FAT POLITICS
The Real Story Behind America’s Obesity Epidemic

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Introduction: A Big, Fat Problem

Over the past two decades, a plague has been sighted in our midst. It is said to afflict one in four Americans and kill as many as 400,000 of us every year. It is purported to cause heart disease, cancer, diabetes, asthma, hypertension, and numerous other ailments. It is estimated to cost us 100 billion dollars annually in healthcare expenses and, according to some, threatens to overwhelm our medical infrastructure. Surgeon General Richard Carmona says it’s a greater threat than terrorism and former health and human services secretary Tommy Thompson has named it a public health “crisis.” Congress and numerous state governments are allocating billions of dollars in search of a cure while the media and health organizations regularly sound the alarms.

The “disease” I’m referring to is obesity and in the United States it has become, by most accounts, a full-scale epidemic. This certainly seems to be borne out by the statistics: in 1980, only about a third of Americans were considered overweight and only 13 percent were classified as obese, rates not much greater than in 1960. But in the past twenty-five years these numbers have skyrocketed. Today, more than 60 percent of Americans are considered overweight and one in four is obese—a two-fold increase in less than three decades. Even more alarming is the rise in juvenile obesity; today, 15 percent of American children are considered obese, more than twice as many as in 1980. As a result of their weight, today’s teenagers will be, according to some projections, the first generation in modern American history to live a shorter life span than their parents.¹

America, it seems, has a big, fat problem.

Or does it? This is what I thought when I started writing this book. Like many people, I, too, believed that America’s growing weight was a genuine health quandary. Indeed, my initial plan for this book was to look at why we were gaining so much weight and what we could do to stop it. But then I started to examine the evidence and a funny thing happened—the more I read, the more I realized how misguided my initial assumptions about obesity were.² While it was true that Americans were getting heavier, it was less obvious that this was putting them in mortal danger or even that it was causing ailments such as diabetes, heart disease, and cancer. Like many headline-grabbing issues,
the truth behind America’s “obesity epidemic” was far different than the story I once believed.

What I came to realize was that, contrary to the conventional wisdom, obesity is not a problem because more than 60 percent of Americans weigh “too much.” Nor is it a problem because hundreds of thousands are dying from being too fat. Nor is it a problem because it costs us hundreds of billions in healthcare expenditures. Obesity is not a problem for any of these reasons because none of them are true. While Americans do face many health challenges, few of these arise from our increasing weight. Our growing weight is merely a symptom of some fundamental changes in our diet and exercise patterns which may (or may not) affect our health. There is, however, little evidence that obesity itself is a primary cause of our health woes. In other words, telling most Americans they need to worry about their weight is like telling someone dying of pneumonia that they need to worry about how much they are coughing; it conflates the real source of our health problems with a relatively benign symptom.

Now, understandably, you might view these claims with some skepticism. After all, there is no denying that America is a very fat country. One need only take a stroll through any airport or shopping mall to witness the ample size of our population. And our fatness does carry some genuine health consequences—being very heavy puts more stress on one’s joints and makes it harder to exercise, which is very important for one’s health. Then there is all the information we get in the media about obesity’s dangers—hardly a week goes by without some new story about another health problem that purportedly comes from being too fat. From fashion magazines to television shows such as The Biggest Loser, we are surrounded by messages that fatness is not only unhealthy, but also unsightly and immoral. And who among us doesn’t worry, at some time, about weight too much? Considering all our anxiety about our appearance and weight, it is perfectly logical to assume that our growing obesity portends a national health catastrophe. But if you suspend all these preconceptions, at least for a moment, and look briefly at the scientific evidence, you’ll see a much different picture.

Let us start with the much-cited claim that obesity is a major killer. In April 2004, researchers from the Centers for Disease Control and Prevention (CDC) released a report in the prestigious Journal of the American Medical Association estimating that obesity
was killing 400,000 Americans a year.iii Given that *JAMA*, as the journal is known, is the most prestigious medical journal in the country and that one of the article’s authors was the head of the CDC, this estimate had all the trappings of official truth. Soon afterward, public health officials began to use these numbers in press releases, reports, and in congressional testimony as evidence that obesity was a major threat. Scientists repeatedly cited them as a justification for further research funding to address this important problem. Newspaper headlines across the country trumpeted how obesity would soon overtake smoking as the number one cause of preventable death in America.

A closer look at the numbers, however, shows that they don’t add up. Partly, this was because the CDC researchers did not calculate the 400,000 deaths by checking to see if the weight of each person was a factor in his or her death; rather they estimated a figure by comparing the death rates of thin and heavy people using data that were nearly 30 years old. Although heavier people tend to die more frequently than people in mid-range weights, it is by no means clear that their weight is the cause of their higher death rates. It is far more likely that weight is simply a proxy for other, more important factors such as their diet, exercise, or family medical history. The researchers, however, simply assumed that obesity was the primary cause of death, even though there was no clear scientific rationale for this supposition.iv Moreover, they also made a number of errors in their basic calculations. When these facts came out (only after a congressionally initiated inquiry), the CDC was forced to conduct an internal investigation and ended up amending the report.

In fact, a 2005 study, also published in *JAMA*, by a different set of CDC researchers, offers entirely different mortality estimates. Rather than being in peril, it appears that moderately “overweight” people live longer than those at a “normal” weight. And instead of causing 400,000 deaths, the new study estimates that fewer than 26,000 Americans die each year from weighing too much, a number that is even smaller than those who are estimated to die from being “underweight.”v Weighing “too much” is less dangerous, it seems, than weighing “too little.”

A similar problem exists with the links between obesity and most diseases. Doctors, government health agencies, and the news media frequently warn of the connections between being too fat and various ailments. *Time* magazine, for example,
recently proclaimed that, “being overweight significantly increases the risk of a long list of medical complaints including coronary artery disease, congestive heart failure, hypertension, diabetes, depression, deep-vein thrombosis, fatigue, insomnia, indigestion, and impotence.” After reading such a long indictment, it would be quite natural to assume that being fat puts your health in grave danger. However, this, too, is a misperception. Obesity has not been found to be a primary cause of any of these conditions. Yes, heart disease, diabetes, and other ailments are more common among the obese than the nonobese, but there is little evidence that adiposity (that is, excess fat tissue) is producing these pathologies. Indeed, some types of body fat are actually protective against many diseases, particularly for women and people over 65. Nor is there clear evidence that, by itself, weight loss reduces the risk of death or most diseases. In other words, not only do we have lack proof that being fat causes us to contract most major illnesses, we do not have any evidence that losing weight makes us any healthier.

The same situation holds for the prognostications about how obesity is costing Americans so much money. The researchers who estimated that obesity costs us 100 billion dollars a year did so by calculating all the expenses associated with treating type 2 diabetes, coronary heart disease, hypertension, gallbladder disease, and cancer, but, like the estimators of deaths, they did not take into account other factors such as diet, exercise, or genetics that also might be causing these conditions. Once again, they simply assumed that if you got heart disease or breast cancer it was because you were fat.

In short, nearly all the warnings about obesity are based on little more than loose statistical conjecture. While heart disease, cancer, stroke, asthma, and diabetes are undoubtedly serious and costly health concerns, there is no convincing evidence that such ailments arise from our growing weight. In many respects, body weight is no different than any dozens of other physical traits, such as height, age, sex, skin color, and even left-handedness that can be associated with higher rates of death and disease. Based on our current evidence, blaming obesity for heart disease, cancer, or many other ailments is like blaming smelly clothes, yellow teeth, or bad breath for lung cancer instead of cigarettes; it conflates an associated trait with its underlying cause.

Given these facts, I soon found myself trying to answer a much different set of questions. Rather than trying to figure out how to “solve” the obesity problem, I realized
that the more interesting question was why so many smart and seemingly well-intentioned people were claiming that obesity was a problem in the first place. Why was our growing weight being labeled an “obesity epidemic”? What does it mean to judge our health and well-being by how much we weigh? And why were we really gaining weight? The answers to these questions came as a great surprise.

The Real Sources of the “Obesity Epidemic”

What I came to discover was that, contrary to the conventional wisdom, the primary source of America’s obesity epidemic is not to be found at McDonald’s, Burger King, or Krispe Kreme Donuts. Nor is it in how little we exercise, our declining smoking rates, a “fat virus,” or any of the other theories that are often used to explaining our rising weights. Rather, America’s obesity epidemic originates in far less conspicuous sources.

The most important of these is America’s public health establishment. Over the past two decades, a handful of scientists, doctors, and health officials have actively campaigned to define our growing weight as an “obesity epidemic.” They have created a very low and arbitrary definition of what is “overweight” and “obese” so that tens of millions of Americans, including archetypes of fitness such as President George Bush or basketball star Michael Jordan, are now considered to “weigh too much.” They have also inflated the dangers and distorted the statistics about weight and health, exaggerated the impact of obesity on everything from motor accidents to air pollution. And, most important, they have established body weight as a barometer of wellness, so that being thin is equated with being healthy.

