## Introduction to Gary Taubes

We've gotten ourselves into this fix with obesity and diabetes, not because people just doing the research didn't think very hard about what they were doing.

They didn't never question their assumptions. They never said to themselves something simple like, why is I'm thin in this person's like my Aunt Elma is not because I've seen a reach.

You doesn't need a lot of food. And yet she's rotund and I'm lean. So how can I blame her obesity on eating too much? If she eats less than I did. This is doctor talks.

So thanks for joining us, Gary. It's you again. Thank you for having me, Jason. Yeah. So I just want to introduce our viewers to Gary Taubes. In case you don't know him, he's sort of the OG.

He's like one of the guys in this space. Everybody knows and everybody really respects. Because you've been probably one of the most influential voices in nutrition, which is interesting because that wasn't your original background, in fact.

But he came to it sort of happenstance. And in your sort of groundbreaking book, Good Calories, Bad Calories, really laid into some of the myths of nutrition and some of the really things that were just sort of, I don't know, I guess a lot of the stuff had never been looked at critically and really sort of laid it to bear.

And so that's one of the reasons everybody really respects you and really been trying, you know, you've been in this space for a bit now and really has been a champion of good science, which I really appreciate you.

So thanks for being here. I guess let's start off with your background, how you got into the space. You know, what made you interested? I mean, I think that's a great story, a great back story to start with.

Okay. So yeah, I'm a journalist. I'm not a physician. I'm not a I'm a PhD. I'm not a nutritionist. I have a hard science background, physics and engineering.

And then when I went into journalism, I sort of stumbled into studying bad science. Technical term might be pathological science, which is the science of things that aren't so.

And my first book was about high energy physicists who discovered, much to their chagrin, nonexistent elementary particles. And I documented how they came to learn they were wrong.

My second book was about the sort of great scientific fiasco of the second half of the 20th century, called cold fusion. Cold fusion now shows up in every other science fiction movie as the, the, the, the fuel that will change society.

In doing these books, I was mentored by some exquisite experimental scientists in sort of what's necessary to get the right answer in science, how critical you have to be, how skeptical you have to be, how office you have to be, because the possibility that you've screwed up and over interpreted your evidence is just much greater than the possibility that you have to get something right and do something worth paying attention to.

And when I finish my cold fusion book, I had a lot of fans in the physics community, and some of them said to me, that book was called Bad Science, and it came out in 1991.

And some of the my physicist friends said, if you're interested in bad science, you should look at the stuff in public health. That's terrible. And I did, and they were right.

That's the short form. Everything I had learned from these, these hard nosed scientists working in hard sciences and hard sciences or sciences, where you can actually test your hypotheses rigorously.

Everything that I had learned about what you had to do to make sure you got the right answer was considered kind of a luxury in public health, because for the most part, we can't do it.

If you have a hypothesis at some aspect of your diet or environment causes a chronic disease, you can't really test that hypothesis and experiment.

## How Taubes Got Into Bad Science

You can't randomize people to, for instance, smoke cigarettes or not smoke cigarettes and see run it out for 20 years and see if the people who smoke it more lung cancer.

So instead of doing these experiments, I mean, you technically you can do them. They're always possible, but they're either unethical or exorbitantly expensive.

They could take decades to do. And it's very easy to screw up an experiment. So if you take a decade doing an experiment, you don't actually know if you got the right answer or not.

So then you have to spend another decade replicating it, and you have to get the funding to do all that. And so you have to convince the people in the government that they should spend tens of millions of dollars funding a trial that may or may not get the right answer.

And when you're done with that, they're going to have to fund the same amount of money again to do the study over or to improve upon it, to increase the likelihood that you got the right answer.

So instead, we ended up with these fields public health and nutrition and chronic disease, where for the most part, our hypotheses are never really rigorously tested.

And people believe them in part because their colleagues believe them and their colleagues believe them because they believe them. And you end up with dogmas that may or may not be correct.

And in the late 90s, I was working then doing most of my work for the journal science as an investigative reporter, and I launched into first an investigation of this conventional thinking.

