Taubes: Okay, I'm Gary Taubes. I'm a co-founder of the Nutrition Science Initiative. I'm, you know, a longtime investigative journalist and author, and I'm also a contributor to CrossFit Health. And I'm here with Barry Sears, author of *The Zone* — the diet philosophy that CrossFit recommends — and CrossFit labs, creator of CrossFit labs. And we're gonna talk, maybe argue a little — find out a lot about each other. See how it goes. And as I was telling Barry, I've never done this before. I've been an interviewee. I've never been an interviewer, so —

Sears: You're doing a great job.

Taubes: Oh thank you, Barry. Good, good start. Okay, well, so tell me — let's start. First of all, you're here in L.A. for your 50th college reunion, and there was a time in your life when you, I must have assumed, you would never have a 50th college reunion.

Sears: And that's probably a very good assumption, because I have a long family history of heart disease. My father died in his early 50s of heart disease, and he was a world-class athlete. All of his brothers died in their early 50s of heart disease, as did my grandfather. So, I knew early on when my father died, I had a very strong genetic likelihood of also having an early death from heart disease. I had two choices: I could live fast, die young, or I could do something to solve the problem. The question is: What causes heart disease? It turns out, at the time I was at Boston University Medical School, and their two primary philosophies, really waging a war — one were the cholesterol boys —

Taubes: So tell me what era this was, by the way.

Sears: This was about in the 19— in 1972. I was at BU medical school about a year later, and there was the cholesterol boys who said, “Cholesterol causes heart disease,” and then there was the inflammation guys, which I was part of, saying, “No, it's caused by inflammation.” This is like the Miller Lite commercial: “Tastes great, less filling. Tastes great, less filling.” The problem was it's very hard to describe inflammation. It's a very complex area. And cholesterol? It's easier to say, “I can touch it.” And eventually, then, when they basically had cholesterol testing, they say, “Good, we can show that this proves conclusively that cholesterol causes heart disease.” The data was weak and pathetic, but the fact is, therefore, if it's weak and pathetic, then off-send the inflammation story. But again, the complexity of information made it very difficult. There's no test for inflammation. You can measure cholesterol, but now we can look back nearly 40 years later, and now we can definitely say the underlying cause of heart disease, and virtually all chronic disease, is inflammation.
Taubes: Okay, well, let's step back a little because one of the issues is there were two competing hypotheses — maybe there should have been more than two competing hypotheses — so as these were growing up, you had what we'll call, using your terminology, “the insulin resistance boys.” So, Reaven and his crew at Stanford are doing their research, and this is a story that dates back to the 1950s. So, how does that fit into it? Because insulin resistance certainly associates with inflammation. I know you —

Sears: Insulin resistance is caused by inflammation. Insulin resistance is something that's easy to measure, but again, the underlying cause of insulin resistance is increased inflammation, causing distortions in the signaling of the insulin signal —

Taubes: There are more — There are multiple hypotheses. We're gonna start arguing from the get-go. There are multiple hypotheses about the cause. So, there's the fat accumulation and what — we agree that it begins in the liver — initiates. Although, you've argued in the article you sent me that it begins in the hypothalamus, which triggers the liver, so, have to work back and forth in this biological system.

Sears: But we'll even go a little further back in time, because I have no background in nutrition. In fact, I could have cared less about it. My interest was in heart disease, and from that I morphed into the areas of intravenous drug delivery systems for cancer, and that became a very fascinating area: how to basically reduce the toxicity of cancer drugs. You try to keep them in a therapeutic zone. A form of a pharmacology standpoint that says if you give too little of a drug, it doesn't work; you give too much, it's toxic. And when the Nobel Prize in medicine was awarded in 1982 for understanding the role of a certain group of hormones in the inflammatory process — these are called eicosanoids — I said, “That's it,” that now we can basically — now look to basically use, hopefully, the diet as a drug to keep these inflammatory eicosanoids in an appropriate therapeutic zone: not too high, but not too low. And from that concept 40 years ago, it increases — our knowledge of the inflammatory processes has increased to say how different aspects of our diet will in fact affect the turning on of inflammation, but more importantly, the turning off.

Taubes: So one — one question I had, because one thing that clearly resonated in your book — you're manipulating the eicosanoids by dietary manipulation of insulin and glucagon. Insulin, glucagon, we can measure. The eicosanoids, you point out, are very short-lived and very difficult to measure, and I assume that when you measure them and during the course of a day is going to make a huge difference. And so —

Sears: Over the course of an hour.
Taubes: Right — So how does that — ? I mean, the end product that you're trying to manipulate is eicosanoids, but how would it be different if you said the end product you were trying to manipulate was just insulin and glucagon, and left the eicosanoid story out?

Sears: You couldn't. Because again, the eicosanoid story — initially, I thought I could manipulate the balance of omega-3 to omega-6 fatty acids. And because those are the building blocks of both the inflammatory eicosanoids, but equally important the pro-resolution hormones that balance the effects. But I found that was not effective unless I was able to control insulin and glucagon at the same time. So, I was forced to invent the Zone diet to get a better control of both the eicosanoids, which cause inflammation and drive inflammation, and these other hormones called resolvents, which turn off inflammation.

Taubes: I only saw the term resolvents in your later article. But when you're talking about resolvents you're talking about good eicosanoids as opposed to bad.

Sears: Well again, the term eicosanoids of — is one dealing with hormones that come only from arachidonic acid and EPA. We think of EPA, the omega-3 fatty acid as an anti-inflammatory fatty acid. But in reality, the eicosanoids coming from EPA actually are very weak pro-inflammatory. But by tipping 100 times weaker in terms of inflammatory responses than those coming from arachidonic acid, they look good in comparison. The understanding and discovery of resolvents didn't take place until 2001. So that's why, again, my book the *Omega Rx Zone*, which was published in 2001, probably was the first popular book ever to put forward the concept of these resolvents and their role in the resolution of inflammation.

Taubes: Okay, so now let's go backward to how we understand that, again, the insulin resistance — my understanding circa 2009 is there were multiple hypotheses. That it was one of these interesting fields where the group at Harvard, Houghton's, Mistelgau, who had the inflammation hypothesis, disagreed with the group at Yale, which was Schulman, which had the diacylglycerol fatty acid accumulation, hepatocytes, and there was a group — And the West Coast had a different hypothesis. They all had animal models to support their hypotheses and refute their competitors’ hypotheses. I'm a fan of the fat accumulation hypothesis because it fits my preconceptions, but you're a fan of the inflammation hypothesis.

Sears: I'm a fan of all those. I say it all —

Taubes: When we talk about causal, when we say inflammation causes it as opposed to diacylglycerol accumulation causes it, and all these things are happening simultaneously.
Sears: And they're all interrelated. So in many ways, you have the equivalent of 12 blind men trying to describe an elephant. One grabs the trunk and says, “It's a rope.” Another grabs the leg and says, “No, it's a tree trunk.” They're all partially right. And so, that's why you had to go deeper down and say, “Okay, what is the goal in terms of — ?” We have to have inflammation. If we don't have any inflammation, we'd be a sitting target for microbes. Our physical injuries will never heal. But we have to turn it off. And so, again, once these resolvents were discovered in 2001, that becomes now the balance. So now we can define the Zone, now circa 2018. What is the Zone? It's simply the balance of turning on inflammation, and turning off. If they're balanced, then you basically maintain that inflammatory response in every cell in your body, in our appropriate therapeutic zone. And by doing so, you've done everything in your power to basically minimize the impact of two things we do on a daily basis that can increase inflammation: One is the food we eat, and two, the level of exercise we do.

Taubes: So, the level of exercise can decrease inflammation?

Sears: No, it can increase it.

Taubes: Okay. I was thinking you don't have my knees if you think it could decrease it.

Sears: Well again, let's now go from the aspect of CrossFit. The people who basically are CrossFit enthusiasts, they live in the world of inflammation by choice. Most of us try to avoid that world, but they live in it by choice, and therefore, their biggest goal in life is to turn off the inflammatory responses caused by the intensity of their training. Exactly the same problem that Olympic athletes have, and that's why my initial test subjects were not diabetics, who were not heart patients; they were Olympic athletes. They say, “If you could turn off the inflammatory response, you can help them recover at a faster rate.” You can help them basically have a now significant improvement of performance. Now, that can translate into the cardiovascular patient or the diabetic patient. The reason I chose world-class athletes: I could do human experimentation and have less likelihood of having mistakes.