Now some of this campaign has been motivated by good intentions—for those who are sincerely concerned with Americans’ health, the “obesity epidemic” seems like an effective way to highlight the chronic problems with Americans’ poor diet and lack of exercise. And some sincerely believe that having too much fat leads harm and disease, even if the scientific evidence is inconclusive.

But, much of this campaign is driven by less altruistic concerns and more by the particular interests among the various constituent groups within America’s public health establishment. Consider, for example, what an obesity epidemic means for the following groups. For scientists researching issues of weight, an obesity epidemic inflates their
stature and allows them to get more research grants. For government health agencies, it is a powerful rationale for increasing their programs and budget allocations. For weight-loss companies and surgeons, it is a way to get their services covered by Medicare and health insurance providers. And, for pharmaceutical companies it can justify the release of new drugs and help inflate their stock prices. The very same people who have proclaimed that obesity is a major health problem also stand the most to gain from it being classified as a disease. For America’s public health establishment, an obesity epidemic is worth billions.

Of course, obesity researchers, doctors, and drug companies are not solely to blame. Another reason why our growing weight has come to be viewed as an “obesity epidemic” is because of our cultural biases against body fat and fat people. It is common for white, middle-class Americans, in particular, to think of weight as a barometer of a person’s character—if they are fat, it is only because they are too lazy or irresponsible to “take care” of themselves. With such a strong moral connotation, body weight has become one of our most potent markers of social status whereby those with the resources or wherewithal to keep themselves thin rightly deserve their place at the top of social ladder.

Given all the negative associations we heap on our fatness, it is no wonder our growing weight is seen as a cause for alarm across the political spectrum. For some on the right, the obesity epidemic merely reinforces their beliefs about the cause of the ever-widening gap between the rich and poor or between whites and minorities. After all, if African Americans, Latinos, or the poor are becoming fatter than America’s predominantly white elite, it is only more proof that they lack the responsibility to take care of themselves. For others, our growing weight highlights the precarious social position of the middle class in an ever-stratifying America—if middle-class Americans, particularly middle-class children are getting fat, it surely indicates the frailty of their own class status. And for those on the left, the growth of obesity as further proof that large, multi-national corporations are running amok, fattening a hapless public with their billion-dollar advertising campaigns and super size value meals. The American people, they argue, need to be protected from these corporate behemoths, particularly the poor and minorities who have the highest obesity rates.
Running throughout all of these perspectives is a paternalistic condescension towards fatness and fat people – not only do they assume that fatness is inherently bad, they also presuppose that fat people (i.e., minorities and the poor) are too ignorant to know that they should be thin. Thus when the filmmaker Morgan Spurlock sneers at the black kids who actually like McDonald’s in his film *Super Size Me* or the writer Greg Critser derides the Latinos at his local donut shop in his book *Fat Land*, it is not simply because they are worried about America’s moral decline or unbridled corporate power. For many people, trumpeting the “problem of obesity” is an opportunity for them to express both their own moral superiority and their latent class snobbery and racism.

Yet it is just these kinds of biases about fatness and its origins that are dictating how obesity is being portrayed as a health issue. And this is creating all kinds of inappropriate and ineffective suggestions and responses for dealing with the “obesity epidemic.” Consider, for example, the contradictory policies coming out of Washington and various state legislatures. Either they assume that body weight is solely a matter of individual responsibility, as with the Personal Responsibility in Food Consumption Act, a congressional bill that protects fast-food chains from lawsuits, or they assume that obesity needs to be tackled by reigning in the power of McDonald’s, Coca-Cola, and other food companies as with the proposed restrictions on food advertisements and taxes on snack foods. As a result, we now have a curious combination of laws that simultaneously forbid people from suing restaurants for making them fat but also prohibit schools from selling sodas and snack foods because they allegedly cause our kids to be too fat.

Neither of these approaches, however, will do much either to improve our health or strengthen our moral fiber because they mistakenly equate fatness with both illness and depravity. By calling our growing weight an epidemic disease, America’s public health establishment is sending a message that we can be healthy (and righteous) by being thin. Not only is such a message inaccurate (weight loss has not, by itself, been shown to improve most people’s health or reduce the risk of most diseases), it ignores the real threats to our health of which our weight is only symptomatic. If we want to know why diabetes and other diseases are on the rise in the United States, we need to focus less on the mere fact that our weight is increasing and more on the question of why our weight
is increasing. In other words, we need to listen to what our growing weight is trying to
tell us.

Biological Responses to the American Way of Life

From a biological perspective, fatness is simply a protective mechanism against
an irregular food supply. Our fat cells are the places where our bodies store energy for
times when food is unavailable or when we are too busy or active to eat. Because fatness
is so crucial for our survival, our bodies have numerous means for ensuring that we retain
as many calories as possible, such as giving us an appetite for calorie-rich food and
regulating our metabolism to keep our weight within a certain range. While some people
have a metabolism that keeps them thin, many Americans are inclined to have a weight
range that tends toward corpulence. This is particularly the case for people whose
ancestors came from places, such as Africa and parts of Asia, the Pacific Islands, and the
Americas where a regular food supply was not always present. These same metabolic
 protections are also why it is so difficult for most people to lose weight—most of us are
biologically programmed to operate as if a famine is imminent.

Although our bodies may be expecting another famine, our way of life floods us
with an abundance of foods, particularly sugars and fats, and allows us to expend little
energy at work or in household chores. When our biological safeguards against privation
come into contact with an environment of abundance and leisure, it is not surprising that
many consequences ensue. Not only do we gain weight, but our cholesterol levels
change, our insulin levels rise, and our blood pressure increases.

And it is these other metabolic changes that are behind many of the diseases that
are typically associated with being too fat. The reason that diabetes and some types of
cancer are on the rise is not because Americans weigh too much, it is because their
metabolisms are out of whack. Fatness may result from the metabolic processes that are
behind these ailments, but it is the underlying metabolic processes, and not the weight,
that cause us so much trouble. So if we want to know the real health challenges behind
our growing weight, we need to identify what is causing us to eat so many fats and sugars
and to exercise so little.
Here, again, is where political ideology and cultural stereotypes often cloud our perceptions. Many folks, for example, like to blame our gluttony on the extra-large portion sizes in restaurants and supermarkets—and with a super size value meal weighing in at 1,500 calories it is easy to see why fast-food has been seen as such a villain. But, once again, this stereotype is inaccurate. Americans are not consuming more calories because of how much they are eating during their meals. (Americans consume only slightly more calories in their meals today than they did in the 1970s.) Rather, Americans are consuming more calories because of how much they are eating *in between* their meals. The real culprit behind our increasing calories is snacking.

Similarly, many public health advocates and urban planners like to blame our inactivity on cars, sprawl, and television. Given the four hours the average American spends each day behind the wheel or in front of the TV, this suspicion, too, is understandable. If we want to get Americans exercising more, it seems, we need to figure out ways to get them to drive less, watch less television, and redesign their communities to make walking easier.

Once again, however, focusing on driving and television in order to make Americans thinner misses the real important health issue. For the problem with both our unhealthy diet and inactivity goes beyond how much junk food we eat, TV we watch, or miles we drive; the problem is with the very principles that define us as a society.

Snacking, driving, and television are more than simple conveniences; they are expressions of our very core values—choice, freedom, and liberty. Snacks, sodas, and other prepared foods have liberated the American meal away from the domestic confines of the home, allowing us to eat by ourselves, when and where we want. Automobiles reduce the physical demands of walking and give us near-limitless geographic mobility. Television allows leisure to be spent with little physical effort and provides a terrific arrange of entertainment choices. In short, each is about fulfilling our wants in as efficient and easy a manner as possible, the very benefits that our liberal, free market system promises.

So, from this perspective, the origins of America’s growing metabolic problems, as well as its increasing weight, ultimately derive from its very core principles. The American credo of “life, liberty, and the pursuit of happiness” is about giving us the
freedom to individually pursue our own gratification to the extent that we see fit, the very thing that snacks, cars, and television provide. It is about making us the ultimate arbiters of what is good for ourselves. The market, in turn, responds by providing us what we want in the most efficient and inexpensive manner. Thus if we want to eat tasty foods, move about with great speed and ease, and amuse ourselves in leisurely ways, this is our right. This is why all the public health pronouncements about dieting and nutrition, such as “eat less and exercise more,” are so ineffective. Although such simple advice may seem reasonable, it flies in the face of a consumer economy that is constantly expanding our choices and freedom. Asking an American to “eat less and exercise more” is like asking an Eskimo not to fish or a devout Muslim not to say daily prayers—it runs afoul of the dominant logic of our very culture. Our growing weight is simply a natural and inevitable biological response to living in a consumer-oriented, free market democracy.