That's that high blood pressure is caused by excessive salt consumption. And this evidence to support that turned out to be sort of woefully inadequate to do the kind of public health interventions we were doing.

And it turned out that the researcher who had been most prominent in promoting this low salt diet dogma was one of the worst scientists I had ever interviewed.

And he also took credit for getting the world on the low fat diet we were all eating in the 90s, and when he did that, I literally hung up the phone and called my editor at science and said, well, I'm done writing about salt.

I'm going to write about fat. I have no idea what the story is, but if this guy was involved in any substantive way, I can guarantee there's a story there that we don't know about.

And the low fat dietary dogma that we all grew up believing that a healthy diet is a low fat diet also turned out to be built on, you know, sort of inadequate scientific evidence.

A lot of people seeing what they wanted to see in the studies and ignoring whatever they didn't want to see, which happened in that that situation to be the great bulk of the evidence.

And then that led me into my first book with calories, bad calories. And turned out while I was sort of in doing these

investigations and learning how bad the science was about these, what I think are incorrect theories, dogma that there was a very obvious alternative hypothesis that had always been mostly ignored or swept under the rug because it challenged the conventional thinking.

And that was this idea that refined, processed carbohydrates and sugars are the primary cause of the sort of chronic diseases that are so common in our lives now.

So BCT, diabetes, heart disease, and then you can even get the stroke, cancers, Alzheimer's. The further you go out on that spectrum, the harder it gets to I mean, the more you're speculating.

But I ended up writing my book so that in one hand kind of explaining why, well, we had grown up believing was almost assuredly wrong, and then supplanting that belief system was something I believe to be right, which is where I stepped over the line from being a journalist to an amateur scientist, and probably pissed off people more than what I think is interesting is your hard science background, because the thing about calories is.

So I grew up, I went to medical school in the 90s, so I grew up in the thick of the low fat era. You know, all fat was bad. I learned that through medical school.

Super interesting because when it came to weight and calories, what you always hear about is it's like the first law of thermodynamics. And I'm always like, God, like the hard scientists think that's all crap.

And yet the nutritionists and the, you know, people trying to say it's all calories are equal and all this sort of stuff are always like trying to point to hard science.

I called it physics envy, you know, because they want to try and pretend they got this sort of soft science with very poor evidence, and yet they're trying to link it to physics, which is, you know, much more definitive in that way, especially this whole idea of cutting calories and, you know, fat being bad for you.

And I find it interesting that, you know, that that, you know, the nutritionists think that they're hard science, the hard scientists think that they're just, you know, really, really poor.

Like it's just thought science at all sort of thing. Right? It's just a lot of opinion. I mean, this idea that we get fat because we too much seem so intuitively obvious that, I mean, we all spent decades of our life believing it just seemed natural even.

Yeah. Didn't matter. Like I often look back, I was in the junior high school in the 60s. I'm old enough and, you know, there was two obese kids in my junior, two kids with obesity in my junior high school class.

I knew it because we all did heavyweight wrestling, and there was three of us who were sort of big kids and two who were 40 pounds heavier. We never thought of them as kids who ate too much or kids were lazy, just thought of them as kids who were different.

And you didn't, I? I can bet we didn't treat them with the respect they deserve, but everyone does. But still, we never thought of them as suffering from gluttony and sloth.

But this became the sort of common way that the medical community thought about this disorder. And I do actually. I've spoken a very good physicist. I've tried to enlist in my cause, and I can't get them to understand that it's not necessarily about eating too much, and it may not be about eating too much for anyone, that there are people who are predisposed to get heavy and they're going to get heavier, even if they restrict their diets their whole life.

And there are people who are predisposed to stay lean, and they're going to stay lean even if they eat a lot. I have a sibling like that. No matter how much he ate, he couldn't.

Just like Lean and Boston, I would get sick and he would stay thin. The you know what happened when we talk about good science, one of the founding principles of good science writing, these can never fall in love with your

hypothesis.

