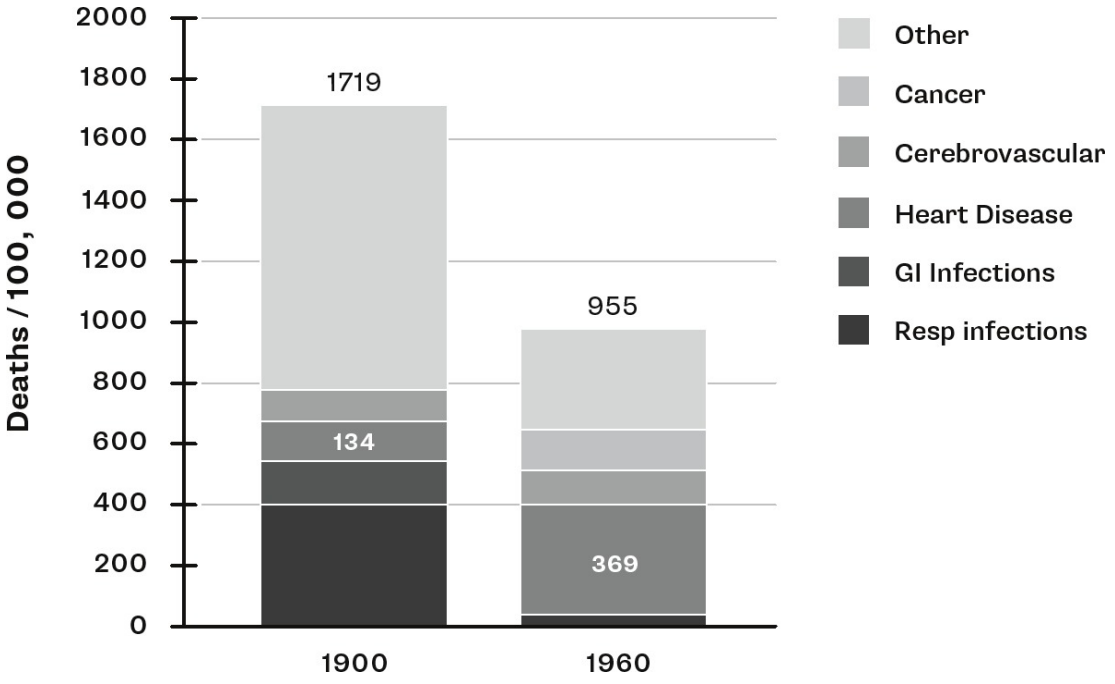

Figure 1.1. Causes of death in the United States 1900 vs. 1960.

The increase in life expectancy from 1900 to 1950 reinforced the perception of a coronary-disease epidemic. For a white male, the life expectancy in 1900 was fifty years.[7] By 1950, it had reached sixty-six years, and by 1970, almost sixty-eight years. If people were not dying of tuberculosis, then they would live long enough to develop their heart attack. Currently, the average age at first heart attack is sixty-six years.[8] The risk of a heart attack in a fifty-year-old man is substantially lower than in a sixty-eight-year-old man. So the natural consequence of a longer life expectancy is an increased rate of coronary disease.

But all great stories need a villain, and dietary fat was cast into that role. Dietary fat was thought to increase the amount of cholesterol, a fatty substance that is thought to contribute to heart disease, in the blood. Soon, physicians began to advocate lower-fat diets. With great enthusiasm and shaky science, the demonization of dietary fat began in earnest.

There was a problem, though we didn't see it at the time. The three macronutrients are fat, protein and carbohydrates: lowering dietary fat meant replacing it with either protein or carbohydrates. Since many high-protein foods like meat and dairy are also high in fat, it is difficult to lower fat in the diet without lowering protein as well.

So, if one were to restrict dietary fats, then one must increase dietary carbohydrates and vice versa. In the developed world, these carbohydrates all tend to be highly refined.