Taubes: All right. So, when you put people on a Zone-favorable diet, they're going — again, you're gonna have a lot of changes in the system. Primarily, it's going to be modulated with — first, you'll be modulating insulin and glucagon and other hormonal responses. I'm always curious when I read about the kind of studies you did, first of all, what these athletes reading beforehand, and so how much of what the improvement they say is because of a general improvement in diet? Without a control group, we don't know. We could have put him on, I mean an example using
your book, but a macrobiotic diet, and I think we'd both assume they would do worse, but that's an assumption. So, a lot of things are changing when you change to your Zone-favorable diet. Can you kind of describe to me what they were consuming before and then what — or if we compared the Stanford team with the University of Texas team, how they're —

Sears: Well actually, we could actually compare the Stanford team to the Stanford team. When I first brought the concept to the Stanford swim coaches, it was based on my — a lot of my work was done with the NFL. And so, they were interested, and I said, “Well this is great. Let's do a placebo-controlled study.” They said, “Are you kidding? Are you kidding? We know the stuff works.” They knew the stuff worked, but telling athletes what to eat, that's like touching the third rail. So not all the swimmers at Stanford bought into it, the concept. Only a few did. But the few who did were the ones who won all the gold medals in Barcelona. Okay, not a controlled experiment, but, you know, the best we can do at the time. What does the standard athlete's diet in the, let's say, early ’90s? Eat lots of carbohydrates. Now say, what — well, but look at swimmers. They look great in swimsuits. They're chiseled of — but because, again, the high levels of intensity could overcome some of the hormonal consequences of that diet. But for those who basically had the same high level of intensity, that didn't change. But we basically had changed their diet. Those are the ones who basically had this remarkable change. I'll use a couple examples. One is Pablo Morales. For the previous three years, he'd been studying in law school. Not the best way to train for the Olympics. And he came back and was, you know, you know, incredibly fastidious. Every morning, the same meal: a bowl of oatmeal, an eight-egg-white omelet. Morning in, morning out. No change. Oh, he won two gold medals at the Barcelona Olympics. Another case was Jenny Thompson. She was a very good swimmer, international, but she came to my laboratory because of the swim coach said — sent — “Have her come down.” I measured Jenny's body fat. I said, “Jenny, you're fat for a world-class athlete.” She says, “How can I be? I'm eating Power Bars all the time.” At the — Power Bars were all carbohydrates. But remember, she's a teenager, but her mother came with her. Her mother was a nurse, and she was a diabetic. So what I was telling Jenny resonated very well with her mother. And so, as of course, teenagers, the mother makes all the foods. And so I saw Jenny six months later at Colorado Springs. She had basically — had changed completely, basically. And in that year, everyone accused her of being on anabolic steroids. They said, “Last year you were chunky. This year you are ripped.” And the reason was because, again, she had followed the Zone diet. Data points, one or two. And that's all we had to start with. But again, it allowed us to say we were seeing some changes. It can now be replicated in controlled clinical studies in people who are not world-class athletes, which is the reason I developed the program in the first place.
Taubes: Okay, so the Zone is 40% carbohydrates. I think of it as a low-glycemic-index diet, in part because David Ludwig credits you with spurring his thinking in the field, and David thinks of it as a low-glycemic index. But in your book, you don’t actually talk about it that much: the low-glycemic index diet. Although, I assume at the end when you give the list of good foods and — or good carbohydrate portions and bad, the high GI carbs are gonna be — Well, bagels I think were moderate —

Sears: No, they’re bad. So, when I said — I use these big words, like “favorable carbohydrates” or “unfavorable carbohydrates.” Well, all the favorable ones were low-glycemic carbohydrates. You know, vegetables and a little fruit. All the unfavorable ones were the high-glycemic grains and starches. Now, six years after I wrote my first book, *The Zone*, in 1995, another book came out. It’s called *The South Beach Diet*. Basically, for three weeks it was Atkins, and for the rest of life, it’s the Zone diet. So he used, now, words which are more consumer-friendly: good carbohydrates, bad carbohydrates. Silly me. I was using these big words. But even with the favorable carbohydrates, a low-glycemic-load diet, you still had to balance the protein to that low-glycemic carbohydrate to get the most appropriate hormonal response. And so, it wasn’t just that any one particular one was the boogeyman. I say, “No, there’s a dynamic balance.” And now that dynamic balance also required, what were the intake of omega-3 to omega-6 fatty acids? Because the enzymes that convert the dietary intakes of omega-3 or omega-6 fatty acids to the respective classes — the enzymes are under hormonal control. In particularly, the hormones that the enzymes that convert omega-6 fatty acids into arachidonic acid are stimulated by the hormone insulin. They — but they’re also inhibited by the hormone glucagon. So, by balancing those two hormones, you can now control the enzymatic activities that could basically now dictate which directions those eicosanoids flow into.

Well, we’ll start with — even going back in time. Let’s start with Bob Atkins and Dean Ornish. Bob Atkins said, “Carbohydrates kill.” Dean Ornish said, “No, fats kill.” Again, the old Miller Lite commercials, again. And yet, both were partially right. That basically Bob was correct: If you eat too many carbohydrates, you basically have too much insulin, but that was the state of thinking. And even Dean Ornish was partially right, if you have too many fats, especially saturated fats, especially palmitic acid. A lot of fat — saturated fats are not adverse, but palmitic acid is the bad boy. So, he was partially right.

Taubes: And palmitic acid does?

Sears: Palmitic acid is the fatty acid, the saturated fatty acid, that can basically enter the brain, go to the hypothalamus, and cause inflammation. And when you basically consume too many carbohydrates, the de novo lipogenesis makes palmitic
acid, again, which is a very powerful pro-inflammatory saturated fat. So, I was saying, everyone was partially right. Now, when — my first introduction to Mike was when he was writing the book on *Thin So Fast*, he was writing an article a — one chapter on eicosanoids. And he didn't know much about that, so he called me up, and I gave him some insights. And that began our relationship, and I consider Mike one of the smartest guys I've ever met across in nutrition, and probably one of the funniest guys. So, he's a great sense of humor. He's just a — but he's very, basically, very fastidious. And so, Mike and I had many things in common, but again, the difference, see — that balance of protein to carbohydrates, or really, the protein to low-glycemic carbohydrates. So, we shared many things in common. We had some certain differences. And so, again, that's the beauty of nutrition: There's no one way, no one route. But the top of the mountain remains the same. How you — how you get to the top, it doesn't matter. Now, there are many pathways to it. Some might be harder, or others. But see, that's why we had to have — when we talk about the Zone, we had to have defined chemical and clinical endpoints. How do I know if I'm in the Zone? It's like, I feel good. It's like, that doesn't count.

**Taubes:** So, Atkins said that about ketosis: “It feels like sex.”

**Sears:** So did — you know, basically Dean Ornish said, “I feel great.” I said, “Guys, show me the blood.” Because that's your marker. You're looking at basically — I need validation to say what will be the best diet for an individual. We're all different genetically.

**Taubes:** Okay, what are we looking at in the blood? What are you looking at?

**Sears:** I look for three things, because there's three components of inflammation. We said one is turning on, another is turning off, and the third is repairing the damage caused by the prior inflammation. Three separate entities. So, in turning on inflammation, I look at basically the ratio of triglycerides to HDL cholesterol. It's a surrogate marker for insulin resistance. Whatever causes insulin resistance, we might have contra — but it's a surrogate marker that basically correlates very well with, now, you know, insulin clamps. So, if you have a high triglyceride-to-HDL level, you probably have insulin resistance. So, you adjust your diet by adjusting the ratio of protein, carbohydrate, fat, and calories until you get the ratio of triglycerides to HDL under one. This hard work. World-class athletes were always about 0.5.

**Taubes:** Let me ask you a ques— I'm gonna skip ahead in our interview to discuss the studies you'd published in 2006, 2007, that you'd sent me on ketogenic versus non-ketogenic low-carb diets. Which — Zone-favorable diets. I noticed in both of them, HDL went down, which was surprising, and I just, since we're on the subject of HD — surprising to me. Why did HDL go down in those trials?
Sears: Well, because they're calorie-restricted trials. Remember, the Zone diet is a calorie-restricted diet, and that is the one way, the one proven way to live longer.

Taubes: Is it always — it's always the calories?

Sears: It has always been from day one. Never — never has changed one —

Taubes: Okay, see, this is again one of the places where you and I differ on our perspective. To me, what calorie restriction — when you speak to the researchers who study calorie restrictions, I've done — the one fundamental thing it does is inhibit the insulin IGF-1 access. And so, it could be mimicking carbohydrate restriction, which does the same thing.

Sears: It could. But again, when you have calorie restriction, you are restricting your — basically the Zone diet is based upon not how many calories you need, it's based upon how much protein you need. The amount of protein you need is unique to you. It depends on your lean body mass, not your weight, and depends on your level of activity. That's unique. Now once you have that, now you spread that protein to maintain your lean body mass throughout the day in even amounts like an IV drip of a drug. Now, to that protein you try to balance that with low-glycemic carbohydrates at each meal to basically maintain enough insulin, but not too much, so you only have enough glucose to maintain constant optimal, brain function.

Taubes: Now, you will get insulin from the protein.

Sears: Of course. Of course, and that's why the glucagon becomes so important, because the protein stimulates the glucose. It's the balance of the insulin and glucagon, and that will be unique to you. So, you keep adjusting your diet until, how do you know you've got the right balance for you? You're not hungry for the next five hours. Now, why are you not hungry? Because you're stabilizing blood sugar levels, and so once you find that right ratio to you — it's not, it's not a wide ratio, but again, it's a unique ratio. Say, “That's the ratio that my body will work at the best.” Now, where will the rest of the calories come from?

Taubes: Let me interrupt for one second. Describe stable blood glucose levels.

Sears: Well, stable blood glucose levels are a consequence of the hormonal interactions with the glucagon, which —

Taubes: I know where. I'm just curious how big of swings are you allowing in stability.
Sears: I'd like to have me no more than about 20 milligrams per deciliter. So, I like to keep it basically, if you're at let's say of no higher than 110, definitely no lower than 70. So, if we can keep it around 90, that's ideal. I'd say, “Now I can treat my food like a drug, but I have to adjust it for my biochemistry.” Now what about the fat? You've got plenty of fat. They can't get out of the fat cells unless you lower insulin. Now, can you can lower it — you can lower it very low, but say the amount you need to lower it isn't that great. So, how do you know if you have the right amount of body fat? You stand stark naked in front of a mirror. Now, if basically you have a six-pack, you probably need a little more body fat. See world-class athletes work best — I'll use swimmers as my example — they work best at about 10% body fat. Now, they don’t have a six-pack, they have a two-pack because they need the extra fat to generate the ATP to repair the damaged tissue.

