What if It's All Been a Big Fat Lie?

By Gary Taubes
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If the members of the American medical establishment were to have a collective find-yourself-standing-naked this might be it. They spend 30 years ridiculing Robert Atkins, author of the phenomenally-best-selling "Dr. Atkins' Diet Revolution," accusing the Manhattan doctor of quackery and fraud, only to discover that ti along. Or maybe it's this: they find that their very own dietary recommendations -- eat less fat and more car rampaging epidemic of obesity in America. Or, just possibly this: they find out both of the above are true.

When Atkins first published his "Diet Revolution" in 1972, Americans were just coming to terms with the pr saturated fat of meat and dairy products -- was the primary nutritional evil in the American diet. Atkins man book promising that we would lose weight eating steak, eggs and butter to our heart's desire, because it was bagels and sugar, that caused obesity and even heart disease. Fat, he said, was harmless.

Atkins allowed his readers to eat "truly luxurious foods without limit," as he put it, "lobster with butter sauc bacon cheeseburgers," but allowed no starches or refined carbohydrates, which means no sugars or anythin even fruit juices, and permitted only a modicum of vegetables, although the latter were negotiable as the die

Atkins was by no means the first to get rich pushing a high-fat diet that restricted carbohydrates, but he pop American Medical Association considered it a potential threat to our health. The A.M.A. attacked Atkins's d: advocated "an unlimited intake of saturated fats and cholesterol-rich foods," and Atkins even had to defend

Thirty years later, America has become weirdly polarized on the subject of weight. On the one hand, we've b certainty by everyone from the surgeon general on down, and we have come to believe with almost religious the excessive consumption of fat, and that if we eat less fat we will lose weight and live longer. On the other, Atkins and decades' worth of best-selling diet books, including "The Zone," "Sugar Busters" and "Protein Po variation of what scientists would call the alternative hypothesis: it's not the fat that makes us fat, but the ca carbohydrates we will lose weight and live longer.

The perversity of this alternative hypothesis is that it identifies the cause of obesity as precisely those refe famous Food Guide Pyramid -- the pasta, rice and bread -- that we are told should be the staple of our healtl or corn syrup in the soft drinks, fruit juices and sports drinks that we have taken to consuming in quantity it are fat free and so appear intrinsically healthy. While the low-fat-is-good-health dogma represents reality as government has spent hundreds of millions of dollars in research trying to prove its worth, the low-carbohy the realm of unscientific fantasy.

Over the past five years, however, there has been a subtle shift in the scientific consensus. It used to be that alternative hypothesis, let alone researching it, was tantamount to quackery by association. Now a small but researchers have come to take seriously what the low-carb-diet doctors have been saying all along. Walter W nutrition at the Harvard School of Public Health, may be the most visible proponent of testing this heretic h spokesman of the longest-running, most comprehensive diet and health studies ever performed, which have
and include data on nearly 300,000 individuals. Those data, says Willett, clearly contradict the low-fat-is-good-health hypothesis that all fat is bad for you; the exclusive focus on adverse effects of fat may have contributed to the obesity epidemic.

These researchers point out that there are plenty of reasons to suggest that the low-fat-is-good-health hypothesis is misplaced. In particular, that we are in the midst of an obesity epidemic that started around the early 1980s when the rise of the low-fat dogma. (Type 2 diabetes, the most common form of the disease, also rose significantly.) Low-fat weight-loss diets have proved in clinical trials and real life to be dismal failures, and that on top of it American diet has been decreasing for two decades. Our cholesterol levels have been declining, and we have incidence of heart disease has not declined as would be expected. "That is very disconcerting," Willett says. "We are in the midst of an obesity epidemic that started around the early 1980s when the rise of the low-fat dogma. (Type 2 diabetes, the most common form of the disease, also rose significantly.) Low-fat weight-loss diets have proved in clinical trials and real life to be dismal failures, and that on top of it American diet has been decreasing for two decades. Our cholesterol levels have been declining, and we have incidence of heart disease has not declined as would be expected. "That is very disconcerting," Willett says."