Why Focusing on Fat May Be Harmful to Your Health

Yet, with respect to our appearance and our health, this freedom is presenting us with a dilemma. On one hand, the amenities that make our lives so easy are ill suited to our physiology, particularly in large doses. The same consumer conveniences that are adding to our waistlines are also contributing to our chronically high levels of diabetes, cardiovascular disease, anxiety, stress, depression, and sleeplessness. While undoubtedly the rise of such maladies is also coming from a medical culture that is quick to diagnose any ailment as a disorder, the prevalence of such chronic diseases is also the by-product of a fast-paced culture of instant gratification and individual license. On the other hand, few Americans want to relinquish their freedom, particularly with respect to what they eat or how they move. Few of us want to ban our snack foods, raise our gasoline prices, get rid of our televisions, or forcibly be put on any diet plan. When it comes to our diet, lifestyle, and health, we basically want to eat our cake and have it too.

The easiest way of resolving this cake-eating dilemma is by simply treating its undesirable side effects. So, if snacking and inactivity are making our cholesterol level too high, we jump at the chance to keep it in check with a pill such as Lipitor, rather than force ourselves to change our diet or start exercising more. And, to the extent that we are able to treat these unwanted symptoms, such a strategy is a great success. For example,
much of the recent decline in deaths from heart disease and other ailments, as well as our improved quality of life, have come from simple and undemanding treatments. Sometimes, alleviating unwanted symptoms of an unhealthy lifestyle is the most efficient and effective response.

Often, however, treating a symptom rather than addressing its root causes only causes more problems than it solves, and this is what is happening with weight. Ask any of the millions of frustrated dieters in America and they will tell you what molecular biologists have long known—for many of us, our bodies are quite resistant to being slender. Nor do we have a safe or effective mechanism for helping us lose weight. Indeed, the same doctors, health officials, and medical researchers who have spent the past four decades telling Americans they are too fat have not been able to devise a sound treatment for becoming thin. As a result, many Americans are going to extreme measures to make themselves lose weight, such as self-starvation, smoking, taking dangerous appetite suppressants, or even having their stomachs surgically shrunk. Not only are such practices often ineffective, they often do more harm than good. Whether it is from a failed diet, a botched gastric-bypass surgery, complications from an eating disorder, or heart damage from diet drugs, every year thousands of Americans are literally dying to be thin.

Thus, with respect to our weight, we have put ourselves into a bind. In calling our growing weight an “epidemic,” we have created a disease out of a physical symptom that, in turn, we are unable to treat. In calling fat people gluttonous and lazy, we are ascribing moral characteristics to what is largely a biological phenomenon. We are now being told to lose weight without understanding that our fatness is actually an expression of forces that are largely beyond our individual control or our collective will to change. From the misguided equation of thinness and health, millions of Americans are being told to lose weight, which is only likely to make them more miserable and possibly do them great harm.

In truth, the only way we are going to “solve” the problem of obesity is to stop making fatness a scapegoat for all our ills. This means that public health officials and doctors need to stop making weight a barometer of health and issuing so many alarmist
claims about the obesity epidemic. This also means that the rest of us need to stop judging others and ourselves by our size.

Such a change in perspective, however, may be our greatest challenge. Our body weight and fatness is a uniquely powerful symbol for us – something we feel we should be able to control but that often we can’t. As a result, obesity has become akin to a sacrificial animal, a receptacle for many of our problems. Whether it is our moral indignation, status anxiety, or just feelings of general powerlessness, we assume we can get a handle on our lives and social problems by losing weight. If we can only rid ourselves of this beast (that is, obesity), we believe we will not only be thin, but happy, healthy, and righteous. Yet, as with any blind rite, such thinking is a delusion and blaming obesity for our health and social problems is only going to cause us more injury over the long haul.

So how might we change our attitudes about obesity and fat? As with any change in perspective, the first place we must begin is in understanding why we think the way we do. In the case of obesity, we need to understand both why we are gaining weight and, more importantly, why we are calling this weight gain a disease. In other words, if we are to change our thinking about fat, we need to recognize the real sources of America’s obesity epidemic.

This book seeks to help in this effort. It is divided roughly into two parts. The first part, examines how and why our growing weight has come to be characterized as an “obesity epidemic.” Chapters 1 and 2 examine the role of the health professions, drug companies, government, and diet industry in promulgating the idea that our growing weight is a dangerous disease. After reviewing both the scientific evidence and the history of “obesity” as a health concept, it becomes clear that America’s “health industrial complex” is far more responsible for the obesity epidemic than any other source. But the health warnings about obesity have not fallen on deaf ears and if Americans are truly worried about obesity it is because of their receptivity to the various health pronouncements. Chapters 3 and 4 examine why we in the West hate fatness so much, particularly in white women, while the rest of the world tends to celebrate it. As we’ll see, our attitudes about fatness have much more to do with our concerns about social status, race, and sex than they do with health.
The second half of the book examines why we are gaining weight and what this weight gain signifies. Chapter 5 looks at the science of fat and what the genetic sources of weight tell us about our expanding waistlines and our health. Chapters 6 and 7 review the charges and evidence concerning food, exercise, and our growing weight. As we’ll see, the most commonly accused culprits (fast food, high fructose corn syrup, television, and automobiles) are merely accessories to the “crime”; meanwhile, the real source of our growing weight (snacking) goes largely unnoticed. Chapter 8 reviews the politics behind the various obesity initiatives coming from our state and federal governments. Not only are most of these policies unlikely to help us lose weight, they also reveal the fundamental problems with making weight-loss a target of government action. In Chapter 9, the conclusion, I discuss what I think our growing weight really means and what we can do to address the real problems of obesity in the United States.
“Am I fat?” My wife, Thea, is six-months pregnant and fretting over her appearance. I try to reassure her and tell her that she is beautiful, but it seems to have no effect. “Look at me—I’m a whale.” I tell her that she looks terrific and remind her that what she is feeling is perfectly normal. As I’m saying these things, she nods her head. “Yeah, I know. Thanks.” Then, after a pause, she looks at me and says, “But tell me, really, do you think I’m fat?”

Most Americans are probably all too familiar with this question. It looms over our culture like an ominous shadow. It haunts adolescence and ruins marriages. It feeds a mammoth diet and fitness industry. It motivates millions to battle with their own bodies. It is a national preoccupation. And, it is at the center of America’s obesity epidemic.

To understand why more than 60 percent of Americans are considered “overweight” and 25 percent “obese,” we need first to understand how these terms are being defined. After all, the very existence of an obesity “epidemic” hinges on what exactly obesity is—if someone is obese only if he or she weighs 350 pounds, then few Americans would qualify and there would be no epidemic; conversely, if obesity can be had with only 200 pounds, then many people will get this label and the epidemic term may be justified.

But the importance of this terminology goes beyond the question of whether too many Americans are “overweight”; it is also in the power this word has in our everyday lives. In America, being labeled or perceived as overweight means your life will be harder on a number of fronts. You may pay more for many goods and services such as airline tickets and insurance. You will receive different medical care. In some cases, you may be denied a job. More important, the designation of overweight or obese also goes to the very core of a person’s identity. To be overweight is to be, by definition, abnormal or different. By calling people “overweight” or “obese,” we are not simply delineating them by their body mass, we are relegating them to the margins of society. Such labels also become internalized by the “overweight” or “obese” who think that something is wrong with them or that they must change their behavior in order to meet a particular
physical ideal. Ultimately, the power of this terminology is not just in the way the overweight are treated by others, but in the way it makes them see themselves.

Yet, the curious thing is that even though we worry so much about being “overweight” and we hear so much about our “obesity” epidemic, these terms have no precise definition.\(^x\) The *American Heritage English Language Dictionary* defines overweight as “weighing more than is normal, necessary, or allowed,” while obese is “extremely fat, grossly overweight” (my italics). These definitions may seem straightforward but they raise a host of political questions: What standard determines “normal”? By what criteria is something deemed “necessary”? “Allowed” by whom? What point is “extreme” or “gross”?

In the case of body weight, these questions have no clear answers. If normalcy and necessity are what define obesity, then one can easily name any number of different standards on which to base a norm or a necessity. For example, what is considered overweight on a Hollywood starlet or a supermodel is probably thin for most ordinary women; conversely what is overweight on most men is skinny on a professional football player. This problem is confounded because so many different groups profit from setting the definition of these terms at one level or another; the diet industry, for instance, benefits from labeling everyone as overweight, while the fast-food industry prefers that no one thinks of themselves as obese. How these terms get defined ultimately depends on who gains from making people concerned about their body size and who has an interest in getting people to try and lose weight. Which leads us to the story of how our current standards of overweight and obese came to be defined.