Taubes: They need to be able to mobilize the extra fat, and the way they melt fat, they can mobilize.

Sears: And that — the way they mobilize that is to basically lower the levels of insulin, but the amount that you had to lower it to, you don't want to take it out of the system completely. Because there's a problem there. If you lower insulin too low, then basically blood-sugar levels will also be lowered, and therefore you'll get a counter response. And they would show this — David Ludwig, you know, on a long-term aspect — you start to see cortisol levels begin to increase. So, once you — you know, the hormones don't operate in a vacuum. You start changing one or two hormones, you'll be changing other ones. So, you also want to basically lower the levels of endogenous cortisol, because it is a stress hormone. So, you're finding that sweet spot, that hormonal sweet spot that says, “I'm not hungry. I'm not fatigued,” with the least number of calories. And now, why — why is it the least number of calories? Because this will activate the gene transcription factor, AMP kinase. This is the gene transcription factor that turns on and controls your metabolism, and so any way you can activate AMP kinase — this becomes critically important for an athlete, because it's AMP kinase that goes in and takes out the damaged mitochondria — it's called mitophagy — and replaces it with a brand new mitochondria that's more efficient.

Taubes: Okay, now I'm going to assume that AMP kinase is activated as insulin decreases and —

Sears: No, it's actually only activated by two things: a decrease in — increase in A and D — A and D, which is the cofactor to make ATP, or a decrease in ATP levels. Either one will activate AMP kinase, and so basically it's an energy sensor. It has no relation to insulin per se, but it is an energy sensor. So, the secret of looking at calorie restriction, it — basically you're lowering ATP levels, and you're turning on AMP kinase. There are drugs that can do it. One drug is called metformin. The
primary drug to basically treat diabetes. Drug of first choice. But metformin does it by a kind of a half-ass way. It poisons the mitochondrial. Not a very good idea. And so, I say, “Is there any way we can turn on AMP kinase besides poisoning the mitochondria?” Well, yes you can. One is to basically restrict calories, but it has to be without hunger or fatigue.

**Taubes:** Okay, so how does your hepatocytes know whether you're restricting calories or just carbohydrates? I mean, one of the questions we’re ultimately going to talk to is, we talk often a lot about excess calories, and I don't actually know what that means. Okay, I mean in a sense, you've described it. Excess calories is anything over the minimum that you can eat and not be hungry for five hours. But for many of us, if we have carbohydrates in our diet, while we’re eating, we don't know what that means.

**Sears:** No but we can see the consequences. Again, we look stark naked in the mirror, say —

**Taubes:** Oh, but again, that's a different hypothesis. So, this is where we differ on the perspective, because to me, the consequences are a result of the adipocytes wanting to accumulate fat. Trapping fat in the fat cells with the result of the in fact excess insulin would be the primary moderator, and if you trap fat in the fat cells, you’re going to have a whole slew of downstream — . It's like semis — internal starvation was how it was described in 1961.

**Sears:** It is, and that's a very good description.

**Taubes:** So, we have — we have — you have a, correct me if I'm wrong — a kind of a hypothalamic-centric view of this universe, and I have an adipocyte-centric view of this universe.

**Sears:** Well, and they're all part of, let's say the — let's throw the liver in there too, because we can't fault that. So, we had to look at all of these interacting: the hypothalamus, basically — and we know this much for high-fat studies in rats, for what they're worth — that the first organ that's affected by a high-fat diet is not the fat cells, nor the liver. It's the hypothalamus within 24 hours.

**Taubes:** The first is always a chicken and egg thing. I'm never so sure that you can define a first. Might be the first that you can notice, but we could go back to the hypothalamus and the BMH lesion. So, bench medial hypothalamic lesions, which the first animal model of obesity — and in those experiments, first done by Hetherington and Ranson in the late 30s. So, Ranson was the leading neuroanatomist of the era. Hetherington was a graduate student. And Hetherington perfected the stereotactic technique to make sure you were lansing
just the right part of the rats, dog, brain. They started with dogs, which were easier because the brains were bigger. Then they went to rats. What they noticed was that they would get obesity in their rats with or without excess eating. So, some of the rats would eat more, they would become hypophagic, they would get obese. Some of the rats would not eat more, and they would become — they would be sedentary and get obese. And rather than assume that the hypothalamus was some kind of hunger, satiety, energy expenditure, organ moderator, the assumption was that what — when you lesion the ventromedial hypothalamus what you're doing is somehow directly influencing fat accumulation. Unfortunately, Ranson died of a heart attack, Hetherington joined the Air Force in the middle of the war, and there was a third graduate student in the lab, Brobeck, who went to Yale, started doing experiments at Yale. When he saw rats that got fat without increasing their intake, he decided that they had done something wrong and didn't count those and promoted this theory that the hypothalamus controlled hunger. And it was, since Brobeck was the only one who survived the war, and he was at Yale, which is a nice prestigious research laboratory, that theory was taken up and fit into this idea that fundamentally obesity is a disorder of overeating or excess-caloric intake. But the 60s, once the radium, you know, assay was available so you can measure hormone levels, it was clear that the first thing that was measurable when the ventromedial hypothalamus was lesioned was hyperinsulinemia. Even cephalic phase hyperinsulinemia, so the animals would hyper-secrete insulin prior to eating, just upon the getting close to eating time. So, now we have a phenomenon that basically says the VMH, among many of the hormones that it regulates — one of them is insulin, and if you lesion the hypothalamus, you're just going to over-secrete insulin. So, it's not about inflammation. It's about just a purely hormonal response.

Sears: Well, again, and you say, yeah — you're quite right. Well, you know, putting a little, basically lesions into a rat brain is hard work, because you might make a miss. That's why we have now more, more sophisticated ways to say we can now look at the markers of inflammation: cytokines. And basically, we can see when we start a, let's say, a mouse on a high-fat diet, that the first increase, the first organ in the body, or the first region of the organ that basically is now seeing an increase in cytokines is now basically the hypothalamus. You begin to see this in the liver cells —

Taubes: Now where are the cytokines coming from in this model?

Sears: Again from the inflammation.

Taubes: And where is the inflammation coming from?
Sears: Comes from the excess consumption or calories. This is, you know, David Kay’s work of many years ago.

Taubes: Okay, so now define excess consumption of calories.

Sears: Well again, more calories than the body needs.

Taubes: But, saying more calories than the body needs assumes that the body has some baseline energy expenditure that doesn’t change. So, energy expenditure meant metabolic rate can go up and down to absorb caloric differences in caloric loads. That’s going to in turn be regulated by — affected but what the fat tissue is doing. If the fat tissue is taking up fat because insulin levels are high, the rest of the body is going to see few calories. This is the internal starvation notion. So, when I hear that term, “excess calories,” I’m just not sure — there’s a lot of assumptions about how much your body needs, how much my body needs —

Sears: That's why we're — That's why we're all genetically different. And that's why one size does not fit all. And that's why you have to basically look to say that we have genetic diversity, and that we had to find the right appropriate dietary mixture in terms of calories, in terms of ratio of protein, carbohydrate, and fat, that basically is gonna be ideal for that individual person. So, what might work for one person might not work for another. And so, you're looking to basically now have markers, like buoys. And that's why the blood markers. So, one thing blood markers that tells you on a longer-term basis is how are you controlling the ratio of triglycerides to HDL cholesterol. That's the marker of insulin. What causes it? Let's not worry about that, but we have an end result. So, you say one thing: I want to adjust my diet so I can keep that ratio always under one.

Taubes: Let me give you an example of where the excess calorie concept comes in. So, the two papers you sent me, or you sent Karen Thompson and made in preparation — so the, two — there were two studies: 2006, 2007. They seem to be the same patient — basically the same feeding study in the same patient population. One was comparing the non-ketogenic low-carb diet, the Zone-favorable diet, the ketogenic —

Sears: Well, that was the Zone diet.

Taubes: Yeah, okay. In your book, you used the phrase, “Zone-favorable” back then.

Sears: All those people were like, “favorable carbohydrates” —
Taubes: And favorable's good. It's a good choice because you're being precise at the time. It's just, you're saying we got to hit the Zone, you're having really — the book isn't out marketing the Zone as a diet yet, so you keep referring to the phrase.

Sears: Oh, let's go back to the book. The fact that people even bought the book was always so surprising to me, and even more surprising to my publisher.

Taubes: Yeah, I can imagine. Okay, I mean it's — Well, what did you sell? 2 million copies?

Sears: That's about 1,995,000, I thought, more than what was going to be sold.

Taubes: Well, I often talk about how my first five books are all about good science and bad science, and the first two were physics and cold fusion, and I did not make a living writing those. And the last three were nutrition, and the very same subject, much higher readership. Getting back to these trials. So, you calorie-restricted both groups. So you estimated energy balance, and you calorie-restrict both groups. What do you think would have happened had you allowed both groups to eat ad libitum.

Sears: We did. Now, we did that. In the first study, what we did — there's four parameters you have to control.

Taubes: Well, the first six weeks was a feeding study.

Sears: That's why you can only control any two at any one time. So, when the parameter is now calories, another is total protein, another's total fat, and total carbohydrates. So, you can control two, clamp two, and basically see how the others change.

Taubes: Well, you could consider eating to satiety —

Sears: No but, that's why that we treat the human beings like lab rats here. I say, satiety is a very, I'll say, subjective aspect. So, we say, “We will basically clamp you for the first six weeks. We're going to treat you like lab rats. We're gonna prepare all the foods for you. We don't want you to think, because you go screw things up if you think.” So, in the first six weeks, we can obviously clamp their diets and of, saying, what will be some of the change? Was there a metabolic advantage, as Bob Atkins would talk about? And we found no metabolic advantage. But we did see a significant increase in a marker of inflammation, which is again the marker of the resolution phase. This is the ratio of arachidonic acid to EPA — significant increase. So, that's why the — our title of the paper was a non-ketogenic diet or a ketogenic diet has no metabolic advantage versus a —
Taubes: Okay, and the argument would be that by calorie-reducing both groups by 30% you would see the metabolic advantage if you had both groups eating ad lib.