So the way to avoid falling in love with your hypothesis, your belief. Because once you fall in love, you're going to just spend it very much relates to romantic attachments as well.

You're only going to see the good things about the person you're in love with. And so you come home and you tell your mother, I'm going to marry this wonderful woman.

And your mother looks at her and sees somebody who's completely inappropriate for you. And all you see are, you know, she's got beautiful hair and look bigger, and she can hold an intelligent conversation with me.

And like, three years later, you're thinking, I should have listened to my mother, or she's thinking she should have listened to her mother. But we see what we want to see once were with someone or some idea.

## Questioning Calories and Obesity Dogma

And the way you get around that and science is you make sure you have alternative hypotheses, so many different ways you can interpret the data. And if you can think of all those many different ways yourself, you'll be equally enamored with different variations, and you won't selectively see only that which sort of feeds your love.

And in nutrition and obesity, they just never did this. They got this idea and based on the laws of thermodynamics, and they thought it was physics, they did have physics envy.

This is like a hundred years ago that this determined why people got that. They would never say it determined why people got tall, even if somebody tall was very heavy, or why children grow or any of these other growth phenomenon.

But that became their belief system. And it's still nowadays people spend a lot of time trying to blame obesity on something other than behavior. So it's like ultra processed food, right?

And why is ultra processed food bad for you? Because if you blame it on processed food, you can blame it on the food industry. The reason it's bad for you is, in theory, because we do much of that, and the reason we too much of it is because it tastes too good.

And then if you ask the question, why do some people eat too much? And other people clearly don't because they're lean. Now you're back to blaming behavior.

And so one of the issues that I brought up in my books is that people we've gotten ourselves into this fix with obesity and diabetes, not because people just doing the research just didn't think very hard about what they were doing.

They didn't never question their assumptions. They never said to themselves something simple like, why is I'm thin in this person's like my aunt, you know, Elma is not because I've seen her reach.

He doesn't need a lot of food. And yet she's rotund and I'm lean. So how can I blame her obesity on eating too much if she eats less than I do? Yeah, I think that the the whole idea, it comes from this sort of calories and calories out sort of idea, which is that, hey, calories in is what you eat and calories out is exercise.

So therefore that's all under conscious control. So therefore if you gain weight, it's your fault. Therefore we can make fun of you. We can shame you because it's, you know, it's that person's fault, which I actually think is super, super unfair.

So, you know, in terms of the, you know, the sort of you point out these sort of prevailing hypothesis, which is, you know, calories and it's just eating too much, it's all too much.

It's all about calories and so on. You know, maybe you can lay out your alternative hypothesis for this, because I think it's very interesting because, you know, when we're talking about longevity, you know, you want to talk about preventing chronic disease, as you said, and maintaining a proper weight is obviously a very good part of that.

Diabetes is another part about about it. And we'll go into that later. But, you know, how would you explain sort of in a nutshell, your alternative hypothesis, which, by the way, I and a lot of other, you know, doctors think is much more correct than the prevailing hypothesis, which unfortunately refuses to die.

It's it's always there. We're always arguing about this hypothesis. So okay. So and this let me link it into longevity. Because one way to think about longevity is the prevention of these chronic, the delaying of these chronic diseases that tend to kill us as long as humanly possible.

So if you can, if you can delay the onset of obesity and diabetes and heart disease and cancer and dementia and stroke, and, you know, the longer you delay it along, your first of all, the longer your health span is.

So the longer you're healthy, the better you feel, the younger you feel because you've delayed these chronic diseases. And then ideally you compress the chronic.

You know, at some point your body can't do it anymore. And it manifests itself in some series of pathological phenomena that will kill you. And they're very likely to be similar to these diseases.

So the question of longevity is also the question of preventing and delaying these major chronic diseases. And when I talked about this alternative hypothesis being sugar refined brain hypothesis that appeared in my research, obvious.

When you look back at this, it's basically there was a whole group of physician researchers, mostly British, because British had this colonial empire around the world, the British Empire.