Low Fat = High Carbohydrate

This dilemma created significant cognitive dissonance. Refined carbohydrates could not simultaneously be both good (because they are low in fat) and bad (because they are fattening). The solution adopted by most nutrition experts was to suggest that *carbohydrates* were no longer fattening. Instead, *calories* were fattening. Without evidence or historical precedent, it was arbitrarily decided that excess *calories* caused weight gain, not specific foods. Fat, as the dietary villain, was now deemed fattening—a previously unknown concept. The Calories-In/Calories-Out model began to displace the prevailing "fattening carbohydrates" model.

But not everybody bought in. One of the most famous dissidents was the prominent British nutritionist John Yudkin (1910–1995). Studying diet and heart disease, he found no relationship between dietary fat and heart disease. He believed that the main culprit of both obesity and heart disease was sugar.[9, 10] His 1972 book, *Pure, White and Deadly: How Sugar Is Killing Us,* is eerily prescient (and should certainly win the award for Best

Book Title Ever). Scientific debate raged back and forth about whether the culprit was dietary fat or sugar.

THE DIETARY GUIDELINES

THE ISSUE WAS finally settled in 1977, not by scientific debate and discovery, but by governmental decree. George McGovern, then chairman of the United States Senate Select Committee on Nutrition and Human Needs, convened a tribunal, and after several days of deliberation, it was decided that henceforth, dietary fat was guilty as charged. Not only was dietary fat guilty of causing heart disease, but it also caused *obesity,* since fat is calorically dense.

The resulting declaration became the *Dietary Goals for the United States.* An entire nation, and soon the entire world, would now follow nutritional advice from a politician. This was a remarkable break from tradition. For the first time, a government institution intruded into the kitchens of America. Mom used to tell us what we should and should not eat. But from now on, Big Brother would be telling us. And he said, "Eat less fat and more carbohydrates."

Several specific dietary goals were set forth. These included

- raise consumption of carbohydrates until they constituted 55 percent to 60 percent of calories, and
- decrease fat consumption from approximately 40 percent of calories to 30 percent, of which no more than one-third should come from saturated fat.

With no scientific evidence, the formerly "fattening" carbohydrate made a stunning transformation. While the guidelines still recognized the evils of sugar, refined grain was as innocent as a nun in a convent. Its nutritional sins were exonerated, and it was henceforth reborn and baptized as the healthy whole grain.

Was there any evidence? It hardly mattered. The goals were now the nutritional orthodoxy. Everything else was heathen. If you didn't toe the line, you were ridiculed. The *Dietary Guidelines for Americans*, a report released in 1980 for widespread public consumption, followed the recommendations of the McGovern report closely. The nutritional landscape of the world was forever changed.

The *Dietary Guidelines for Americans,* now updated every five years, spawned the infamous food pyramid in all its counterfactual glory. The foods that formed the base of the pyramid—*the foods we should eat every single day*—were breads, pastas and potatoes. These were the precise foods that we had previously avoided to stay thin. For example, the American Heart Association's 1995 pamphlet, *The American Heart Association Diet: An Eating Plan for Healthy Americans*, declared we should eat six or more servings of "breads, cereals, pasta and starchy vegetables (that) are low in fat and cholesterol." To drink, "Choose . . . fruit punches, carbonated soft drinks." Ahhh. White bread and carbonated soft drinks—the dinner of champions. Thank you, American Heart Association (AHA).

Entering this brave new world, Americans tried to comply with the nutritional authorities of the day and made a conscious effort to eat less fat, less red meat, fewer eggs and more carbohydrates. When doctors advised people to stop smoking, rates dropped from 33 percent in 1979 to 25 percent by 1994. When doctors said to control blood pressure and cholesterol, there was a 40 percent decline in high blood pressure and a 28 percent decline in high cholesterol. When the AHA told us to eat more bread and drink more juice, we ate more bread and drank more juice.