Sears: And and then, that then for the next four weeks we want them ad lib.

Taubes: Well, by the next four weeks, you've already got study subjects who are losing weight and want to continue losing weight, so they're gonna calorie-restrict naturally. One of the questions that would be interesting would be to have fed them both 100% of energy expenditure rather than 70. Do you think there would be a difference?

Sears: Ah, yes. Ah, well, I don't know. That's —

Taubes: Okay which — this is what we do in science. I mean, I'm a journalist, but we predict. We say with this — we're going to think of a thought experiment where we could test the competing hypotheses. So, in this thought experiment you've already measured, you've estimated their energy expenditure. Now we're gonna add something at 100% of the ketogenic diet versus —

Sears: But now you're talking about calories. Remember that the Zone diet is based upon how much protein you need, not how many calories.

Taubes: So, if they eat a 100% of energy expended, can they not do it on a Zone diet? Or are they now going to — so, I don't understand why you couldn't have fed them the non-keto — well, the Zone diet at 100 — or it's — okay, I get it, by definition it's not a Zone diet if you're eating a 100% of what you want to eat.

Sears: Because again, it's all predicated on how much protein. So, the one thing we clamp, the amount of protein they consumed was identical. The amount of calories they consumed was identical. So the only variable was the ratio of carbohydrate to fat in the two diets for the first six weeks. And so we say, “Okay, we know we have — they have adequate protein.” Nobody should eat more protein than they require. They should not eat any less protein than they require. So, I say, “You're looking to say: protein is our first step, we had to stabilized — give the levels of protein to maintain lean body mass.”

Taubes: Well, what I mean is, you're assuming you have to under-feed to get the weight loss.

Sears: No.

Taubes: Then why did you under-feed?
Sears: We don't under-feed.

Taubes: You under-fed by 30%. 70% of —

Sears: No, I said the under-feeding is saying — first of all it's based upon not how many calories. This is why the Zone diet is different. It's how much protein you require.

Taubes: Okay, so we could have done the same thing. Because I often think of — I think 30% is high-calorie diet — high-protein diet by my understanding of the ketogenic diet or even Atkins. They're high-fat diets. They're not high-protein diets. And today, maybe not in 1994, but today, or 2006 — today when you talk to people about ketogenic diets, like Phinney and Volek, they're talking about 80% fat, 5% carbs, 15% protein. In fact, we often talk about it as a mistake that people think of these high-protein diets, and I think —

Sears: They aren't.

Taubes: Well 30% —

Sears: No. But, but, but again, it's the absolute number of grams of protein.

Taubes: Okay, so then we can take our 70% of energy, estimated energy expenditure, and feed them 100%, but make it a 20% protein diet, so we're gonna have roughly the same amount of protein spread over — but now it's not a Zone diet, so we can't do it. So, you can't actually do the study. You can't imagine the study where they eat 100% of the energy they require, because there's no way you can see that being a Zone diet.

Sears: And that's why we — that's all I that — we didn't do that experiment. We did the experiment we could do, and that is saying, “We will basically clamp the calories and clamp the protein.”

Taubes: Let's just — That's an interesting problem we have here, because now we can't test what happens ad libitum, and we can't test at 100%. One of the mistakes that have always been made when studying these diets is you feed them, you tell people to eat as much as they want, and when they lose weight, the assumption is that they must have eaten less, and that's an assumption. There are many ways if you mobilize fat and oxidize, you can get an increase in energy expenditure. In fact, you know there's studies that might not-for-profit funded. One of them arguably did or did not see an increase in energy expenditure on a ketogenic-isocaloric diet, the other one hasn't been published yet. But it's your friend and mine David
Ludwig's, and it'll be interesting to see what he saw. The — Let's imagine you compare a 100% ketogenic diet to a 70%-of-energy Zone diet. It's a different way to do it because you might still say I wouldn't — I'd be curious what would happen. I don't think I could predict — I think you'd be pretty confident that the Zone diet would do better.

Sears: I would.

Taubes: But it would be an interesting experiment to do because the — in other clinical trials in which they compared, like the Gardner trial, which you cited in your, in your — one of your articles, the 2007 Gardner trial, but typically they compare a calorie-restricted low-fat diet to a ad libitum low-carb high-fat diet. And I've always pointed out, they often see the same thing, but then the question is if you only have to calorie-restrict one of the two diets, it suggests that the calories are not modulating the effect, that there's something else —

Sears: And that's why there are very few studies published — I think because they are hard to, to control the diet. And there aren't that many, but the ones are, they have published, have been pretty conclusive. One is published, 2016 by Starzl. This was looking at people who were — had metabolic syndrome, and they basically, again, clamped the study in terms of, now, amount of calories. They were again, calorie-restricted diets. All the foods were prepared for them, and one was a higher carbohydrate diet, and one was a Zone diet. So, if saying, “Okay, a higher carbohydrate diet — both had about the same amount of protein — .” But what they found in this study here, the six-month study, that in 100% of the patients who had metabolic syndrome, 100% were basically no longer pre-diabetic at the end of six months. On the same diet, which was calorie restricted, only 33% had basically been now decreased their metabolic syndrome. So, it's saying that these types of studies, we have to basically, when dealing with humans, we have to treat them like lab rats.

Taubes: And no — I completely agree. And I co-founded a not-for-profit with precisely that thinking in mind. The um — Let's just step back one second to the to the two papers published. Again, they were the same — clearly the same patient population. One was on ketogenic diets and exercise, and one was on the long-term weight and inflammatory markers, heart disease markers. Regarding exercise, where you reported that people on the ketogenic diet had less energy to exercise and got tired quicker, and now I'm just channeling Steve Phinney and Jeff Volek, because I know what they're gonna say is, they're gonna say, “It's gonna take three weeks to keto adapt, and you reported the two-week data, but we know these were — this was a six-week trial from the other paper.” So, why did you only report the two-week data, rather than give them time to keto adapt and see what happens
at four weeks and six weeks, or even at 10 weeks, because we have four weeks of free-living trial at the end?

**Sears:** Because that was the protocol we did. Actually, there are two separate groups, so they weren’t the same groups. They —

**Taubes:** Well, they looked very similar. You had one dropout due to heart arrhythmia in both groups. In both cases, the one dropout was in the ketogenic diet group. There — you’re right that the numbers were slightly different in terms of weight, but the description very much sounded like exactly the same group.

**Sears:** Well, your pulling from a subset of a population of Arizona, which is, you know, pretty much similar. So, again —

**Taubes:** No, I mean that the 10 versus nine, the dropout from the heart arrhythmia — Same authors —

**Sears:** Always, yes — but with the — same study was saying, “There was a correlation between, basically, fatigue, which is a perception — subjective marker, and the levels of ketone bodies in the blood.” That's all the study said.

**Taubes:** Okay, but what I'm saying is that was at two weeks.

**Sears:** But again —

**Taubes:** So, you’re saying that study was only done at — so it’s a different group of people —

**Sears:** That's all we can talk about, say, “At two weeks, there seemed to be a — there was a statistical correlation” — correlation and causality are two different things, but — “a correlation between the levels of ketone bodies in the blood and perceived exercise exertion.”

**Taubes:** And that study, those people were not on the diet for more than two weeks despite the very considerable appearance of them being the same people who were on the six-week diet?

**Sears:** But again, saying that I take the aspect that you keto-adapt very quickly.

**Taubes:** Okay.

**Sears:** So, I think that usually within a week's time, you have probably pretty well keto-adapted.
Taubes: Yeah, well again, I rely on Phinney and Volek to be my experts on keto adaptation, and they’ll tell you three weeks. And actually your buddy, David Ludwig has been writing papers arguing that it's three weeks, so if you do trials over one week, you miss the adaptation. And if, you know, again — it's, you may be right —

Sears: Who knows? And that's the beauty of nutrition. It's complex, but it's saying that you know, these are all of — remember going back to our key thing about controlling inflammation, you're asking about the markers. Now, one marker's the ratio of triglycerides to HDL, because again this is why you want to basically treat insulin — that's a classical marker. But this only tells you about the diet. It doesn't tell you anything about the diet, how it might affect inflammation. The key factor is the resolution of inflammation, and that marker is the ratio of arachidonic acid to EPA. These are the two building blocks of the hormones that cause inflammation or cause resolution.

Taubes: We have a whole host of markers for inflammation now.

Sears: They're not very good.

Taubes: So, how can you demonstrate that the ratio is better?

Sears: Because you've all — Because one thing, you have to have a ratio that basically you can measure continually. The markers of inflammation — I can look at cytokines, but they're very variable. We talked about earlier, I can measure of eicosanoids, or even resolvents. They're gone within seconds. So I need a marker, a long-term marker that basically has clinical correlations. The ratio or arachidonic acid to EPA has been shown to be strongly correlated with basically increasing lifespan and decreasing cardiovascular disease and a wide number of other diseases.