So they had physicians working all around the world and in government hospitals and missionary hospitals. And they would notice that populations that ate their traditional diets, whatever their traditional diets, were.

So it could be, you know, mostly grain and mostly carbs. And Basia could be mostly, you know, meat and fish and sea mammals and the among, you know, the Arctic regions, it could be cattle and the pastoral populations and buffalo and then elk and the Native American populations.

Whatever it was they were eating, they didn't seem to get these common chronic diseases until they went through what's called a nutrition transition. So they started interacting with the West, with the Europeans and the European descendents, and we start trading them sugar and flour.

And when that happens, you start seeing these epidemics of obesity and diabetes. And as this science was being written about in the medical journals, was fairly prominent, researchers studying heart disease, studying obesity and then diabetes, of course, were identifying high blood sugar and elevated levels of the hormone insulin as possible causal factors in driving high blood pressure.

And this whole science of metabolic syndrome, which begins to sort of appear in the medical literature in the late 50s and 1960s, courtesy mostly of a Stanford endocrinologist named Gerald Raven, and then begins to be accepted.

Only the 1980s to 1990s implicates this whole slew of sort of prediabetes, obesity related risk factors and heart disease, and it's always linked to the sort of carbohydrate content of the diet and the flour.

So it connects completely with this idea of a nutrition transition, these other populations. So the end result is effectively the of the carbohydrates to each other.

Processing and the sugar content drive insulin resistance for lack of a better term, which is what type two diabetes

ultimately is severe insulin resistance.

They drive obesity and elevated blood sugar. Elevated insulin levels are the most likely cause of of atherosclerosis, coronary heart disease. You could link them to stroke, you could link it to cancer, or you could link it to Alzheimer's.

So basically, it's a very picture that emerged in all these different fields in obesity and body weight per se. The idea is that, you know, we don't get fat because what we we get fat because what our body does with what we eat.

This is a line to which I have to give Eric Westman credit, what our body does with what we eat is a process called intermediary metabolism, and it's dominated by the central nervous system and the endocrine system, hormonal glands and the dominant gland.

Driving fat storage is the pancreas, insulin in effect the dominant hormone. So once again, you've got basically you raise insulin levels. Your body preferentially stores calories, is fat, and the insulin inhibits the release of fat from the fat cells.

So if you're insulin resistant, you're, you know, storing fat, not burning it in effect. And everything ties together very neatly. It doesn't mean it's right, but I think it moves the tax better than than the whole calories.

Well, I mean the calories thing and we could get into it. And however depth you want explains nothing. And that's the simplest way the value of a theory is what it can explain, you know, explicate power and the idea that you get fat because you eat too much has an effect.

Is your explanatory power not explicate, explanatory, even, even the you know, if you say that all calories are equal, what you get, you know, this is the sort of dominant sort of theory.

Even now, I find it very strange because, you know, the body runs on hormones, right? That's how like, everything in our body is run by hormones, pretty much, you know, pretty much.

And certain foods stimulate certain hormones. Okay. That's not controversial in any way. Some foods will stimulate insulin and some foods won't really won't stimulate.

And again, that's not controversial. That's just fat. It's accepted fact that this point because you can measure these things. So it's like so if you eat 100 calories of certain food which stimulates a lot of insulin, say white bread, or you eat an egg which stimulates 100 calories, same calories, which stimulate zero insulin.

Yet somehow we have to believe that the super high insulin versus no insulin, same 100 calories makes no difference to the human body. Oh, it's like, why wouldn't it make a difference like this?

Nonsensical. You know, and this is the problem with people growing up with a belief system. Because you could sit down and I've done this. You sit down with these very esteemed, respected researchers or their professors at their institutions, their head of committees.

They serve on consulting boards. They work with the American Heart Association, they write journal articles and commentaries. They host conferences. And you sit down and make the argument that you just made.

And I've made this with them. I mean, it doesn't matter what. We're not talking about energy here. We're just talking about the effect on the human body or any organism and the macronutrient content of the food has, whether it's a carbohydrate or a protein or a fat or a very different effect on the endocrine response of that food.