The primary reason why more than 60 percent of Americans are “overweight” has nothing to do with fast-food, cars, or television; it is not because Americans are eating too much and exercising too little; nor is it because of any “fat” gene within us. The reason why a majority of Americans are overweight is because a nineteenth-century astronomer, a twentieth-century insurance actuary, and a handful of contemporary scientists concocted some ideas about what a normal weight should be. These definitions have little to do with scientific evidence about weight and health and a lot to do with simple mathematics, insurance premiums, and the pecuniary interests of the pharmaceutical industry. If Michael Jordan is “overweight” or Arnold Schwarzenegger is
obese,” (which they are according to our current standards), it is not because of their poor fitness or their precarious health; it is because a handful of people are defining these terms in ridiculous ways.

Damning Statistics

America’s obesity epidemic originates in a simple measure, the body-mass index, or BMI. Most of us have had our BMI checked at one time or another: it’s the test where you type in your weight and height and you’re told if you’re overweight. It is the most common method for classifying people’s weight—used by nearly all doctors, government officials, and health organizations. The surgeon general and the Centers for Disease Control and Prevention (CDC) have used BMI as the primary basis for claiming that obesity is a major health epidemic. The National Heart, Lung, and Blood Institute and many weight-loss companies feature BMI calculators on their websites and many of us get pop-up ads asking us to check our BMI to see if we’re overweight. BMI has become a ubiquitous part of the American lexicon. At first glance, BMI seems to be a relatively neutral way of determining weight status. It is simply the proportion of a person’s height to weight. BMI has a curious history though, that reveals much about the origins of America’s “obesity epidemic.”

Interestingly, the concept of BMI was not developed with any connection to body fat. The first person to use it was not even concerned with losing weight or with health but, rather, he was interested in the laws of the heavens. In the 1830s, a Belgian astronomer named Adolphe Quetelet was trying to test whether mathematical laws of probability could also be applied to human beings. These statistical laws were used in astronomy to predict the likelihood of a phenomenon based on repeated observations. Quetelet believed that such laws also governed human affairs. To predict human behavior, all one needed to do was gather information on a large enough sample of a population and calculate general trends.

To test his ideas, Quetelet began collecting information from French and Scottish army conscripts. Along with other details, Quetelet measured their weights and heights and plotted them along a distribution. For each height, he found a range of weights in what statisticians would later call a normal distribution or, more famously, a bell curve.
At the center of the distribution (or top of the bell curve) Quetelet found the most number of cases, which was the average weight of the group. In charting these distributions, he happened to observe that the weight of “normal” conscripts (that is, those closest to the middle of the distribution) was proportional to their height squared; this general formula would later be used to determine BMI. But Quetelet did not stop there. Since the average conscript’s weight was proportional to his height, Quetelet reasoned that this must be what the ideal weight should be; anyone who deviated from this average could be considered either under- or overweight. This pseudoscientific conception of an “ideal” weight thus provided the first scientific notion of what overweight could be.

Quetelet’s idea had deep political implications. Among his many accomplishments, Quetelet first derived the concept of the “average man.” Because most people congregated around average points in their physical characteristics, Quetelet believed that deviants, criminals, or troublemakers could be identified by their physical abnormalities. A similar technique was now available for body weight. Not only could Quetelet’s method determine what was the “normal” weight for a population (which was simply the average), it could also mathematically define who was abnormal or “overweight” by calculating how far someone “deviated” from the norm. The farther someone was from the average weight, the more they violated other social norms and the more they could be monitored, institutionalized, or controlled.

Quetelet’s scheme was a harbinger of a larger wave of scientific attempts to measure and differentiate groups in society. Throughout the nineteenth and early twentieth century, scientists became enamored with measuring skulls, brows, body proportions, and other aptitudes. Following Darwin and the development of biology, it was the golden era of classification. Although these efforts were often done in the name of science, they sought to do more than merely taxonomize the population. Most efforts at measurement were meant to identify miscreants and justify racial and economic prerogatives among a white, aristocratic elite. For example, in the late 1800s public officials and scientists went to great lengths to catalogue the physical characteristics of criminals, arguing that their delinquency was tied to their physiognomy. By claiming that elite groups had certain traits, scientists could rationalize racial inequities in wealth, employment, and education—something that we see with the controversial claims linking
race and I.Q. test scores today. From Quetelet’s measurement of BMI, the groundwork was laid for a similar process of classification for body weight. A high (or low) body weight, simply by being different from the average, was not only systematically identified, it was also problematized. Even though there was no linkage between weight and health, delinquency, or any social ill, just by being far from the average, overweight and underweight people were marginalized.

Although Quetelet’s methods provided a “scientific” basis for classifying (and standardizing) body weight, BMI did not become widely used until a century later because body weight was not a very good mechanism for social differentiation. Most people in the nineteenth century struggled to get enough to eat and few had the luxury of worrying about whether they were too fat. Since only the rich and well-off could afford to be corpulent, there were few groups who were looking to differentiate people for being too heavy. That is, except for one—the insurance industry. For years, life insurance companies had been desperately trying to find mechanisms that would predict early deaths. In the early 1900s, when medical technology was still crude, they had few diagnostic tools for determining who might die early, and thus be a greater policy risk. Even though they suspected that body weight (like other physical traits) could be a predictor of mortality, they had no way of systematically using it calculate their insurance premiums. In other words, because they had no way of knowing how much more of a risk a 240-pound man was than a 220-pound man, they did not know how much more that person should be charged for life insurance.

Seeking to answer this question, Louis Dublin, a statistician at the Metropolitan Life Insurance Company, started charting death rates of its policyholders in the 1940s using a height-to-weight index. In line with industry expectations, Dublin found that thinner people lived longer. But, more important, he also found that the closer a person’s weight was to that of the average twenty-five-year-old, the longer he or she would live, or least live before cashing out on their life insurance. From these findings, Dublin came up with some ranges for each height of what was an “ideal” body weight (that is, which weight had the longest lifespan). Although Dublin’s classification scheme was primarily intended for insurance actuary tables, they soon took on a whole new function, thanks in large part to his tireless promotion of weight as a determinant of early mortality.
Following Dublin’s lead, doctors, epidemiologists, and the federal government soon adopted these tables to analyze the “health” of the population. By the 1950s, the Met Life table was the method for determining who was overweight.

It is important to remember, however, that up until this point, BMI was never intended to be a gauge of someone’s health. When Adolphe Quetelet came up with BMI, he was simply trying to classify the population and not make any predictions about death or disease. Nor were Louis Dublin’s Met Life actuary tables based on any biological rationale; Dublin did not specify why heavier people would die earlier, nor did his model account for genes, diet, exercise, or many other influences on mortality. Rather, Dublin used weight because it was easy to measure and had a lot of statistical power to predict the likelihood of early death. But as a result of his use of the statistics, people came to think that body fat caused early death, an idea that Dublin himself propagated. Ultimately, the most influential factor in determining what Americans considered to be overweight was not based on criteria of health but criteria of profit and measurement within the insurance industry.

Despite this dubious history, BMI remains the basis for much of our official health policy today, both in the way we think of obesity and how we measure it. Government health agencies, such as the Centers for Disease Control and Prevention (CDC) and the National Institute of Health (NIH), rely on BMI as the primary indicator of weight, health, and mortality risk in the American population. Today, almost all government agencies consider anyone with a BMI of 25 or more as “overweight.” For an American man who is the average height of 5’9” that would be 170 pounds; for an American woman who is the average height of 5’4” that would be 145 pounds. A BMI of 30 or more is “obese” (that is, 204 pounds for a 5’9” American male and 175 pounds for a 5’4” American woman). According to government health officials and many obesity experts, these BMI scales are a simple and easy method for gauging your health. All you need to do is check your height and weight and “voilà!” you can tell, not only if you are “overweight” or “obese,” but also how well you are. Except for one problem—it’s not true.

The Problem with BMI
Despite its ubiquity among government agencies, medical practitioners, and health researchers, BMI is not only a poor measure of health, it is actually a lousy measure of obesity. To begin with, BMI is a measure of proportionate body weight and not a measure of body fat. This is why many professional athletes are technically “overweight” or “obese” even though they have little body fat. If we think of obesity as an excess of body fat (which most people do), then BMI is an inaccurate gauge.

And the problem does not stop there, for BMI is also a poor predictor of mortality. It is not a simple fact that the heavier a person is, the more likely he will die. The association between mortality and BMI is more of a U shape—those at both the low and high ends of the weight spectrum have higher mortality rates than those in the middle. The rates of mortality at the high end of the BMI scale do not become prominent for men or women until a BMI is generally over 35 (which applies to less than 10 percent of the population). This correlation also varies by age—among older people, a BMI is negatively related to mortality (that is, the heavier you are, the less likely you are to die).

Nor does BMI tell us much about why thinner or heavier people die. Although people at the either end of the BMI scale may have higher death rates, we simply do not know if early death comes from having too much or too little adipose tissue or whether BMI is simply reflecting other unmeasured influences. BMI does not take into account fitness, heart rate, or fat distribution, all of which relate to disease and mortality. By some accounts, fat distribution may actually be a better predictor of mortality than body weight—one study found the mortality associated with higher BMI levels can be completely accounted for by waist circumference. Fat on someone's hips and thighs seems to have little or no relationship to the risk of death, it is only fat in the belly that seems to be problematic. Thus, if our concern with obesity is that it is supposedly killing thousands of Americans, then actually BMI tells us very little about who those people may be.