The science behind the alternative hypothesis can be called Endocrinology 101, which is how it's referred to by Harvard Medical School who runs the pediatric obesity clinic at Children's Hospital Boston, and who prescribe carbohydrate-restricted diet to his patients. Endocrinology 101 requires an understanding of how carbohydrates affect insulin and blood sugar (and in turn fat metabolism and appetite). This is basic endocrinology, Ludwig says, which is the study of hormones, and it is still considered radical because the low-fat dietary wisdom emerged in the 1960's from researchers almost exclusively concerned with the regulation of cholesterol and heart disease. At the time, Endocrinology 101 was still underdeveloped, and so it was ignored clear, it has to fight a quarter century of anti-fat prejudice.

The alternative hypothesis also comes with an implication that is worth considering for a moment, because it is an obstacle to its acceptance. If the alternative hypothesis is right -- still a big "if" -- then it strongly suggests that in America and elsewhere is not, as we are constantly told, due simply to a collective lack of will power and laziness. It occurred, as Atkins has been saying (along with Barry Sears, author of "The Zone"), because the public health authorities told us unwittingly, but collectively quite effectively, that all fat is bad for you; the exclusive focus on adverse effects of fat may have contributed to the obesity epidemic. Researchers will be suitably scientific if the alternative hypothesis is right, then a low-fat diet is in practice, such a diet cannot help being high in carbohydrates, and that can lead to obesity, and perhaps even heart disease. At the time, Endocrinology 101 was still underdeveloped, and so it was ignored clear, it has to fight a quarter century of anti-fat prejudice.

Scientists are still arguing about fat, despite a century of research, because the regulation of appetite and weight in the human body happens to be almost inconceivably complex, and the experimental tools we have to study it are still remarkably inadequate. This combination leaves researchers in an awkward position. To study the entire physiological system involves feeding real food to real people on a regular basis, which is prohibitively expensive, ethically questionable (if you're trying to measure the effects of foods that might cause disease) and virtually impossible to do in any kind of rigorously controlled scientific manner. But if researchers seek to study something less costly and more controllable, they end up studying experimental situations so oversimplified that their results are questionable. This then leads to a research literature so vast that it's possible to find at least some published research on any topic of interest. The result is a balkanized community -- "splintered, very opinionated and in many instances, intransigent," according to the Food and Nutrition Board of the National Academy of Science -- in which researchers seem preconceived notions are correct and thoroughly uninterested in testing any other hypotheses but their own.

What's more, the number of misconceptions propagated about the most basic research can be staggering. Researchers describing the limitations of their own experiments, and then will cite something as gospel truth because the example is the statement heard repeatedly that 95 percent of all dieters never lose weight, and 95 percent of This will be correctly attributed to the University of Pennsylvania psychiatrist Albert Stunkard, but it will go unquestioned, even if the data is based on 100 patients who passed through Stunkard's obesity clinic during the Eisenhower administration.
With these caveats, one of the few reasonably reliable facts about the obesity epidemic is that it started around the early 1980's. According to Katherine Flegal, an epidemiologist at the National Center for Health Statistics, the percentage of obese Americans stayed relatively constant through the 1960's and 1970's at 13 percent to 14 percent and then shot up by 8 percentage points in the 1980's. By one in four Americans was obese. That steep rise, which is consistent through all segments of American society through the 1990's, is the singular feature of the epidemic. Any theory that tries to explain obesity in America has to account for that. Meanwhile, overweight children nearly tripled in number. And for the first time, physicians began diagnosing Type 2 diabetes, which often accompanies obesity. It used to be called adult-onset diabetes and now, for the obvious reason, is not.