Taubes: Okay, but they — if I'm remembering correctly, and this is outside my area of expertise, but the arachidonic acid content that the blood, again is going to be triggered, regulated by what insulin is doing, if I remember correctly —

Sears: Exactly, yeah —

Taubes: And so, we're still back to the same fundamental — so when insulin —

Sears: Insulin and — insulin and omega-6 fatty acids. You need two to tango. So, the typical omega-6 fatty acid, which is one oleic acid, which is in great abundance in the American diet, if you had low levels of insulin, you might not make much of arachidonic acid. That'd be good. But if you have even low levels of arachidonic —
of oleic acid, but high levels insulin, you're gonna make more arachidonic acid. But the EPA again, because it's the EPA that is the marker, the marker resolvents. It's the resolution of inflammation. These are things — we talk about how diet and exercise can turn on but how basically we can turn it off, and here we have much less controversy. Much less controversy, because we know the mechanism of turning off inflammation: It's the increase of the hormones that cause the resolution process. And these are all derived only from EPA and DHA, two long-chain omega-3 fatty acids. So, regardless of what diet one is following, and it could be uh — and that's why I say there's many roads the top of the mountain, saying, “How can I reduce the intensity of the inflammation by my diet?” There might be a lot of controversy, and that's, that's good, but in terms of turning off the inflammation, there's very very little controversy. Because this is basically all mediated by one group of hormones, these resolvents that come only from the omega-3 fatty acids.

**Taubes:** And some of the recent papers you've sent me were arguing you're working now in higher dose. You believe a lot of the lack of effect you see in clinical trials with omega-3 supplements is because the doses are too small in effect.

**Sears:** That's correct.

**Taubes:** And an argument I've seen for vitamin C as well, and often convinces me to take mega doses of vitamin C when I feel a cold coming on — I have no idea if it's a placebo effect. The — tell me what you're doing with the omega-3s now and the high doses and what you're seeing.

**Sears:** Well again, we titrate the goal. This is the aspect of saying, “How much omega-3 fatty acids do you need?” It depends — it depends how much inflammation you have and where it's located. Let's say, then, we have a good body data starting from 1989 in the *New England Journal of Medicine*, taking Harvard Medical students — supposedly healthy — but they can give them high levels of omega-3 — that's about five grams a day — and within 10 weeks, they could see dramatic drops in the levels of cytokines in the blood. Especially tumor necrosis factor. The drugs like Remicade, Humera, Enbrel, 25 billion dollars a year in sales, for what? They say, “We want to inject an antibody to reduce the levels of tumor necrosis factor.” But as shown in the *New England Journal of Medicine* 30 years ago, high doses of fish oil, about five grams per day, could achieve the same reductions of those — of those tumor necrosis factor without any side effects. We also know that the ratio of arachidonic acid to EPA — Much is done by the Japanese. They've done that much, much of the work — is strongly correlated with cardiovascular disease. We know from a data recently published about two years ago, looking at the Women’s Memory Initiative, that those who had a lower level of arachidonic acid to EPA had a significant increase in lifespan. So, we have now a long-term
marker, a stable long-term marker. C-reactive protein is not a very good marker because very small amounts of bacterial infection might raise it very quickly, but the ratio of arachidonic acid to EPA is a very good long-term stable marker as an indicator of the balance of eicosanoids and resolvents in the blood and also in the tissues. We knew more recent studies, this was published last year, of giving now four grams a day of omega-3 fatty acids. You be you can begin to see these resolvents in the blood. And if you don't have enough omega-3 fatty acids, you simply can't make the resolvents. And if you can't make the resolvents, you can't turn off inflammation, and this becomes important not only for the cardiovascular patient, but also for the athlete that says, “I'm basically, I'm inducing inflammation, and the more intense — more intense we exercise, the more inflammation I'm inducing. Now, I want to basically get back in action as quickly as possible. I have to resolve the inflammation.” The only way we can do that — and that's why I say this is very little controversy here, is we have to have adequate levels of omega-3 fatty acids. And you can measure them — they're very difficult to measure at the end products, the resolvents, but you can measure their pools. They're like a reservoir in the blood. Do I have enough to make the resolvents that basically allows me to turn off inflammation?

**Taubes:** And which cells are making the resolvents?

**Sears:** Every cell in the body.

**Taubes:** Okay, and how are the omega-3s getting into the cells to make the resolvents.

**Sears:** They're getting into the cells as any fatty acid would: through lipoproteins. The lipoproteins, especially LDL, is the transport agent to bring omega-3 fatty acids, omega-6 fatty acids, monounsaturated fatty acid into every cell. Now, they can be utilized as an energy source, so like any fatty acid, but that's why I say, “Do I have the right balance?” The average American — the ratio of arachidonic acid EPA is about 20 to one. If we go back to our estimates, what the ratio was in Paleolithic times, it was about one to one. And so, basically that dramatic increase in the levels of omega-3 — of omega-6 fatty acids — is that enough? And that's why one of the key factors says we try to say, “What basically has caused these problems? Ergo, why do we have so many inflammatory-related problems today?” It's saying, “Because we're basically inducing more inflammation in our bodies, and we've taken out, we've taken out dramatically the one entity, the one nutrient that can reduce inflammation: the omega-3 fatty acid.”

**Taubes:** Okay, so where are you gonna find the omega-3s, other than supplements?
Sears: Oh, fish. In fact, you know, the — the people who have the lowest ratio in the world today are the Japanese. They're the largest fish consumers in the world. Now, a recent article was published — that how many populations — if we take an arbitrary cutoff, 8% of the fatty acids, consuming — consisting of EPA and DHA? Only about, you know, only in very limited locations in the world, primarily Japan, beat that level. And so, as estimated that if all the fish were consumed today and made into fish oil, there'd be enough fish in the world to basically meet the needs of six percent of the world's population.

Taubes: All right, so what happens when the Japanese immigrate to the U.S.? What happens to their ratio?

Sears: It goes up.

Taubes: Okay. And do we know that their fish consumption goes significantly down?

Sears: Well, we — all we know is the ratio goes up.

Taubes: Okay, so here's one of the things I noticed reading your book, is there's virtually no mention of sugar, and it's an interesting issue. Are the — I argue this with David Ludwig. Is sugar just a high-glycemic-index carbohydrate? Are sugary beverages just a very high-glycemic-index carbohydrate? But again, in The Zone you don't — you really don't talk about it. And it's funny — my experience, I remember back in the 90s, sitting behind a fellow at a restaurant in L.A., a takeout restaurant that had healthy food, I can't remember the name, but I remember exactly where it was on Main Street in Santa Monica — Main and Rose. And he was very much on your diet, and he was trying — asking whether or not the sauces that they were putting on the skinless chickens had sugar in it. He may have misunderstood the book, but I'm wondering where it fits in, and one of the reasons: If you've read my book you know I argue sugar is a potential cause of insulin resistance, is in part because the fructose is metabolized in the liver and converted to fat in high doses. And the people like the Japanese are traditionally very low sugar consumers, and when they come here they may or may not eat the same amount of fish. It's easy to imagine that they increase their sugar intake. But none of this, at least in The Zone, the 1995 —

Sears: Or the 2018 version.

Taubes: Yeah, so where does sugar fit into all this, and how would it change your thinking if you had reasonable evidence that sugar was triggering insulin resistance?
23 Years in the Zone: Journalist and Author Gary Taubes Interviews Dr. Barry Sears

Sears: Well, the dose is always in — the poison is always in the dose. You know, that's why you — when I talk about if insulin-variable carbohydrate. Sugar was not on the list of the good boys. It was a bad boys. Because, but again, fructose has been demonized. If you basically are following a moderate-carbohydrate diet that is very very rich in non-starchy vegetables, the levels of fructose, even though fructose is a major component, 30% of the sugars in vegetables are fructose — 50% is from table sugar —

Taubes: When you're defining sugar, in that case, you mean the digestible carbohydrates?

Sears: Yes.

Taubes: So, is that 30%? I would be surprised if it's that high.

Sears: Because, first of all, it's very hard to over-consume carbohydrates.

Taubes: Well, it can't be 30%, because then it would be the equivalent of 60% sucrose and green vegetables are not 60% sucrose.

Sears: But they're also very rich in polymers of fructose. Inulin is a basic — of you know —

Taubes: Inulin? Inulin has just been labeled by the FDA to be a fiber, oddly enough.

Sears: That's fine, but not — no — actually a fermentable fiber. Not a fiber. The FDA hasn't quite got that far. Because they have to distinguish between fiber and fermentable fiber. Fermentable fiber is the only source our gut microbes have to basically nourish themselves, and if they don't get enough fermentable fiber, they get mad, and then they get angry because they're hungry. They start digesting the mucus barrier.

Taubes: You've mentioned of — what happens in populations that effectively didn't consume fiber? We have these, and what were, appeared to be very healthy populations. We don't know how long they live, but the Maasai warrior class, the Inuits, the Native Americans of the Great Plains that were not fiber eaters, right? This is a question — I was just at a meeting in the, in Zurich, a British Medical Journal meeting, and I was asking this question to the leading English fiber gut biome: If you remove — do they need the fiber to feed on, or is it simply that the fiber-rich foods, low-glycemic index carbs usually replace high-glycemic index carbs and sugars, which would otherwise field bad microbes? He couldn't answer the question. But I'm always curious — the world is now — there are even individuals who consider themselves zero carbers, and at least the ones who are still alive
appear very healthy. Or the ones who are still alive and capable of blogging, clearly they don't need fiber maybe their gut lining is slowly being eaten away. I don't know —

Sears: I'd say probably the latter. Probably slowly eaten away, but that's why you need controlled studies. But we do know one thing: The gut is really the last unexplored organ in the body. We know nothing about the complexity of the gut. It has a 100 times more DNA than our human cells. It can make and do things that we can't do, and but it's been this kind of a black hole, almost a black hole biology, because there's very little ways we can see what's actually taking place in the gut. But the one thing where it's becoming more clear now, that with high levels of fermentable fiber that basically would not enter the bloodstream but become critical for a gut health, that basically gut health and *Escherichia coli* are basically reducing, now, basically, insulin resistance, reducing diabetes and obesity.