But these issues pale in comparison to the biggest problem with BMI; we have no clear criteria of what points on the BMI scale should be classified as “overweight” or “obese.” Over the past two decades, the BMI thresholds for these terms have yo-yoed, sometimes being pegged at one level, sometimes at another. For example, between 1980
and 2000, the U.S. Dietary Guidelines (a joint report from the Departments of Agriculture and Health and Human Services) have defined overweight at various levels ranging from a BMI of 24.9 to 27.1. In 1985, the National Institutes of Health (NIH) consensus conference recommended that overweight be set at a BMI of 27.8 for men and 27.3 for women—by this standard, a 6’ man would be overweight at 205 pounds, a 5’7” woman would be overweight at 175 pounds. Then, in the 1990s, the World Health Organization (WHO) came out with a recommendation that a BMI of 25 to 29 should be considered overweight and a BMI of 30 or more obese (more on this below). To make the United States consistent with this standard, many federal health agencies and researchers soon began adopting the lower BMI standards, thus creating a confusing set of standards and guidelines.

Partly to sort through these conflicting measures, in 1988 the NIH convened a panel of more than two dozen experts from the fields of health research, epidemiology, and nutrition to review the “evidenced based” research of the past twenty years. This NIH report concluded that the official designations of overweight should be set at a BMI of 25 and obesity at a BMI of 30, the same standards established by the WHO. Soon, this became the definitive guide for determining what was officially overweight and obese in the United States.

At the time it came out, the NIH report caused a lot of controversy because, overnight, more than 37 million Americans suddenly became “overweight,” even though they had not gained an ounce. What few people noticed, however, was that the scientific “evidence” to justify this change was nonexistent. According to the NIH report, the classification of overweight at a BMI of 25 was based on the putative linkages to mortality. According to the report, people who have a BMI of more than 25 had “significantly higher mortality” rates than those under 25, but in both the WHO and NIH reports, none of the research really substantiated this claim. For example, the major source cited by the NIH board was a 1996 review of studies linking BMI and mortality by the nutritionist Richard Troiano and his associates. Yet, strangely enough, Troiano’s findings actually contradict most of the recommendations of the NIH panel. Not only did he discover that mortality was highest among the very thin as well as the very heavy, but also that the increased mortality was typically not evident until well beyond a BMI level
of 30. And until one gets to a BMI of 40 or more, the differences in mortality are still within the bounds of statistical uncertainty. From these findings, Troiano concluded that “This analysis of mortality suggests a need to re-examine body weight recommendations. Weight levels currently considered moderately overweight (i.e., a BMI > 27) were not associated with increased all-cause mortality.”**x**x**x**x Interestingly, although the NIH panel did recalibrate body weight recommendations, they did so in the opposite direction, lowering the BMI designation of what would be considered overweight and obese.

The fact of the matter is that, with our current data and measurement techniques, it is impossible to calculate the mortality risks of obesity accurately. The epidemiological studies that have estimated the links between BMI and mortality are not based on studies of the entire population or of all deaths, but on large pools of survey data from various projects tracking health. In these samples, epidemiologists simply measure the association between death rates or various diseases and body weights. If deaths or diseases are more common as weight goes up, a trend is identified. But calculating mortality in this way is a tricky business, largely because the illnesses that cause most deaths in America (that is, heart disease, cancer, and stroke) have so many sources. In other words, heavier people may have a higher mortality rate but this does not necessarily mean that it is their body fat that is killing them. Their weight may simply be capturing the effects of other unmeasured variables. The validity of this research depends largely on the variables that are included in the estimates and the margins of error from the coefficients. In order to determine that a certain trait has a really significant impact, the differences in the statistical estimates must be great enough not to simply be caused by random error. Moreover, it must be verified that the trait in question is the direct source of the problem and not simply a proxy for other causes. For example, mortality rates may be higher among the obese because heavier people are less likely to seek regular medical care, the consequence of the prejudice they often encounter among medical professionals.

The problem is that the major studies on the number of deaths due to obesity are fraught with all sorts of problematic assumptions. The two most commonly cited studies that linked higher BMI with mortality were written by the obesity researcher David Allison and his colleagues in 1999 and by a collection of CDC researchers in 2004. Both appeared in the *Journal of the American Medical Association (JAMA).* **x**x**x**x And both
studies calculated that obesity (defined as a BMI of 30 or above) was causing several hundred thousand deaths a year. Yet, in neither of these studies did the researchers actually measure the linkage between obesity and death nor did they take into account other explanatory factors, such as genes, diet patterns, or exercise, that might also explain why heavier people had higher mortality rates.

In the 1999 study, Allison and his colleagues assumed “that all excess mortality in obese people is due to obesity.” But, in reality, no one has proven that adiposity (excess amounts of fat) is an independent cause of heart disease, cancer, and stroke. Moreover, their methods of calculating the obesity effects were incredibly crude: they divided the population between non-obese and obese and assumed that any deaths that occurred among the latter were due to their excess weight. Even if an obese person died in a car accident or from a snakebite, the cause of his or her death was attributed to body weight. These claims are as ludicrous as arguing that the difference in mortality rates between blacks and whites are the result of their skin color.

The 2004 JAMA study is more careful in its language but equally problematic in its conclusions. This study, authored by researchers and the director of the CDC, calculated that the diet and inactivity associated with obesity causes 400,000 deaths a year in the United States. While they actually attribute these deaths to “poor diet and physical inactivity,” they nevertheless assumed that these factors work primarily through obesity rather than having a negative impact on their own. In their view, the problem with inactivity and poor diet is that they make you fat and being fat is what kills you rather than simply saying that poor diet and inactivity are themselves problematic. Yet, as we’ll see in later chapters, there is much more convincing evidence about the immediate health hazards of a poor diet and inactivity than there is about being too fat.

Indeed, if you look at the actual numbers on death, you’ll find a much different story. Each year, roughly two million Americans die from all causes. About 70 percent of these two million deaths are among people who are over 65. Among the elderly, obesity is not a major cause of death because overweight and obese senior citizens (those with a BMI above 24) actually live longer than those at a normal or below normal body weight (a BMI below 24). Yes, that is correct—older people who are heavier live longer than those who are thin. So if obesity is not killing the elderly, it means that obesity can only
be responsible for some part of the roughly 600,000 deaths among the remaining population under the age of 65. And what are these people dying from? The biggest killers among the non-elderly are largely unrelated to obesity; the top cause of death among people who are under forty-five is unintentional injury, primarily from automobile accidents; the top killer among people between forty-five and sixty-five is cancer, the leading cause of which is smoking. Among the top ten causes of death for people under sixty-five, only heart disease, diabetes, and a small fraction of cancer deaths have any plausible connection to body weight.

And, even if we assume that all the deaths from heart disease, diabetes, and other organ ailments are attributed to obesity, we only get, at most, about 174,000 deaths a year among people under sixty-five. Yet this number is also too high because we know that plenty of thin people are also dying from heart disease, diabetes, and organ failure. In reality, we have no clear idea whether any deaths at all can be attributed solely to a person’s body weight. After these and other discrepancies in the 2004 CDC report were brought to the attention of Congressman Henry Waxman, in the summer of 2004 he asked the General Accounting Office of the U.S. government to launch an investigation. The CDC ended up retracting their earlier estimates and after an “internal review” in which the miscalculations were blamed on a “computer error” they released new estimates for which the annual rate of death attributed to obesity was only about 365,000 per year. Even these numbers were based on specious reasoning; a more recent and reliable estimate from Kathryn Flegal, a researcher at the National Center for Health Statistics, puts the number of deaths attributable to “weighing too much” at less than 25,000 a year. In addition, Flegal found that people who are “overweight” (with a BMI of 25 to 29.9) live longer, on average, than those at a normal weight (a BMI between 18.5 and 24.9).

These inferential problems get even worse when the discussion turns to the question of diseases. The 1998 NIH report made an extensive list of diseases that were “associated” with higher BMIs including hypertension, type 2 diabetes, coronary heart disease, stroke, gall bladder disease, osteoarthritis, and some types of cancer. As the NIH report went through the list of these diseases, it cited hundreds of studies to back its claims, the result of a seemingly exhaustive search of the scientific literature. This
evidence was supposed to prove that, even more than being a cause of death, a high BMI was a major health risk. Except, once again, there were two major problems.