So how did this happen? The orthodox and ubiquitous explanation is that we live in what Kelly Brownell, a Yale food environment' of cheap fatty food, large portions, pervasive food advertising and sedentary lives. By this theory, we are at the mercy of the food industry, which spends nearly $10 billion a year advertising unwholesome junk food and especially fast food, are so filled with fat, they are both irresistible and uniquely fattening. On top of this, so has successfully eliminated physical activity from our daily lives. We no longer exercise or walk up stairs, nor do our children bike to school or play outside, because they would prefer to play video games and watch television. And because some of us are weight while others are not, this explanation also has a genetic component -- the thrifty gene. It suggests that evolutionary advantage to our Paleolithic ancestors, who had to survive frequent famine. We then inherited liability in today's toxic environment.

This theory makes perfect sense and plays to our puritanical prejudice that fat, fast food and television are inherently evil. But there are two catches. First, to buy this logic is to accept that the copious negative reinforcement that accompanies obesity and physically -- is easily overcome by the constant bombardment of food advertising and the lure of a supersize bargain meal. And second, as Flegal points out, little data exist to support any of this. Certainly none of it explains what changed so significantly to start the epidemic. Fast food consumption, for example, continued to grow steadily through the 70's and 80's, but it did not take a sudden leap, as obesity did.

As far as exercise and physical activity go, there are no reliable data before the mid-80's, according to William Dietz, who runs the division of nutrition and physical activity at the Centers for Disease Control; the 1990's data show obesity rates continued to remain unchanged. This suggests the two have little in common. Dietz also acknowledged that a culture of physical exercise was in the United States in the 70's -- the "leisure exercise mania," as Robert Levy, director of the National Heart, Lung and Blood Institute, described it in 1981 -- and has continued through the present day.

As for the thrifty gene, it provides the kind of evolutionary rationale for human behavior that scientists find hard to test. In other words, if we were living through an anorexia epidemic, the experts would be discussing the equally untestable "spendthrift gene" theory, touting evolutionary advantages of losing weight effortlessly. An overweight homo erectus, they'd say, would have been easy prey for predators.

It is also undeniable, note students of Endocrinology 101, that mankind never evolved to eat a diet high in starches or sugars. "Grain products and concentrated sugars were essentially absent from human nutrition until the invention of agriculture," Ludwig says, "which was only 10,000 years ago." This is discussed frequently in the anthropology texts but is mostly absent from the obesity literature, with the prominent exception of the low-carbohydrate-diet books.

What's forgotten in the current controversy is that the low-fat dogma itself is only about 25 years old. Until now was that fat and protein protected against overeating by making you sated, and that carbohydrates made you hungry for instance, an 1825 discourse considered among the most famous books ever written about food, the French gastronome Jean Anthelme Brillat-Savarin says that he could easily identify the causes of obesity after 30 years of listening to one "stout party" bread, rice and (from a "particularly stout party") potatoes. Brillat-Savarin described the roots of obesity as
with the "floury and feculent substances which man makes the prime ingredients of his daily nourishment." fecula -- i.e., "potatoes, grain or any kind of flour" -- were seen sooner when sugar was added to the diet.

This is what my mother taught me 40 years ago, backed up by the vague observation that Italians tended to much pasta. This observation was actually documented by Ancel Keys, a University of Minnesota physician staying power," by which he meant they are slow to be digested and so lead to satiation, and that Italians we had studied. According to Keys, the Neapolitans, for instance, ate only a little lean meat once or twice a weee: for lunch and dinner. "There was no evidence of nutritional deficiency," he wrote, "but the working-class we By the 70's, you could still find articles in the journals describing high rates of obesity in Africa and the Cari exclusively carbohydrates. The common thinking, wrote a former director of the Nutrition Division of the U one that prevented obesity, snacking and excessive sugar consumption, was a diet "with plenty of eggs, beef cooked vegetables." This was the identical prescription Brillat-Savarin put forth in 1825.

It was Ancel Keys, paradoxically, who introduced the low-fat-is-good-health dogma in the 50's with his theo levels and gives you heart disease. Over the next two decades, however, the scientific evidence supporting th ambiguous. The case was eventually settled not by new science but by politics. It began in January 1977, wh McGovern published its "Dietary Goals for the United States," advising that Americans significantly curb th "killer diseases" supposedly sweeping the country. It peaked in late 1984, when the National Institutes of H Americans over the age of 2 eat less fat. By that time, fat had become "this greasy killer" in the memorable v Public Interest, and the model American breakfast of eggs and bacon was well on its way to becoming a bow glass of orange juice and toast, hold the butter -- a dubious feast of refined carbohydrates.