Inevitably, sugar consumption increased. From 1820 to 1920, new sugar plantations in the Caribbean and American South increased the availability of sugar in the U.S. Sugar intake plateaued from 1920 to 1977. Even though "avoid too much sugar" was an explicit goal of the 1977 *Dietary Guidelines for Americans,* consumption increased anyway until the year 2000. With all

our attention focused on fat, we took our eyes off the ball. Everything was "low fat" or "low cholesterol," and nobody was paying attention to sugar. Food processors, figuring this out, increased the added sugars in processed food for flavor.

Refined grain consumption increased by almost 45 percent. Since carbohydrates in North America tended to be refined, we ate more and more low-fat bread and pasta, not cauliflower and kale.[11]

Success! From 1976 to 1996, the average fat intake decreased from 45 percent of calories to 35 percent. Butter consumption decreased 38 percent. Animal protein decreased 13 percent. Egg consumption decreased 18 percent. Grains and sugars increased.

Until that point, the widespread adoption of the low-fat diet was completely untested. We had no idea what effect it would have on human health. But we had the fatal conceit that we were somehow smarter than 200,000 years of Mother Nature. So, turning away from the natural fats, we embraced refined low-fat carbohydrates such as bread and pasta. Ironically, the American Heart Association, even as late as the year 2000, felt that low-carbohydrate diets were dangerous fads, despite the fact that these diets had been in use almost continuously since 1863.

What was the result? The incidence of heart disease certainly did not decrease as expected. But there was definitely a consequence to this dietary manipulation—an unintentional one. Rates of obesity, defined as having a body mass index greater than 30, dramatically *increased,* starting almost exactly in 1977, as illustrated by Figure 1.2.[12]

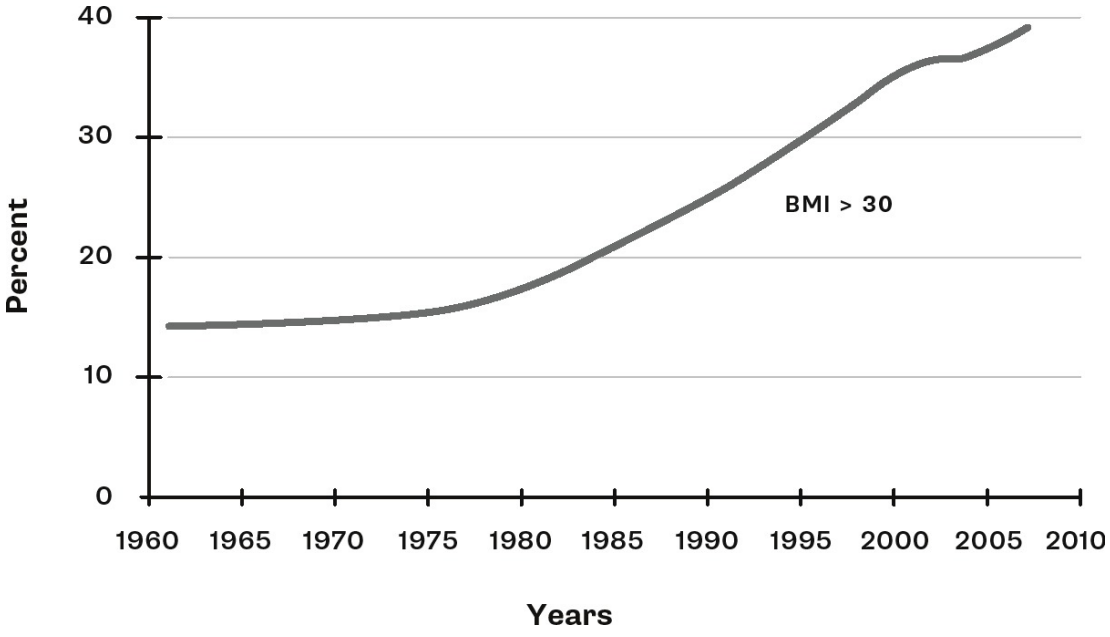

Figure 1.2. Increase in obese and extremely obese United States adults aged 20–74.

The abrupt increase in obesity began exactly with the officially sanctioned move toward a low-fat, high-carbohydrate diet. Was it mere coincidence? Perhaps the fault lay in our genetic makeup instead.