The endocrine response has a very, you know, the fat tissue and the non fat tissue, the liver and the organs and the muscles that are burning the fuel respond to the endocrine response differently depending on which hormones are secreted and how much.

And so you've got different macronutrients, different endocrine response, different fat storage or fuel oxidation or

mobilization of fuel or use of fuel, even choice of fuel, whether you're going to burn glucose or glycogen or whether you're going to burn fat or, you know, ideally not protein.

And they'll look at you like, I mean, it takes used to take me seriously. I'd like to think they still do. But the problem is, is what you're saying to them is they have been wrong about the most profound concept in their field for their entire career.

And everybody they know, all the people they respect, who think like they do, are also wrong. And, you know, Jason Fong and a allergist in Toronto, or Gary Taubes, a journalist of all things, is explaining to them why they're wrong.

And nobody it's like I compare this. It's imagine, you know, be an atheist going in to speak to the Pope, and you're going to explain to him why God doesn't exist.

Yeah. Or the pope going to, you know, a you're a, you know, a devout Catholic going to Richard Dawkins or one of these very famous atheists explaining to them what God does exist.

And you just yeah, we're not built to accept this possibility. None of us can do it. Yeah. And we part, I think, is that the sort of if you just follow the logic, it's so logical.

That is, insulin is not an evil hormone because I get this all the time, right? Oh, you're saying insulins evil? I'm like, no, I'm not saying insulin.

Evil insulin is like a normal hormone. But to tell your body to store fat and to block fat burning, which is lipolysis, that's its job, right. And again, that's not controversial.

That's his job. I'm just saying that some food stimulate a lot of insulin.

## The Carbohydrate-Insulin Hypothesis

Therefore it will encourage you to store that energy as opposed to leaving it for you to use, which is, you know, to me is is not controversial in any way.

And yet they can't see past. And that's I totally agree with you. That's where that belief system, it just sort of Trump's everything. And in the end it's always hard to sort to discuss.

But even harder I think, is that they looked at the studies and then everything gets interpreted in that way that they're it is all about calories. And like, this is yeah, you can show.

So one of the reasons we believe we're right is you can tell people. So the simplest view of what often called the carbohydrate insulin model is carbohydrates stimulate insulin secretion.

You know, the sugar has an effect that's different than, than grains or starches. But that effect is and result is to increase insulin secretion. And insulin stimulates fat storage, inhibits the release of fat.

To eat carbs, you get fatter, you don't eat carbs, you get leaner. So we now have, well, there's 200 years of history of very influential commentators, physicians, writers saying, look, you know, carbohydrates make people fat.

That's the simplest way to think about it. Don't make everyone fat. Clearly not. But those of us who get fat get factors of the carbon content, the diet.

And if we don't eat those foods, we get leaner. And now we have 25 years of clinical trials confirming that. So we have a test. But the problem is often when people get leaner, they also eat less.

That's because I would say because they're burning their own fat also. So when you're weight stable, you're not

burning more fat than you're storing because it's staying stable.

But if you're losing weight, you're burning that fat. That's where it's going. So you don't have to eat as much so you're not as hungry. And so you eat less.

And now people say, look, we did the clinical trial. These people didn't eat as much. Therefore they lost weight because they ate less. So there's always a way to interpret the evidence.

Yeah. Not always. The key to doing good science is to creating an experiment where there is not an alternative way to interpret the evidence if, depending what you see.

But there's always a way around it, so they can keep their belief system intact, and they don't ever have to confront the possibility that they made a mistake of this magnitude.

Yeah, I hear what you're saying. I think it's because there is a confounding between calories and insulin effect, because if you keep the food stable, the more you eat, the more the insulin effect.

So then people say, well, it's the calories, whereas other people will say, well, it's the insulin. But it could also be the calories include carbohydrate calories. Yes.

Yeah. So, you know, if you let's say you starve someone, this is always the observation. When I was doing my research for good calories, bad calories, I said, we don't talk about calories because when we starve people, they get them leaner.