Taubes: Yeah, I haven't seen that data yet. You might be —

Sears: It's coming out — it's coming out in my next book, *The Resolution Zone*.

Taubes: Yeah, I remember one of the — I got an opportunity to interview Sir Richard Doll, a great British epidemiologist who was knighted for his work on cigarettes and lung cancer, and the thing we talked about — this is about a year before he passed away, so it would have been around 2003 — and I asked him about the fiber story, and he said, circa 2003, the only thing fiber seems that we've demonstrated that fiber is good for seems to be constipation. And all the other hypotheses had fallen by the wayside —

Sears: Fortunately, we've gone a long distance from 2003 to 2018. We now know basically that there is, uh, things like insoluble fiber that's good for constipation, you know? But if it's not fermented by the microbes, not fermented to make the short-chain fatty acids that control the immunological composition of the gut, we're gonna be in trouble. So, again as we understand more the complexity of the gut, you stand back in amazement saying, "We've basically missed a very large opportunity." One thing we can look at that's been known for many years by serendipity: Farmers have learned very quickly that if they gave animals very low levels of antibiotics, it would fatten the cattle up very quickly. If high level — because what happens, you're basically causing now dysbiosis in the gut, causing quote what is commonly called a "leaky gut," now small microbial fragments can enter the bloodstream and interact with toll-like receptors found in every cell to basically set off an inflammatory response. Now, if you have a lot of microbes entering your bloodstream, you get sepsis and you die. But very small amounts, maybe one-hundredth the amount of basically the lipopolysaccharides, the LPS, are
enough to basically, significantly increase systemic inflammation to be a strong driver of basically, now, obesity and diabetes and other metabolic diseases.

**Taubes:** So, you're saying this has got — dysbiosis is driving this. Caused by antibiotics or caused by the foods, do you think?

**Sears:** Caused by both. Caused by both.

**Taubes:** Okay, and let's get back to the role of sugar, for instance. Because again, you didn't mention in your book — Wasn't in the mid-1990s, it was out of fashion as a cause of chronic disease? It had been in fashion, and it had fallen out of fashion? You talked about, even one of the things you talk about in your article is a high-fat diet and animals being used to trigger insulin resistance, and those high-fat diets — the original awareness, Gerry Reaven's lab at Stanford, was that high-sugar diets were the easiest way to cause insulin resistance in animals. And I would bet you that in virtually every high-fat diet we find that causes insulin resistance that's reported, the researchers aren't bothering to report that they're 20% sucrose and that the fats are corn oil.

**Sears:** I don't, I don't disagree. I don't like the word “high.” I don't like “high fat” —

**Taubes:** I don't like “excess,” so we're even there, okay? I don't know how you define excess other than that you look in the mirror and you're fat, but —

**Sears:** Oh, you can measure it too —

**Taubes:** That to me is a tautology.

**Sears:** No, but that — but that's why again, looking at the end result is fat accumulation.

**Taubes:** Or well, the initial result could be fat accumulation.

**Sears:** Well again, but the final result is — and that's why I do, and that my book *Toxic Fat*, which I wrote in 2008, said we should view obesity as a form of cancer. We should see basically the spreading of fat — atopic fat, as a form of metastases.

**Taubes:** Well not only that, and if you've read my book you know that prior to World War II, Julius Bauer was a leading endocrinologist of the era, was arguing that fat accumulation should be viewed as similar to a tumor, and that it had an agenda all of its own, independent of that of the rest of the body. Slightly different, I mean similar thinking, slightly different implications. The — getting back to sugar. Where
does it fit in this, and where does it fit in the Zone, and how would it change your thinking if you knew that the fundamental cause of —

Sears: Doesn't — doesn't change at all. Because I say don't — don't eat sugar. Eat lots of vegetables. Who could argue with that? I say, “Eat lots of vegetables and a little fruit. Don't eat greens and starches.” And if I don't say, “Don't eat grains and starches,” then I'm — you have to say by assumption, “And don't eat sugar.” It's stupid.

Taubes: Yes and no. You never — actually when I first started my research and I — and it's funny. I mean, again, the message is very similar all the way along the line, which is: The things we're arguing today — so it used to be a healthy diet was don't eat fat, don't eat salt, and the assumption was you would eat a lot of green vegetables, but they weren't actually told to do that because then the government would sound like our mothers —

Sears: No, grandmothers.

Taubes: Pardon me?

Sears: Grandmothers.

Taubes: Grandmothers. Well, my mother. We're both old enough that our mothers told us that. Anyway, now you know part of the success of all this movement is the healthy diet starts with removing refined grains and sugars, and then some of us, and including some of the academics, will include starchy vegetables as something to avoid as opposed to green vegetables. But when I first started my research, so one of the first things I did back in 2002 when I was writing those infamous *New York Times* magazine articles, I spent three days with David Ludwig at his childhood obesity clinic at Boston Children's Hospital, and I'm stunned that the advice I had gotten from my mother, which is eat a lot of vegetables — you know, there's — every dinner was a green vegetable, a starch, protein, fat combination —

Sears: Wise woman.

Taubes: Yes. She was in many ways, but the smoking killed her prematurely, so it was wise woman with an addiction. Most people — many people were completely ignorant of what many cultures — there was just none of this. If it's in the market and particularly if it's in the market and advertised as healthy, and the iconic example would be a low-fat fruity sugar-rich yogurt that says “heart healthy,” and if the portions are small, it's not only heart healthy, but a diet food. So, I'm not sure that you don't have to give more advice — in fact, much of the public health motion, movement going on today is to label sugar. Label sugar.
Sears: It's the wrong thing. It's not so much the sugar, I think, in many ways. It's what they basically — you pulled out. You have pulled out, now, the fermentable fiber. You have pulled out the polyphenols, and why are these polyphenols important? Because they in large concentrations, just like the fatty acids, can now activate gene transcription factors — the one I talked about earlier: ANP kinase. And so, so when you basically now start taking out fermentable fiber, causing basically gut problems, now, you basically have taken out two of the primary factors which are key to our health: polyphenols and fermentable fiber. And so, many of our old wives tales — “You can’t leave the table until you eat all the vegetables” — we now know they have a very very firm scientific understanding, because of the role of polyphenols, and you know, fermentable fiber.

Taubes: There we, there we had a different generation, because we couldn't leave the table without finishing everything because people were starving in China —

Sears: In Bangledesh — And that's why that, you know, I made clear my, the start of The Zone Diet — first of all, I had to invent the Zone diet to basically get a better balance of eicosanoids. But my, how do I say, how did I come about the Zone diet? And actually, I believe it's chapter eight of my book back in 1995, I said, “It all came from Boyd Eaton.” He has a little small article in the New England Journal of Medicine making an estimate — I said, okay fine this is, you know, I said, “This makes sense.” So, I just took, basically what was the balance of protein to carbohydrate he had in that diet —

Taubes: Although, I interviewed Boyd for my book, and it was Boyd Eaton and who was his co-author? The um —

Sears: He's a surgeon, and I'm drawing a blank on his name too. Mill something —

Taubes: This is embarrassing. We should both be eating more —

Sears: We're both getting older!

Taubes: Yes that's true too, and we both probably do too much work. They made some assumptions about the fat content of those Paleolithic diets that were probably not —

Sears: And now we go fast-forward — we go fast-forward to 2010. Now —

Taubes: Eaton and Cordain.
Sears: Yes. Now this is a wide number of other very well-respected academics, now in the *British Journal of Nutrition*, making their best —

Taubes: Remember in my world, from my research, very respected academics in nutrition are not necessarily something —

Sears: Well, I could remember one of the authors was Loren Cordain, and the one that was Boyd Eaton. And two very smart guys, and the other guys I all knew individually. But they made their best estimate, their best estimate of what the Paleolithic diet was in East Africa 15,000 years ago. They came down to that it was about 40% low-glycemic carbohydrates, obviously had to be fruits and vegetables, 29% protein, and 31% fat. And, but they had one other thing, probably buried in there. That their best estimate of what Neo-Paleolithic man ate was between seven and 14 grams a day of omega-3 fatty acids.

Taubes: Do you think of the Zone as a Paleolithic diet?

Sears: I said basically on chapter eight, it comes from Boyd's work. I say the ratio of protein to carbohydrate, which has been fairly invariant from 1990 and 1985 to today, at least the academic estimations, is basically the Zone diet.

Taubes: So, then there is I think a fundamentally different way that we think about a lot of things. So, there's multiple fundamentally different things. One thing is, so even on the polyphenols story, which is an argument for fruits and vegetables, which are associated with good health in epidemiologic surveys, that association is not causal, as we'll both agree.

Sears: Agreed —

Taubes: It tells us nothing about whether fruits and veg— by the same token you'll see red meat has a slight negative association with health, saturated fats have a slight negative association with health, and these surveys — I worry in those surveys that what you're seeing is purely the result of socioeconomic status, or what one would call healthy user biases. So, in the 70s, you start telling people they should eat their fruits and vegetables and stay away from red meat because it causes colon cancer, and only — I mean who eats processed meats other than bacon? They're either on the Atkins diet, or keto, or they are too ignorant to know they're not supposed to eat bacon because it'll kill you immediately. So, you have a whole world of people who are healthy, health-conscious and they're eating exactly what we're told to eat. And then you have people who are too poor, or too ignorant, or just don't care to do what they're told to eat. And then you study them for 30 years and low and behold —
Sears: Again, yes, that's why I basically look at epidemiological studies and meta-analysis like sausage: Basically, it's an almost meaningless. It's good hypothesis-generating material, and yeah — So, but on the polyphenols, there were a couple of studies published in Italy, from an Italian population, the Chianti trial. They were asking the question. This — saying that all the old people who live in the upper hills of Tuscany who have long spent, say, perhaps because they eat lots of polyphenols —

Taubes: Or perhaps because when they were young, A: They starved through the Second World War, and long periods of famine might be terrific for your health, and B: They probably had excruciatingly little access to sugar because they're up in the hills.