First, there is no uniform point on the BMI scale at which all these diseases become more evident. The relationship between BMI and each disease varies considerably depending on the condition in question. For some conditions, such as diabetes, an increased likelihood can start as low as a BMI of 22; for other health conditions, such as many types of cancer, the increased risks do not begin until a BMI is much greater than 30. And again, in many instances, these health pathologies might not arise from adipose tissue but from associated causes. For example, the association between heart disease and obesity may come from greater insulin resistance among the obese, a factor that can be alleviated through exercise, even when weight isn't lost.xxxvii These health effects are also subject to significant differences depending on race, gender, and age. Even worse, some studies show that higher BMIs are actually associated with lower rates of cancer and heart disease.xxxviii

Indeed, the problem of misattribution appears throughout the NIH report. For example, throughout the report boxed statements are given particular emphasis with messages such as “Weight loss produced by lifestyle modifications reduces blood pressure in overweight hypertensive patients.”xxxix This type of message is repeated with numerous other diseases. Yet in none of the studies cited was it conclusive that the weight loss itself was responsible for the remediation of the illness rather than the change in lifestyle. In fact, it is far more plausible that the increase in exercise and change in diet that affected the weight loss is the real cause for the health improvement.xl This is like saying “whiter teeth produced by elimination of smoking reduces the incidence of lung cancer.” Nevertheless, the NIH report continues to emphasize that weight loss was a causal factor.

Second, nearly all the studies linking obesity with disease are epidemiological studies; that is, they are simply surveys of the population and not clinical experiments. Not only are these data often problematic (for instance, body weight is self-reported), the inferences that can be made from the data are unclear. Epidemiology is a tricky business—one tries to find relationships between phenomena by examining large surveys of the population and seeing where statistical associations exist. To determine causality,
such as with smoking and lung cancer, epidemiologists look for the strength of association (what percent of smokers get lung cancer?), the timing of the association (does lung cancer follow years of smoking?), and whether there is a plausible scientific explanation between the two (is there something in cigarette smoke that would trigger lung cancer?). If these links are all clear, then causal inferences can be made with some confidence.

But in most studies linking body weight and disease, these conditions for determining causality are not met. Numerous critics, such as the editors of the New England Journal of Medicine, Dr. Glenn Gaesser of the University of Virginia and Professor Paul Campos of the University of Colorado, have pointed out that most of the evidence linking obesity, mortality, and disease is fraught with questionable methodological assumptions. In fact, many studies do not taken into account other factors that might account for diseases such as smoking, access to medical care, family history, exercise, or diet.

Perhaps the biggest problem with this research is that we do not have a good theory on why obesity causes heart disease, cancer, or other ailments. Indeed, the evidence does not support many of our common stereotypes about the health risks of obesity and disease. For example, it is common for people to think that being fat clogs your arteries, but there is no conclusive proof that having more body fat results in more atherosclerosis independent of one’s diet. There are only two medical conditions that have been shown convincingly to be caused by excess body fat: osteoarthritis of weight bearing joints and uterine cancer that comes from higher estrogen levels in heavier women (although this is can be treated medically without weight loss). All the other diseases are only linked to obesity through associations in large populations. It is not clear why having a lot of fat tissue would make someone more likely to have heart disease, asthma, or many of the other diseases commonly attributed to obesity.

Herein lies the biggest problem of making health-related claims about obesity—there is far more we do not know about the consequences of excess fatty tissue than we do. The hypotheses about the causal links between excess fatty tissue and most health pathologies are largely untested. Although some obesity researchers now believe that many diseases may be caused by the hormones and signaling compounds produced by fat
cells, they still have not proven how excess levels of these hormones may be harmful. The effect of these hormones in causing disease is still a matter of speculation. Nor, more important, is there any conclusion about at what level of obesity such excess hormones become dangerous. As obesity researcher and NIH panel chair Xavier Pi-Sunyer says, “It’s a very complicated system, and the more we learn about it, the more complicated it becomes.”

The Politics of Defining Obesity
So, if the scientific evidence about the relationship between BMI, mortality, and other health conditions is so unclear, why did the NIH, putatively the most objective public health institution in the United States, endorse these low thresholds of overweight and obese? According to one NIH panel member, the overweight designation came from the “best scientific judgment” of the committee members. But the decision to lower the weight scale was not based on any revolutionary research in the scientific community about what an ideal weight should be—with such fuzzy evidence, science could not have possibly informed this decision. The U.S. government’s proclamation of what BMI level was overweight or obese was based, in reality, on a subjective and arbitrary call on the part of just a few researchers. Ironically, the same NIH panel that strove for an “evidence-based” and objective criteria, ended up making a major proclamation that, in retrospect, appears to have been for reasons that had do nothing with health and a lot to do with the funding dynamics within the scientific professions and the pharmaceutical industry.

To understand this point, it is important to go back to the 1995 World Health Organization report that helped establish the idea of that a person is overweight with a BMI of 25. This document probably had more impact on determining how obesity was defined than anything else. And who wrote this important document? Most of it was drafted and written under the auspices of the International Obesity Task Force (IOTF). On the surface the IOTF seems to be a credible association of scientists interested in obesity research and policy. According to its website, the IOTF’s mission is to “inform the world about the urgency of the problem and to persuade governments [sic] that the
time to act is now.” Their website also displays the logo of both the WHO and the International Association for the Study of Obesity, legitimate health organizations, making the IOTF seem like a purely scientific organization.\footnote{xlviii}

In reality, however, the IOTF is anything but an unbiased congress of scientists. The IOTF is an organization primarily funded by Hoffman-La Roche (the maker of the weight-loss drug Xenical) and Abbott Laboratories (the maker of the weight-loss drug Meridia).\footnote{xlix} Like other organizations financed primarily by drug companies that don the “neutral” mantle of science (including the American Obesity Association), the primary mission of the IOTF is to lobby governments and advance particular scientific agendas that coincide with the pharmaceutical industry’s goals. Indeed, the initial mission of the IOTF was to get the lower BMI standards imposed on the WHO report. Few realize that the effort to establish a worldwide standard for what is overweight and obese was sponsored primarily by a company that makes a weight-loss pill.\footnote{\it{}}

The IOTF’s chair, British nutritionist Philip James, typifies this conflict of interest. James, a well-regarded scientist, also has many financial links to the pharmaceutical industry. He has been amply paid for conducting clinical trials of Sibutramine (Meridia) and Orlistat (Xenical). He also engages in regular promotional activities for Hoffman-La Roche and Knoll Pharmaceuticals, offering regular praise of their products in press releases. In fact, in 2003, he presented the Roche Gulf Awards for Obesity Journalism to reporters who promoted studies (on which James consulted) showing, not surprisingly, that patients taking Xenical were 37 percent less likely to develop type 2 diabetes than those losing weight through lifestyle changes alone.\footnote{li} In short, James is not only a consultant for the drug industry, he also works as one of their pitchmen.

The influence of the pharmaceutical companies doesn’t stop with such faux health organizations. The drug industry financially supports many researchers who are on the advisory panels to both the WHO and NIH. Pharmaceutical companies influence the tenor of scientific research and interpretation both by funding research and by contracting with various health researchers to serve as “consultants” for their various products. For example, the chair of the NIH committee (and a member of the WHO panel) is a doctor and medical researcher named Xavier Pi-Sunyer (the same one quoted above). In addition
to being a professor of medicine at Columbia University, Pi-Sunyer is also the director of the Obesity Research Center at St. Luke’s-Roosevelt Hospital in New York City and the director of the VanItallie Center for Weight Loss. While Pi-Sunyer has these impressive scientific credentials, he also is on the advisory board or is a paid consultant to several diet and pharmaceutical companies, including Wyeth-Ayerst labs (makers of the fen-phen diet drug that ended up causing heart valve damage), Knoll, Eli Lilly Pharmaceuticals, Genentech, Hoffman-La Roche, Neurogen, and Weight Watchers International.iii Pi-Sunyer has been the highly paid principal investigator on recent clinical trials of the drug Rimonabant made by Sanofi-Aventis. Indeed, Pi-Sunyer has been named in a lawsuit against the drug company Wyeth-Ayerst because he agreed to have his name attributed to scientific articles about the costs of obesity that were actually written by Excerpta Medica, a medical consulting firm, and paid for by Wyeth-Ayerst.iii Not surprisingly, Pi-Sunyer is also a member of the IOTF.

Pi-Sunyer is not alone in his connections to the pharmaceutical industry—many of the researchers on the NIH board (once again the group that basically defined what overweight and obese mean in the United States), as well as most of the top obesity experts in the United States, such as David Allison, George Blackburn, Tom Wadden, James Hill, and Judith Stern, are financially tied to diet and pharmaceutical companies. A particularly egregious example among this group is George Bray, one of the first obesity experts and the editor of *Obesity Research*, the leading academic journal on obesity studies. In addition to his long list of research publications, Bray also has a side job as a developer and marketer of a “weight-loss” thigh cream that has had little long-term success. It is difficult to find any major figure in the field of obesity research or past president of the North American Association for the Study of Obesity who does not have some type of financial tie to a pharmaceutical or weight-loss company.