In the intervening years, the N.I.H. spent several hundred million dollars trying to demonstrate a connectio disease and, despite what we might think, it failed. Five major studies revealed no such link. A sixth, howeve alone, concluded that reducing cholesterol by drug therapy could prevent heart disease. The N.I.H. adminis Basil Rifkind, who oversaw the relevant trials for the N.I.H., described their logic this way: they had failed to eating less fat had any health benefits. But if a cholesterol-lowering drug could prevent heart attacks, then a should do the same. "It's an imperfect world," Rifkind told me. "The data that would be definitive is ungetta available."

Some of the best scientists disagreed with this low-fat logic, suggesting that good science was incompatible effectively ignored. Pete Ahrens, whose Rockefeller University laboratory had done the seminal research on McGovern's committee that everyone responds differently to low-fat diets. It was not a scientific matter who harmed, he said, but "a betting matter." Phil Handler, then president of the National Academy of Sciences, t effect in 1980. "What right," Handler asked, "has the federal government to propose that the American peo experiment, with themselves as subjects, on the strength of so very little evidence that it will do them any gc

Nonetheless, once the N.I.H. signed off on the low-fat doctrine, societal forces took over. The food industry of reduced-fat food products to meet the new recommendations. Fat was removed from foods like cookies, c had to be replaced with something as tasty and pleasurable to the palate, which meant some form of sugar. { Meanwhile, an entire industry emerged to create fat substitutes, of which Procter & Gamble's olestra was fir meats, cheeses, snacks and cookies had to compete with a few hundred thousand other food products marks dedicated considerable advertising effort to reinforcing the less-fat-is-good-health message. Helping the cat "huge forces" of dietitians, health organizations, consumer groups, health reporters and even cookbook wrt healthful eating.

Few experts now deny that the low-fat message is radically oversimplified. If nothing else, it effectively ignores the fact that olive oil, are relatively good for you: they tend to elevate your good cholesterol, high-density lipoprotein (H.D.L.), at least in comparison to the effect of carbohydrates. While higher L.D.L. reduces it.

What this means is that even saturated fats -- a.k.a., the bad fats -- are not nearly as deleterious as you would think. True, bad cholesterol, but they will also elevate your good cholesterol. In other words, it's a virtual wash. As Willett explained to me, there is no health benefit by giving up milk, butter and cheese and eating bagels instead.

But it gets even weirder than that. Foods considered more or less deadly under the low-fat dogma turn out to actually look at their fat content. More than two-thirds of the fat in a porterhouse steak, for instance, will depress your triglyceride profile (at least in comparison with the baked potato next to it); it's true that the remainder will raise your L.D.L. The same is true for lard. If you work out the numbers, you come to the surreal conclusion that you can eat lard straight out of the can and conceivably reduce your risk of heart disease.

The crucial example of how the low-fat recommendations were oversimplified is shown by the impact -- pot diets on triglycerides, which are the component molecules of fat. By the late 60's, researchers had shown that these are common in heart-disease patients as high L.D.L. cholesterol, and that eating a low-fat, high-carbohydrate diet would, for many people, raise their triglyceride levels, lower their H.D.L. levels and accentuate what Gerry Reaven, an endocrinologist at Stanford University, called Syndrome X. This is a cluster of conditions that can lead to heart disease and Type 2 diabetes.

It took Reaven a decade to convince his peers that Syndrome X was a legitimate health concern, in part because low-fat diets will increase the risk of heart disease in a third of the population. "Sometimes we wish it wasn't how to deal with it," said Robert Silverman, an N.I.H. researcher, at a 1987 N.I.H. conference. "High protein. High fat is bad for your heart. Now Reaven is saying not to eat high carbohydrates. We have to eat something."