Sears: Well, they still have a little access. But what's, what they did have was a very —

Taubes: Despite Coca-Cola's best efforts, they probably didn't have a Coke machine.

Sears: But the interesting part of the study, they said, "Perhaps we can now measure the levels of polyphenols by food diaries," for what they're worth. And say, "Is there any correlation between longevity?" And they were looking at people age 65 and, say, looking at the end point: death. Death's a very good end point — clear-cut, and no hand-waving. And they say, "Something's wrong here because there seems to be no correlation between the amount of polyphenols they're eating and their mortality."

Gary: Or something's right. So, as soon as they say, "Something's wrong," you've got a bias that makes you —

Sears: What they said — say, "Okay, let's look a little more carefully. Maybe it's not how much they put in their mouth, it's how much they get in the blood." It's very hard to measure polyphenols in the blood, like measuring eicosanoids, but you can measure their end products in the urine because the only way it can get in the urine: It has to be metabolized through the kidney. So, now they looked at the kidneys — the urine samples, and now they find a dramatic difference in not only basically the decrease in mortality, a 30% decrease in mortality —

Taubes: The problem — we know kidney function is being moderated by hyperinsulinemia and other factors, so this — can that affect polyphenol —
Sears: But, you know, but, but, but you see it, again, you're seeing that — seeing the levels of poly — So, again only those who basically get into the blood, because if they don't get the blood they don't do you any good.

Taubes: No, what I'm saying is we've now assumed that, that, that their health status, their insulin resistance status, hyperinsulinemia, whatever else, is not going to affect how their kidneys process the polyphenols and so how much they are going to excrete through their urine.

Sears: But all that being as a given — which was you know, true. But nonetheless, I was saying that what they found was a significant difference in mortality. And then, what they also found: a significant difference in, you know, frailty, sarcopenia. So, again, we have some indications of, you know — polyphenols are very difficult to measure, just like eicosanoids. They're like the will-o'-the-wisps. They say, “Where are they?” Cytokines are no different. So, we now know that — and we now can understand as we — In 1995, there was virtually no knowledge on polyphenols and their health effects. Today, we have a very very large body of knowledge. But the most interesting thing from the Zone standpoint, is the effect of polyphenols to really now affect gene transcription. And that's something of a fascinating aspect — able to basically affect gene transcription, or really the AMP kinase, which orchestrates a wide range of other gene transcription factors that basically control our metabolism. So, now we know, we have at least a hypothesis as we remove polyphenols from the diet by increased food processing. What we are doing, we are taking out of our system a powerful agent to modify gene expression. And we're the worse off for it.

Taubes: Oh, and we're also adding whatever we are adding in the process —

Sears: That's why I say, see —

Taubes: I mean, that was the debate in the 1970s, because you had Peter Cleave arguing that refined grains and sugars cause these diseases. And then Denis Burkitt, another missionary physician, comes along and says, “Well, we're never going to tell people to eat less sugar and drink less beer. That's a non-starter.” And he was obsessed with constipation, so he decided, it was — started the fiber discussion by saying, “It's what we remove, rather than what we put in.”

Sears: And now we can now look 20, 30 years later. Now we have the sophistication to say, “You know, they're probably both right, in a way.” So, again we're, as we have more sophistication —

Taubes: I'm a little more skeptical.
Sears: Oh, I'm a total optimist.

Taubes: And that's a good thing. So, let me ask you a question: What would you do and think differently — so we've been — the Zone came out 23 years ago. What would you have argued differently, or suggested differently? Or now, you know, going to your 50th reunion, what would you have done differently, even socially, to get this message across? I mean, considering whatever the response you got back then. I wasn't paying enough attention, or maybe that was a good thing. And then —

Sears: Nope, nope, nope. I would say, nothing — Nothing in my message has changed in the last 23 years. You know, now there's been some more of, you know, of greater understanding. The role — the discovery of resolvents. They were not discovered until 2001. The role of polyphenols. The role of basically fermentable fiber and gut health — this is all new information that we didn't — couldn't do until we had a basically gene sequencing of microbes in the gut. So, but everything that basically has even happened since 1995, it's not changed one iota of what I wrote. In fact, if anything, it has basically only solidified — it was a working hypothesis, which we now have a great number of published clinical trials under highly controlled circumstances that say, “Yes, it does work.” And therefore saying — but it's not one diet for everyone. That's why I say it starts with one thing: How much protein you need, and that's unique to individuals. Not how many calories, how much protein, and then once you have the protein, everything falls out mathematically, just like quantum mechanics.

Taubes: Okay, but do you still think — because again we live today in a world where we could probably walk outside here in Manhattan Beach and get a megaphone and go, “How many people are on the ketogenic diet?” And voices will pop up all over. This you know — you're restricting some of the same things. They don't believe that 40% of — they think for many people, insulin levels are going to be too high to mobilize and oxidize fat with 40% carbs —

Sears: No, no, no, no, this — that's a misunderstanding of the Zone diet. It's a calorie-restricted diet, so the amount of carbohydrates you're consuming is not a massive amount. If you're consuming 1,500 calories per day, and 40% of those are carbohydrates, that's about 150 grams of carbohydrate per day. The brain needs about 130 grams of glucose per day —

Taubes: It doesn't need it.

Sears: It does. It does need it.
Taubes: No, it uses 130 grams in a carb-rich environment. But it certainly does not need 130 grams of —

Sears: It uses it — it uses it because it has to make a lot of ATP to basically maintain neural activity. You had to get the ATP from something. The brain can't use a high-octane fuel, which is fat.

Taubes: The brain can use ketones as fuel.

Sears: I said a high-octane fuel. Ketones are low-octane fuel.

Taubes: Okay.

Sears: So it says, “I had to have lots of ketones,” but if — Remember we have a certain degree of, perhaps, you know, optimism. The body basically moves toward optimization. If ketones were the optimal format for, you know, brain function, then basically we will have evolved as a species to use almost ketones primarily. But even in cases of complete starvation, this is the Cahill studies, complete starvation, the glucose levels never drop to zero.

Taubes: Now let me ask you a question, because the Keys starvation studies — yeah, and the glucose is never gonna drop to zero, so that's not an issue. But then the Keys starvation studies, they were feeding — he was feeding his conscientious objectors 1,600 calories a day, and they were, they went crazy on these numbers, and one of them even cut off his fingers to get out of the study, and now you're suggesting that 1,500 is a reasonable amount.

Sears: Most people, including you and I, would be hard-pressed to eat 1,500 calories a day on the Zone diet. Let me give you what that would be. On the Zone diet, you'd be eating about 30 grams of protein at each meal. How much is that? The amount you could put on the palm of your hand. There's, every dietician will say that, say, “Never consume any more protein at a meal than you can put on the palm of your hand.” I agree, but I had never consumed any less. Now, for carbohydrates, let's say you need 1,500 calories per — say of this case about the — Oh, about what we need about — Oh, 600 — about 140 calories — grams of carbohydrates. That will be about 12 servings of vegetables.

Taubes: Green vegetables.

Sears: That's right. Non-starchy vegetables.

Taubes: Okay.
**Sears:** And say — you say, “No man could eat that much.” I say, “You’re not done yet. You need some fat.” Now what’s 30% of calories of — oh, it's about 50 grams of fat. It's about two, maybe three teaspoons of olive oil. One teaspoon of olive oil each meal. Okay, we have the amount of protein in each meal you fit in the palm of your hand. We have a massive amounts of vegetables, and some olive oil. I say, “No one can eat that much.” There's the Zone paradox: A calorie-restricted diet, it's virtually impossible to eat all the food.

**Taubes:** Well, again, if we're eating the 12 — what is it? — 12 cups of green vegetables —

**Sears:** Yeah.

**Taubes:** Okay. Yeah, I can imagine that that's difficult. What are the vegetables we're making for breakfast now?

**Sears:** Well, let's say you like things like, um onions? Do you like peppers? Do you like asparagus?

**Taubes:** For breakfast?

**Sears:** Yeah. Why not?

**Taubes:** Cooking asparagus for breakfast?

**Sears:** Why not? Hey, have you ever had, basically gone to a restaurant and gotten a vegetable omelet?

**Taubes:** Not in a long time. But I used to in my youth when I ate something —

**Sears:** I want them — Well I want the vegetable. I want the vegetable omelet, and I want three more servings, side servings, of vegetables.

**Taubes:** Okay, let's assume we do that. So, that's what I'm eating every day for the rest of my life?

**Sears:** That's right.

**Taubes:** Okay, so I'm now calorie restricting. I'm not so sure I'm — you know, whether or not I can eat that amount of green vegetables? And we'd have to do an experiment here, but there are some less-than-ideal carbohydrates.
Sears: Of course, a less favorable — less favorable carbohydrates. Things that you're not supposed to eat.

Taubes: Are there any on the list — it's a long list, and there, they have a low-digestible carbohydrate content. So, in many ways — I'm not sure 12 cups — how many digestible carbohydrates would that be? How much?

Sears: Fermentable? Nobody knows. There is no way to measure them, because it depends on, what is the definition, and do you have the right microbes in the gut that can digest them?