While the pharmaceutical industry did not necessarily dictate the decisions of the obesity experts, the conflicts of interest among the leading researchers in the obesity field are both undeniable and problematic. The IOTF’s campaign to lower the standard of what is overweight directly coincides with the economic interests of the diet and pharmaceutical industries, especially in the case with weight-loss drugs such as Meridia and Xenical.iv By lowering the BMI standard and making more people think they are
overweight, the pharmaceutical industry can create a much larger market for diet drugs and diet plans.

Of course, the pharmaceutical industry is not alone in wanting to lower the standard for being overweight and to increase the number of people that fall into that category. Significant financial incentives also exist for university health researchers and health agencies within the U.S. government. In fact, the coincidence of interest between the pharmaceutical industry and public health researchers has created something in the field of obesity that could best be described as a “health-industrial complex.” The health-industrial complex is built upon a symbiotic relationship between health researchers, government bureaucrats, and drug companies. Drug companies sponsor research that defines current health issues and fund researchers who sit on the NIH board and within medical schools; government bureaucracies such as the NIH and CDC rely on the expertise of researchers to back their claims for increased congressional funding; and drug companies use health warnings issued by the CDC to promote their products. As the health writer Thomas Moore notes, “The same medical school physicians who serve on NIH consensus and other panels also work as consultants to these drug companies and are paid handsome fees to speak at the medical conferences that these companies finance. It is a tightly interlocking system.”

A key part of the health-industrial complex arises from the funding imperatives within medical research institutes. Within most of the research institutions and universities where health research is conducted, a significant portion of the salaries of scientists and their staffs is based on grants from foundations and support from private industry or the federal government. This application process for these grants is very political—who gets funded and at what level depends on a number of factors including the importance of the research problem in question and how well it fits within the established health paradigms. Similarly, the funding levels of the CDC, the NIH, and other government agencies depend upon perceptions of the U.S. Congress about the validity of their efforts. Lobbying groups such as Research!America tirelessly promote the possibility of looming health catastrophes in order to secure greater federal funding of health research. Getting funds to do health research and promotion, whether one is in the private or public sphere, depends largely upon how serious a health problem one is
researching. Pathologies affecting large or vocal populations, such as cancer or AIDS, get more money; conditions affecting smaller or less-represented groups get less.\textsuperscript{lvii}

For obesity researchers, this means there are significant incentives to lower the threshold of what is considered overweight. By adopting the overweight standard of BMI at 25, they can add nearly 40 million people to the population at risk. For medical researchers focusing on weight loss, this quickly inflates the importance of their own efforts; for government agencies such as the CDC and NIH this gives them a new reason for expanding their own missions and increasing their budget requests; for both groups it provides a rationale for expanding their power.\textsuperscript{lvii} Thus, it was not mere coincidence that soon after the NIH report the congressional budget appropriations for obesity-related programs in the CDC and NIH were increased. Nor does it seem like mere coincidence that one of the lead authors of the \textit{JAMA} article that attributed 400,000 deaths a year to obesity was the director of the CDC (Julie Gerberding) and that she used the information in this article in her congressional testimony requesting a larger CDC budget.\textsuperscript{lviii}

These efforts to lower the BMI threshold of what is considered overweight have further obfuscated the complicated relationships between body weight and health. Once the “experts” come out with evaluations of what is overweight or obese, even the most exaggerated or misinterpreted claims take on a life of their own and become accepted as unquestioned truth. After researchers first identified the growing weight of the American population in 1994, a host of news items and scientific articles sounded the alarm about the increase in body weight of the American public.\textsuperscript{lix} Obesity began to be paired with terms such as “epidemic,” first, in a careful or measured way, but inevitably without criticism or introspection.\textsuperscript{lx} By the end of the decade, scholarly articles, government reports, and the news media were proclaiming obesity an epidemic with little acknowledgment of the medical complexity and problematic assumptions of this claim.

Although the media often sensationalize and oversimplify complicated issues in order to attract public interest, in the case of obesity this is has been taken to great lengths. Consider these two examples. On March 10, 2004, the \textit{New York Times} ran a headline “Death Rate from Obesity Gains Fast on Smoking.” This major story presented the conclusions of the 2004 \textit{JAMA} article that “obesity-related” deaths in the United States would soon top 400,000 and overtake smoking as the number one cause of
preventable death in the United States without a single hint of criticism.\textsuperscript{lxvi} It reiterated the various costs associated with obesity but never questioned how these numbers were reached. Similarly, on January 8, 2003, the Associated Press ran a story with the headline “Obesity at Age 20 Can Cut Life Span by 13 to 20 Years.” Only later did the story reveal that the obesity in question was at a BMI of 45 (that would be more than 340 pounds for a six-foot man), which affects less than 1 percent of the population.

With this growing consensus about the threat of the obesity “epidemic,” it became increasingly difficult for ideas or findings that contradicted, or least questioned, the claims about obesity to gain any attention or audience. This occurred not only in the press but also among the very research institutes and government agencies that issue the reports. Last year, the writer Elliot Marshall reported in the journal \textit{Science} that many researchers at both the NIH and the CDC had concerns about the “loosey-goosey” estimates of the number of deaths attributable to obesity, particularly in the way that age was used to calculate mortality.\textsuperscript{lxvii} One CDC staffer, who did not want to be quoted on the record for fear of losing his job, said that many at the CDC felt the conclusions of this report were not open to question, particularly as one of its lead authors, Julie Gerberding, was the director of the CDC. Marshall stated that many people believe that the \textit{JAMA} article’s “compatibility with a new anti-obesity theme in government health pronouncements—rather than sound analysis—propelled it into print.”\textsuperscript{lxviii} Glenn Gaesser also reports that the NIH basically ignored the alternative studies that challenged the link between BMI and higher mortality when writing its report about the dangers of obesity.\textsuperscript{lxix} While one might not expect a researcher to cite all the evidence about obesity, the NIH report omitted a number of studies questioning the link between BMI and mortality when setting the threshold of those considered overweight.\textsuperscript{lxx}

Standardizing Our Weights

Now some may question why this is a very big deal; after all, does it really matter if the government gets its weight standards exactly right? Isn’t it a good idea to lose weight, even if weight is merely a proxy for other, more serious problems? Actually it is a big deal, for the use and definition of terms such as “overweight” and “obese” have a number of important consequences. First, being classified as overweight has an
immediate impact on the lives of millions of Americans: it can determine whether they
can work at certain jobs, whether they are considered fit parents, or whether certain drugs
or medical procedures will be paid for by insurance or tax money.\textsuperscript{lxvi} Our current
standards wrongly compel doctors to tell their patients they are sick and convince
millions that they should starve themselves with dangerous crash diets and other weight
loss strategies. The current designations of overweight and obese may cause all sorts of
unfair, unhealthy, and unnecessary behaviors on the part of Americans who think they
need to be thin in order to be healthy.

Even more problematic is that these “official” pronouncements about what
constitutes a healthy body weight are being thrust upon the general public in a coercive
manner. Although they don’t have a clear scientific rationale, our current standards of
overweight and obese affect the self-image of many, imposing a standard by which most
of us don’t measure up, particularly if we are female, poor, or a minority. And, by
evaluating ourselves relative to weight standards defined by BMI, we fall under the
power of the medical and science professions that tell us how we should think about our
bodies and how we should behave. Our current standards of overweight and obese is that
they are affecting the very conception of who we are.

But the biggest problem with our current definition of “obese” is that it makes
weight a central determinant of health when, in fact, the relationship between body fat
and health is far more complicated than what can be found with a BMI, particularly for
the general population. Everyone has his or her own ideal body weight. My grandmother,
who lived to a vigorous ninety-eight years old, was technically obese for most of her
adult life. What is an optimal body weight for one person may be far heavier or lighter
than for another.\textsuperscript{lxvii} Even if it is possible to identify an “ideal weight” for any one person,
there is no way to create a uniform standard that can be applied for a large population.

Nevertheless, our government and the public health community continue to
emphasize that we should evaluate our health relative to how much we weigh and to
advise millions of Americans, who otherwise would not consider their weight a problem,
that they should lose weight. Yet this same government and health community is not
providing any clear or safe guidance of how Americans should actually meet this thin
ideal. Without any clear understanding of what is actually causing our weights to rise, the
government’s warnings about the dangers of body fat will only encourage people to take up unworkable or unhealthy diet plans, which often do more health damage than anything else. However, the campaign to shape our perceptions does not simply stop with making millions of us wrongly think we are overweight or obese; as we’ll see in the next chapter, they also want us to change how we understand what this obesity means.