Surely, everyone involved in drafting the various dietary guidelines wanted Americans simply to eat less junk food the way they do in Berkeley, Calif. But we didn't go along. Instead we ate more starches and refined carbohydrates, because calorie for calorie, these are the cheapest nutrients for the food industry to produce, and they can be sold at the highest profit. Rare is the person under the age of 50 who doesn't prefer a cookie or heavily sweetened yogurt to a head of broccoli.

"All reformers would do well to be conscious of the law of unintended consequences," says Alan Stone, who was staff director for McGovern's Senate committee. Stone told me he had an inkling about how the food industry would respond to the new dietary goals back when the hearings were first held. An economist pulled him aside, he said, and gave him a lesson on market disincentives to healthy eating: if you are the first to create a new market with a brand-new manufactured food, give it a brand-new fancy name, put a big advertising budget behind it, you can have a virtual monopoly of the market and force your competitors to catch up. You can't do that with fruits and vegetables. It's harder to differentiate an apple from an apple."

Nutrition researchers also played a role by trying to feed science into the idea that carbohydrates are the ideal nutrient. It had been almost a century, and considered mostly irrelevant to the etiology of obesity, that fat has nine calories per gram compared with four for carbohydrates and protein. Now it became the fail-safe position of the low-fat recommendations: reduce the amount and you will lose weight. Then in 1982, J.P. Flatt, a University of Massachusetts biochemist, published his research demonstrating that, in any normal diet, it is extremely rare for the human body to convert carbohydrates into body fat. This was then a few scientists to mean that eating carbohydrates, even to excess, could not make you fat -- which is not the misinterpretation developed a vigorous life of its own because it resonated with the notion that fat makes you harmless.
As a result, the major trends in American diets since the late 70’s, according to the U.S.D.A. agricultural eco
decrease in the percentage of fat calories and a "greatly increased consumption of carbohydrates." To be pre
increased almost 60 pounds per person, and caloric sweeteners (primarily high-fructose corn syrup) by 301
suddenly began consuming more total calories: now up to 400 more each day since the government started

If these trends are correct, then the obesity epidemic can certainly be explained by Americans’ eating more45
after all, are what causes us to gain weight -- and, specifically, more carbohydrates. The question is why?

The answer provided by Endocrinology 101 is that we are simply hungrier than we were in the 70’s, and the
psychological. In this case, the salient factor -- ignored in the pursuit of fat and its effect on cholesterol -- is
and insulin. In fact, these were obvious culprits all along, which is why Atkins and the low-carb-diet doctors

The primary role of insulin is to regulate blood-sugar levels. After you eat carbohydrates, they will be broken
molecules and transported into the bloodstream. Your pancreas then secretes insulin, which shunts the bloo
fuel for the next few hours. This is why carbohydrates have a significant impact on insulin and fat does not.
caused by a lack of insulin, physicians believed since the 20's that the only evil with insulin is not having en

But insulin also regulates fat metabolism. We cannot store body fat without it. Think of insulin as a switch. ’
eating, you burn carbohydrates for energy and store excess calories as fat. When it's off, after the insulin has
So when insulin levels are low, you will burn your own fat, but not when they're high.

This is where it gets unavoidably complicated. The fatter you are, the more insulin your pancreas will pump
you'll develop what's called "insulin resistance," which is the underlying cause of Syndrome X. In effect, you
action of insulin, and so you need ever greater amounts to keep your blood sugar in check. So as you gain w
fat and harder to lose it. But the insulin resistance in turn may make it harder to store fat -- your weight is b
But now the insulin resistance might prompt your pancreas to produce even more insulin, potentially starti
- the obesity, the elevated insulin, known as hyperinsulinemia, or the insulin resistance -- is a chicken-and-t
resolved. One endocrinologist described this to me as "the Nobel-prize winning question."