Taubes: Okay. Now, when you feed — so, I'm eating four servings of green vegetables for breakfast, four servings of green vegetables for lunch and four servings of green — No I'm not, because I actually have to divide it up into snacks too, if I remember correctly.

Sears: But if you're not hungry, you don't need a snack.

Taubes: I don't have to have a snack if I'm not hungry. Okay.

Sears: Because you're stabilizing blood sugar levels.

Taubes: Okay, and that would be better than eating, let's say, eggs and salmon for breakfast, as much as I want. And then lunches and dinners are gonna be similar, but now I'm out of the Zone. So, I'm gonna need my four green vegetables, and for lunch with a palm-sized portion of protein, which has fat in it, so unless it's skinless chicken breasts, it's about 50% carbs and protein and fat by calories. I don't know, not by grams, but by calories, and the same for dinner. So, all I've done is I've switched my breakfast, because, I don't — I can't imagine it — I can't imagine eating onions for breakfast. I can't imagine eating asparagus.

Sears: I bet you can. I bet you can.

Taubes: No, I mean, I don't want to. Let's just put it that way.

Sears: Yes. No, that's a different, that's from —

Taubes: But now I'm out of the Zone.

Sears: Yeah, but the fact is, the hormonal responses to the diet will last five hours. They will last only five hours, and basically, each meal the game starts again. It's all dependent on the ratio of protein, carbohydrate and fat. And David —
Taubes: But what I'm saying is if I'm having my eggs and bacon — eggs and salmon. Leave the bacon out of it, because that's a loaded — Eggs and salmon for breakfast, and I'm not having lunch, I mean I'm not having lunch until that's — we're gonna eat breakfast at 8 o'clock, we eat lunch at 1 o'clock, that's reasonable. I'm out of sync in the mornings. Then I get back in sync and then I stay in sync until about 1 o'clock at night. Is there gonna be a significant difference in my quality of life if I do this? Would you actually expect me to live longer if I have asparagus for breakfast than if I have salmon for breakfast.

Sears: Well of course, the answer is yes. Of course I expect you to live longer. But we don't know. We don't know. And that's why you say, we take the best estimate we have in science and nutrition, which is limited at best.

Taubes: Well, arguably, if we're right — if either one of us is right, any best estimate from the nutrition community with — unless we carefully select-slash-cherry-pick the articles we like, is going to be of limited value.

Sears: I agree. And that's why we do clinical studies. That's the only way you have to, basically — any type of dietary philosophy has to be analyzed in the crucible of controlled clinical trials. There's no other way, you see. Otherwise, you become in the area of faith. Science is not based on faith. It's based on, “Show me the facts.” But to do controlled clinical trials, most people who do diet studies are incredibly lazy.

Taubes: Yeah, well believe me, we don't have to go there. Can we get back to the 2007 study that —

Sears: Yes.

Taubes: A calorie-restricted ketogenic diet versus a calorie-restricted non-ketogenic low-carb diet. One of the arguments for ketogenic diets going back to the 1930s by researchers who studied them is that if I can allow people to eat more, they'll be happier, and they're more likely to sustain the diet for life if they're not consciously trying to restrict how much they eat. You are saying that while they eat the Zone, they won't be hungry. We don't actually if that's true. I mean, I can imagine it sure — I certainly can imagine if they go from a standard American diet to drop the carbs, drop the glycemic load, get rid of the sugar, they won't be less hungry, but they may not be as less hungry as they will be — the people. The advocates of ketogenic diets will say, “You look at every study ever done on ketogenic diet,” I mean even the American Medical Association said anorexia was an undesired side effect of the diet. You know, people just aren't hungry. So, if you allow them to eat as much as they want, they'll be happier and healthier and they'll just — happier — They'll sustain it longer, they'll lose more weight, et cetera, et cetera. I could see the
benefits of calorie restricting if the benefits of calorie restriction aren't just the same as carbohydrate restriction. But with a conscious attempt to eat less.

**Sears**: Well, I don't know if it's a conscious attempt to eat less. It basically, the hypothalamus says, “Don't eat.”

**Taubes**: And why does the hypothalamus say, “Don't eat?”

**Sears**: Because of getting inputs — hormonal inputs from the blood, the fat in the gut giving you basically a guidelines, as basically, do we have enough nutrients in the body? That's why from the fat, the leptin will interact with the hypothalamus saying, “There's enough fat down here.”

**Taubes**: But the fat will also — the leptin would interact with cells in the body to tell the body —

**Sears**: But the hypothalamus basically is your control central.

**Taubes**: Well again, that's dependent on the theory that —

**Sears**: Good theory.

**Taubes**: No, no, no — It's dependent on a worldview, a way of thinking that ignored the fact that these lesioned animals — The whole hypothalamic thinking is a control center of appetite and satiety came out of a world in which they didn't realize that what the hypothalamus might be doing is controlling fuel partitioning. Fuel use in the body —

**Sears**: Well, but — it can only tell what the — The brain is basically blind up there, so it has to have hormonal inputs. The amount of protein the body needs is basically coming from the release of the proteins PYY and GPL-1 from the L-cells in the gut. The levels of basically of sensing how much fat do we have. The only way the brain can find out is saying, “Is leptin interacting with the hypothalamus?” I come from the old school, saying kind of the brain is your — kind of the apex of the body.

**Taubes**: And I don't. That's the interesting —

**Sears**: So, and —

**Taubes**: I think the old school happens — It used to be that we thought the Earth was the center of the universe, and it's natural to think the brain is the center of the body. But one of the arguments against that is the brain is the organ that's most
protected from fuel, lack of fuel. The brain will be protected far longer, so why put a sensor of fuel availability in the brain when the body is gonna die before the brain?

**Sears:** But it's not this — The brain is not sensing the fuel. It's sensing the hormones. The brain is sensing the hormones from the gut. It's sensing the hormones from the fat and —

**Taubes:** What I'm saying: An alternative hypothesis is that the liver is sensing the actual availability of fuel. This was a hypothesis which is that, it's not it's, it's actually monitoring AP — ATP production.

**Sears:** Well again, but that's why the ATP production is key. If you can maintain ATP production —

**Taubes:** You won't be hungry.

**Sears:** Right. Exactly.

**Taubes:** Yeah. So, and one of the interesting things about sugar that I found so fascinating is that you require more ATP to metabolize fructose, and you produce — you get a decrease in ATP production when you consume sugar, which could —

**Sears:** Activate AMP kinase.

**Taubes:** The decrease.

**Sears:** That's right. So, that's why I say in terms of sugar — Sugar, remember, there's two fuel sensors that basically the cells have. One is A and D, and the other is ATP. Glucose requires a lot of use of, you know, A and D to basically make ATP. It's all about ATP production: The actual amount or the the byproducts being consumed. So, these are the two real fuel sensors as saying either to eat or not eat. So, one of the things we're trying to do is say how can I basically generate the greatest amount of ATP with the least number of calories? And to do that, I also have to make one other thing. I had to have my mitochondria working at peak efficiency.

**Taubes:** Again, you could argue, how could I generate the greatest amount of ATP with the least amount of carbohydrates.

**Sears:** Or the least amount of calories.

**Taubes:** I don't see where the — We do have a — So we have research suggesting that — although, even the calorie restriction research — There's a whole host of
mouse models that do not live longer when they're calorie restricted, and there's some that actually died prematurely when they're calorie restricted, and we know in the mon— You mentioned in ’95 that those monkey studies were in order, were in the works, and there were two primate studies, one at Wisconsin, one at NIH —

Sears: Two very very different protocols.

Taubes: And very very different — And the researcher who was doing the Wisconsin study pointed out that at least in their diet, the monkey chow, again is, it's corn oil and sugar, and maybe the animals live longer because they get less corn oil and sugar, not because they get a lot fewer calories. So again, there's a lot of ways this can be interpreted, and we tend to interpret it from the perspective we embrace already, but that might not be the right one.

Sears: Don't we all?

Taubes: Yes, and that's why I use the word “we.”

Sears: Yes, and but, and — That's why I say, “Good.” Now, because we have — we go back to a hypotheses generating of ideas. We have a hypothesis. Now, if we put together a good clinical trial, now good is basically of —

Taubes: This is why when I — you did that in 2007, and that's why I'm saying there was another body calorie-restricting both groups to, in order to have a legitimate Zone diet, you no longer had a legitimate ketogenic diet.

Sears: Well, and that's why I can only control two parallels at one time.

Taubes: That's why you do multiple studies.

Sears: You're speaking to the choir.

Taubes: Yeah.

Sears: Speaking to the choir.

Taubes: Anyway, Barry, I think we've — we could go on for hours, but I'm not sure our viewers can.

Sears: Oh, pity them. Pity them. No but, again, I think — yeah, I agree with you 100%, because this is a fasting area. It is about, you know, everything that is central to our future as mankind. What should we eat and why? And then say, and realize, and then say, “How can we adjust it to the individual?” A dietary program.
“diet” comes from the Greek root meaning “way of life.” What will be the dietary program, the way of life you'll follow for maximizing your health span? Not how long you live, it's your health span. And this is the — it really is a critical aspect, because right now we have abdicated our future to the processed food companies and the drug companies. So at some point in time, you have to stand up and say, “I'm gonna take back.” You know, be like of the character out of Network. So, put your head outside the window, say, “I'm mad as hell. I won't take it anymore!”

Taubes: Remember, I have to remind you he commits suicide in that movie.

Sears: So, hopefully that will not be our end.

Taubes: Yeah.

Sears: My pleasure.

Taubes: Take care, Barry.