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ii I didn’t know it at the time, but independently, Paul Campos, author of The Obesity Myth, was coming to a similar conclusion and whose argument was influenced largely by Glen Gaesser’s pioneering book Big, Fat Lies.


iv A full description of this will be provided in chapter 1.

v The CDC researchers estimated the number of deaths at numerous weight ranges. Compared to people with a “normal weight” (i.e., a BMI of 18.5–24.9), overweight people (a BMI of 25–29.9) had 86,094 fewer deaths, while those who are “obese” (a BMI of 30–34.9) had only 29,843 deaths; “extremely obese” people (a BMI of 35 or above) accounted for 82,066 deaths. If you subtract the number of overweight people who are living longer from the number of obese people who are living shorter, you only get 25,814 deaths, fewer than the number of deaths from underweight people (i.e., a BMI of under 18.5) which is 33,746 (Flegal, K. 2005. JAMA). The editors of the prestigious New England Journal of Medicine, after evaluating the numerous studies on obesity and mortality, came to the following conclusion:

The data linking overweight and death, as well as the data showing the beneficial effects of weight loss, are limited, fragmentary, and often ambiguous. Most of the evidence is either indirect or derived from observational epidemiologic studies, many of which have serious methodologic flaws . . . Although some claim that every year 300,000 deaths in the United States are caused by obesity, that figure is by no means well established. Not only is it derived from weak or incomplete data, but it is also called into question by the
methodologic difficulties of determining which of many factors contribute to premature
death. (Kassirer, J., and M. Angell. 1998. “Losing Weight—An Ill-Fated New Year’s


viii For a nice description of this, particular as it is evident in Greg Critser’s book *Fatland*, see: Campos, P.
(Winter).

ix Similarly, if obesity is termed as a “disease,” than some may be entitled to government tax benefits.

x Medical definitions of obesity are no less problematic. Webster's New World/Stedman's Concise Medical
Dictionary defines obesity as ”an abnormal increase of fat in the subcutaneous connective tissues” but
subcutaneous fat is far less dangerous than intra-abdominal visceral fat some types of subcutaneous inhibit
cardiovascular disease.

xi Shell.

xii In many ways, this made Quetelet the father of modern, quantitative social science.


xv Ibid.


xviii Unlike other ways of gauging fat such as skin folds or water displacement, Bray, G. 1998.
Contemporary Diagnosis and Management of Obesity. Handbooks in Health Care.

For example, much of the standard for defining overweight comes from the mortality data from Metropolitan Life. But from a scientific perspective, the predictions from the Met Life tables are untenable because they were derived from Met Life’s insurance pool which was overly white, male, and middle-class, and not a representative sample of the general population. In addition, the methods Met Life used to calculate mortality rates relative to the weight ranges are themselves problematic. According to one statistician, the methods used to calculate the mortality rates are largely incomprehensible. See Jarrett, R. 1986. “Is There an Ideal Body Weight?” British Medical Journal 293: 493-95.


These numbers were not based on mortality figures but on the distribution of weight among the 20-29 year old population. Like Quetelet, the U.S. health agencies simply defined overweight relative to the weight distribution in the general population (albeit the young population). Of course, this type of scheme also meant that if the population started getting heavier, then the definition of overweight would necessarily have to rise. And indeed, this is what began to happen.


My italics. The only publicly available and refereed article cited by the NIH board was Troiano, R, et al. 1996. “The Relationship Between Body Weight and Mortality: A Quantitative Analysis of Combined Information from Existing Studies.” International Journal of Obesity 20: 63-75. This study is a review of all of studies linking BMI and mortality and not even a direct study of any data on this issue. Not only did they find that the insurance data differed dramatically from the population at large, they also found a U-shaped relationship between BMI and mortality. Moreover, the increased mortality was typically not evident until well beyond a BMI level of 30.


Allison et al., 1999.

Fontaine, K et al. 2003. “Years of Life Lost Due to Obesity.” Journal of the American Medical Association. 289:187-193. This research finds virtually no effect of body weight on expected mortality among the elderly, except among African Americans, where it has a positive effect (i.e., obese African Americans actually live longer). The only measurable impact is among white women with a BMI over 40 – elderly women with this much weight make up only a tiny fraction of the population.

Another factor with these studies is that these estimates inevitably are rounded to very high levels, generally to the 100,000 level. With such large rounding, it is quite difficult to know the magnitude of the trend. Moreover the increasing deaths seem to fly in the face of other trends. In 1980, an article by the Carter Center gave an estimate of 290,000 deaths per year due to over-nutrition, a decade later research by McGinnis estimated only 300,000 deaths. After 20 years of population growth and increased obesity, Mokdad’s estimate is up to 400,000, yet with a slight reinterpretation of the number (which was within the confidence interval), Mokdad might have reported 300,000 deaths, which would have been a decrease in the rate of obesity-related mortality relative to the population. In other words, despite the massive increase in obesity in the American population, one could easily estimate that the number of deaths attributable to obesity have either remained constant or even declined during the past twenty years!
xxxv As of January 2005, the CDC reports that the problems with the earlier estimates were due to a “computer error” or a “software error” which seems highly implausible given the state of most statistical software at this point in time.

xxxvi Flegal, K. et al. 2005. “Excess Deaths Associated with Underweight, Overweight, and Obesity.” JAMA 293: 1861-1868. This number is calculated by considering the estimated number of deaths from obesity at around 110,000 a year and the deaths attributable to overweight which is a minus 86,094.


xl For example, see. Wei, M et al. 1999. “Relationship between Low Cardio-respiratory Fitness and Mortality in Normal-Weight, Overweight, and Obese Men.” JAMA 282:1547-1553.


xlii Most of the studies rely on self-reported measures among the respondents particularly regarding their diet, and exercise patterns, if they are asked at all. Other problems include the failure to measure weight over time (or diet and exercise over time), a failure to take smoking into account, and most importantly to elucidate a causal pathway between the fat and disease that can be directly measured. Campos, P. 2004. The Obesity Myth: Why America’s Obsession with Weight is Hazardous to Your Health. New York: Gotham Books. Gaesser 2002.


xlvii Quote from William Dietz.

xlviii In order to gain status as a non-governmental (i.e., non-profit) organization and thus claim official links with the WHO, the IOTF was forced to link itself with the IASO as an “ad hoc committee.”
Although the IOTF resists full disclosure of their funding sources, according the London *Daily Mail*, 75 percent of its million dollar plus budget comes from Hoffman-LaRoche and Abbott. The IOTF has also been supported by Servier the maker of the weight loss drug Redux. The Mail on Sunday. (March 6, 2005).


According to the website of the International Union of Nutritional Sciences, “In June 1997 the WHO, together with the IOTF, held an expert consultation on obesity to review the extent of the obesity problem and examine the need to develop public health policies and programmes [sic] to tackle the global problem of obesity. The consultation resulted in the publication of an interim report: "Obesity - preventing and managing the global epidemic" (WHO 1998) and the subsequent WHO Technical Report Series 894.” This puts a nice spin on what the North American Association for the Study of Obesity calls “the first major initiative of the IOTF.”


Information from the Center for Science in the Public Interest website.

Birmingham, Karen. 1999. “Lawsuit reveals academic conflict-of-interest.” Nature Medicine: 5: 717. Although Pi-Sunyer did not take a fee for the articles he did not write but put his name on, he has been paid numerous consulting fees by other pharmaceutical companies.

Another indication of the power of these recommendations can be seen on the website of Roche Labs. It cites both the WHO report (which they are responsible for authoring) and the low BMI standards for overweight and obese.


For example, Pi-Sunyer, wrote an article in the *Journal of the American Medical Association* that accompanied the 1994 report on rising obesity rates. Locating the sources of obesity primarily in the body he said: “No permanent resolution to this [obesity] problem is likely until we better understand the
underlying biological determinants of obesity . . . To gain this much needed knowledge, *greater support of obesity research* is vitally important” (my italics). As with much of his writing, the paper ends with a call for greater research funding. These scientists and agencies employ a variety of practices to ensure and expand their appropriations, including linking with the private sector (including the diet and drug industries), direct public information campaigns, and with claims to their own expertise. This is particularly the case with the federal bureaucracy. Government agencies justify their budgets and missions relative to their professional expertise: the military assess threats and specifies how much money and men it needs to defend the country; the Department of Transportation evaluates how much is needed for highways, and so on. In the case of obesity this expertise has been used to make claims about the severity of America’s weight problem as well as the funding needed, primarily for more research, about its solution.

lviii Statement by Tommy G. Thompson Secretary Department of Health and Human Services on Preventing Chronic Disease through Healthy Lifestyle before the United States Senate Committee on Appropriations Subcommittee on Labor, Health and Human Services, Education, July 15, 2004.
http://www.hhs.gov/ash/testify/t040715.html

lix Kuczmarski, RJ et al. 1994.

lx Some of the first common usages of the idea of an obesity epidemic occurred in JAMA in 1999 in a special edition on obesity.


The studies referred to are Allison et al., 1999 and Mokdad 2004, which use identical methods. Once again, these were hazard ratios that had enormous error terms and provided very small confidence intervals.

lxiii Ibid.

lxiv Gaesser 1996.


lxvi In Chapter Three, I will review all of the research on discrimination against the obese.