Insulin also profoundly affects hunger, although to what end is another point of controversy. On the one ha
hunger by lowering your blood sugar, but how low does blood sugar have to drop before hunger kicks in? Th
works in the brain to suppress hunger. The theory, as explained to me by Michael Schwartz, an endocrinolog
is that insulin's ability to inhibit appetite would normally counteract its propensity to generate body fat. In c
your body would generate more insulin after every meal, and that in turn would suppress your appetite; you

Schwartz, however, can imagine a simple mechanism that would throw this "homeostatic" system off balance
sensitivity to insulin, just as your fat and muscles do when they are flooded with it. Now the higher insulin p
fatter would no longer compensate by suppressing your appetite, because your brain would no longer regist
would be a physiologic state in which obesity is almost preordained, and one in which the carbohydrate-insul
role. Schwartz says he believes this could indeed be happening, but research hasn't progressed far enough to
says. "It still needs to be sorted out."

David Ludwig, the Harvard endocrinologist, says that it's the direct effect of insulin on blood sugar that doe
diabetics get too much insulin, their blood sugar drops and they get ravenously hungry. They gain weight be
promotes fat deposition. The same happens with lab animals. This, he says, is effectively what happens whe
particular sugar and starches like potatoes and rice, or anything made from flour, like a slice of white bread.
high-glycemic-index carbohydrates, which means they are absorbed quickly into the blood. As a result, they
surge of insulin within minutes. The resulting rush of insulin stores the blood sugar away and a few hours la

was before you ate. As Ludwig explains, your body effectively thinks it has run out of fuel, but the insulin is burning your own fat. The result is hunger and a craving for more carbohydrates. It's another vicious circle, obesity.

The glycemic-index concept and the idea that starches can be absorbed into the blood even faster than sugar had no influence on public health recommendations, because of the attendant controversies. To wit: if you then you had to accept that the starches we were supposed to be eating 6 to 11 times a day were, once swallowed indistinguishable from sugars. This made them seem considerably less than wholesome. Rather than accept simply allowed sugar and corn syrup to elude the vilification that befell dietary fat. After all, they are fat-free.

Sugar and corn syrup from soft drinks, juices and the copious teas and sports drinks now supply more than 80's saw the introduction of Big Gulps and 32-ounce cups of Coca-Cola, blasted through with sugar, but 10c insulin and blood sugar, these soft drinks and fruit juices -- what the scientists call "wet carbohydrates" -- π accounts for less than a quarter of the soda market.)

The gist of the glycemic-index idea is that the longer it takes the carbohydrates to be digested, the lesser the and the healthier the food. Those foods with the highest rating on the glycemic index are some simple sugar flour. Green vegetables, beans and whole grains cause a much slower rise in blood sugar because they have which slows down digestion and lowers the glycemic index. Protein and fat serve the same purpose, which is beneficial, a notion that is still unacceptable. And the glycemic-index concept implies that a primary cause of diabetes and obesity is the long-term damage caused by the repeated surges of insulin that come from eating. This suggests a kind of unified field theory for these chronic diseases, but not one that coexists easily with the

At Ludwig's pediatric obesity clinic, he has been prescribing low-glycemic-index diets to children and adolescents: recommend the Atkins diet because he says he believes such a very low carbohydrate approach is unnecessary patients to effectively replace refined carbohydrates and starches with vegetables, legumes and fruit. This managed to convince the N.I.H. that such diets are worthy of study. His first three grant proposals were sun why much of the relevant research has been done in Canada and in Australia. In April, however, Ludwig rec test his low-glycemic-index diet against a traditional low-fat-low-calorie regime. That might help resolve so insulin in obesity, although the redoubtable Robert Atkins might get there first.

The 71-year-old Atkins, a graduate of Cornell medical school, says he first tried a very low carbohydrate diet the Journal of the American Medical Association. He lost weight effortlessly, had his epiphany and turned a practice into a thriving obesity clinic. He then alienated the entire medical community by telling his readers they wanted, as long as they ate little to no carbohydrates. They would lose weight, he said, because they wouldn't be hungry; and they would have less resistance to burning their own fat. Atkins also noted that sta event because they raised triglyceride levels and that this was a greater risk factor for heart disease than cho Atkins's diet is both the ultimate manifestation of the alternative hypothesis as well as the battleground on which controversy is likely to be fought scientifically over the next few years. After insisting Atkins was a quack for now finding it difficult to ignore the copious anecdotal evidence that his diet does just what he has claimed. Stunkard has been trying to treat obesity for half a century, but he told me he had his epiphany about Atkins recently when he discovered that the chief of radiology in his hospital had lost 60 pounds on Atkins's diet. "the hospital are doing it," he said. "So we decided to do a study." When I asked Stunkard if he or any of his colleagues considered testing Atkins's diet to children and adolescents for five years now. He does...
diet 30 years ago, he said they hadn't because they thought Atkins was "a jerk" who was just out to make nobody took him seriously enough to do what we're finally doing."

In fact, when the American Medical Association released its scathing critique of Atkins's diet in March 1973, probably worked, but expressed little interest in why. Through the 60's, this had been a subject of consider Atkins-like diets were low-calorie diets in disguise; that when you cut out pasta, bread and potatoes, you'll h vegetables and cheese to replace the calories.

That, however, raised the question of why such a low-calorie regimen would also suppress hunger, which At characteristic of the diet. One possibility was Endocrinology 101: that fat and protein make you sated and, l swings of blood sugar and insulin, you stay sated. The other possibility arose from the fact that Atkins's diet insulin falls so low that you enter a state called ketosis, which is what happens during fasting and starvation fat for energy, as does your brain in the form of fat molecules produced by the liver called ketones. Atkins se start weight loss. He also liked to say that ketosis was so energizing that it was better than sex, which set hin criticism of Atkins's diet has been that ketosis is dangerous and to be avoided at all costs.

When I interviewed ketosis experts, however, they universally sided with Atkins, and suggested that maybe media confuse ketosis with ketoacidosis, a variant of ketosis that occurs in untreated diabetics and can be fa says Richard Veech, an N.I.H. researcher who studied medicine at Harvard and then got his doctorate at Ox Laureate Hans Krebs. "They're always worried about diabetic ketoacidosis. But ketosis is a normal physiolog normal state of man. It's not normal to have McDonald's and a delicatessen around every corner. It's normal Simply put, ketosis is evolution's answer to the thrifty gene. We may have evolved to efficiently store fat for also evolved ketosis to efficiently live off that fat when necessary. Rather than being poison, which is how th make the body run more efficiently and provide a backup fuel source for the brain. Veech calls ketones "mag heart and brain run 25 percent more efficiently on ketones than on blood sugar.

The bottom line is that for the better part of 30 years Atkins insisted his diet worked and was safe, Ameri millions, while nutritionists, physicians, public- health authorities and anyone concerned with heart disease expressed little or no desire to find out who was right. During that period, only two groups of U.S. research published their results. In the early 70's, J.P. Flatt and Harvard's George Blackburn pioneered the "protein- postsurgical patients, and they tested it on obese volunteers. Blackburn, who later became president of the Nutrition, describes his regime as "an Atkins diet without excess fat" and says he had to give it a fancy name. The diet was "lean meat, fish and fowl" supplemented by vitamins and minerals. "People loved it," Blackbu couldn't run them off with a baseball bat." Blackburn successfully treated hundreds of obese patients over tl of papers that were ignored. When obese New Englanders turned to appetite-control drugs in the mid-80's, applied to the N.I.H. for a grant to do a clinical trial of popular diets but was rejected.

The second trial, published in September 1980, was done at the George Washington University Medical Ce agreed to follow Atkins's diet for eight weeks and lost an average of 17 pounds each, with no apparent ill eff did go up. The researchers, led by John LaRosa, now president of the State University of New York Downstz concluded that the 17-pound weight loss in eight weeks would likely have happened with any diet under "the experimental conditions" and never pursued it further.

Now researchers have finally decided that Atkins's diet and other low-carb diets have to be tested, and are d -low-fat diets as recommended by the American Heart Association. To explain their motivation, they inevi Stunkard, told me that someone they knew -- a patient, a friend, a fellow physician -- lost considerable weig
their preconceptions to the contrary, kept it off. Others say they were frustrated with their inability to help t low-carb diets and decided that Endocrinology 101 was compelling. "As a trained physician, I was trained to says Linda Stern, an internist at the Philadelphia Veterans Administration Hospital, "but I put myself on th maybe this is something I can offer my patients."

None of these studies have been financed by the N.I.H., and none have yet been published. But the results h by researchers at Schneider Children's Hospital on Long Island, Duke University and the University of Cinc Philadelphia V.A. Hospital. And then there's the study Stunkard had mentioned, led by Gary Foster at the U director of the Center for Human Nutrition at Washington University in St. Louis, and Jim Hill, who runs t Human Nutrition in Denver. The results of all five of these studies are remarkably consistent. Subjects on so overweight adolescents on the diet for 12 weeks as at Schneider, or obese adults averaging 295 pounds on t Philadelphia V.A. -- lost twice the weight as the subjects on the low-fat, low-calorie diets.

In all five studies, cholesterol levels improved similarly with both diets, but triglyceride levels were consider Though researchers are hesitant to agree with this, it does suggest that heart-disease risk could actually be r the diet and starches and refined carbohydrates are removed. "I think when this stuff gets to be recognized,' shake up a lot of thinking about obesity and metabolism."

All of this could be settled sooner rather than later, and with it, perhaps, we might have some long-awaited : whether it is indeed preordained by societal forces or by our choice of foods. For the first time, the N.I.H. is studies of popular diets. Foster, Klein and Hill, for instance, have now received more than $2.5 million from Atkins diet with 360 obese individuals. At Harvard, Willett, Blackburn and Penelope Greene have money, al foundation, to do a comparative trial as well.

Should these clinical trials also find for Atkins and his high-fat, low-carbohydrate diet, then the public-health problem on their hands. Once they took their leap of faith and settled on the low-fat dietary dogma 25 years contradictory evidence or a change of opinion, should such a change be necessary to keep up with the sci experience is noteworthy. Klein is president-elect of the North American Association for the Study of Obesit respected member of his community. And yet, he described his recent experience discussing the Atkins diet experience. "I have been impressed," he said, "with the anger of academicians in the audience. Their respon on the Atkins diet!"

This hostility stems primarily from their anxiety that Americans, given a glimmer of hope about their weigh that simply seems intuitively dangerous and on which there is still no long-term data on whether it works at fear. In the course of my research, I have spent my mornings at my local diner, staring down at a plate of sci that somehow, some way, they must be working to clog my arteries and do me in.

After 20 years steeped in a low-fat paradigm, I find it hard to see the nutritional world any other way. I have clinical trials and in real life, and they certainly have failed in my life. I have read the papers suggesting that have not managed to lower the incidence of heart disease in this country, and may have led instead to the st diabetes. I have interviewed researchers whose computer models have calculated that cutting back on the sc recommended by the American Heart Association would not add more than a few months to my life, if that. with relative ease by giving up carbohydrates on my test diet, and yet I can look down at my eggs and sausag onset of heart disease and obesity, the latter assuredly to be caused by some bizarre rebound phenomena th begun to describe. The fact that Atkins himself has had heart trouble recently does not ease my anxiety, des related.
This is the state of mind I imagine that mainstream nutritionists, researchers and physicians must inevitably take to the controversy. They may come around, but the evidence will have to be exceptionally compelling. Although this is happening at the moment to John Farquhar, who is a professor of health research and policy at Stanford University and has worked in this field for more than 40 years. When I interviewed Farquhar in April, he explained why low-fat diets might lead to weight gain and low-fat diets might lead to weight loss, but he made me promise not to say he believed they did. He attributed the cause to "force-feeding of a nation." Three weeks later, after reading an article on Endocrinology 101 by David Ludwig in the Journal of Medical Association, he sent me an e-mail message asking the not-entirely-rhetorical question, "Can we get..."