And I would say, yeah, but when you start thin people, they also get thinner. Yeah. They don't say they're thin because they eat too much, but you're willing to say they're fat because they eat too much, because the people with obesity also get thinner when you starve them.

But the point is, if you somebody is eating 2500 calories a day, or let's say 3000, that's a lot. Now, it wasn't a lot 60 years ago, and 50% of that is carbs.

That means you're eating 1500 calories a day. So now you starve them and you can say 600 calories. That's a classic very low calorie diet. And they're getting 300 calories from protein or 200 calories from protein.

And the rest is even if the rest is all carbs. You've cut their carb content from 1500 calories to 400 calories a day, so maybe that's why you're seeing what you're seeing.

And again, a good scientist says, oh, that's an interesting point. What experiment can I do to examine that hypothesis and see if it's valid? And you can plan experiments to do that.

But in order to do that, you have to legitimately be curious. You have to be legitimately accepting the possibility that your belief system is wrong, so that you can devote energy and ingenuity and effort into figuring out which is right. And that all comes back to having to accept the possibility that you screwed up.

Yeah, it's hard for people to do that. I mean, it's almost impossible. And again, I get accused of the same thing. And then I say, well, you're right. If I'm wrong, I'm never going to.

There is no evidence that will convince me. Yeah. And then they say, albums is so close minded and we can't trust anything. I said, I'm not being close minded. I'm being honest.

Yeah, I'm accepting that. I'm just like everyone else. We're all like this. The history of science. So the history of science for sure. I mean, I think that, you know, what to me is very interesting in your book, and I think this was quite sort of eye opening to a lot of us who read it, at least a lot of our physicians, because I've spoken to a lot of the physicians in the low carb sort of space, is that there was a lot of history of this being a, well, sort of accepted, you know, that carbohydrates, you know, they didn't know about insulin, of course, in the 20s, 30s, 40, 50, that

wasn't they didn't know about it, but they yet they knew and not just physicians, but almost everybody knew that sort of some foods make you fatter and some foods don't make you fatter.

So some foods are more inherently fattening than others. And those foods tend to be sugars and refined starches. And that was sort of just sort of accepted wisdom.

And that part was very interesting to me because one, I think, you know, if you ever ask, like your grandmother, they'll tell you exactly that, right?

You know, they'll be like, nobody gets that eating broccoli, right? It's like, but eat cookies. Yes, you're going to get fat. So stop eating cookies if you want to get me.

Yet there was actually a huge history, you know, and not elsewhere, like, but in North America, everybody was sort of well accepted for so long. And then it sort of all changed in the sort of 70s 80s.

So a lot of us just don't remember if this is, you know, Rich and exactly the time that people started that obesity started becoming such a noticeable problem, that diet books were becoming bestsellers regularly and clinicians would experiment on themselves.

Right. So they'd get this idea. There were some research articles through the 40s. Guy named Pennington, who was a work for the industrial medical division at DuPont.

And he had done experiments putting executives on low carb diets. And then people like Robert Atkins and Herman Taylor, who wrote a famous book called Calories Don't Count.

And so they tried it on and it worked. And you tried it on your patients. It's this conversion experience that we've all gone through. People say, well, you only wrote this because it worked for you.

And I said, well, that's not actually true. But if it didn't work for me, I would have been a much harder sell. The science wouldn't have meant so much to me if I when I tried carbohydrate restriction, I got fatter, for instance.

And there are people out there who swear this has happened to them, and I'm willing to believe them because I think we should believe everyone until remarkable evidence demonstrates otherwise.

But it just it's, you see, the same thing happen and virtually everyone and again, it's hard to judge without really well done clinical trials. What proportion of people benefit?

I mean, you know, in your patients the, you know, do you give these foods up, you get healthier. But this was happening in the 1960s. Again, there are other dogmas that entered into this field.

And one of them, beginning in the 1960s, was this idea that dietary fat causes heart disease and particularly saturated fat. And so now you have obesity.

Doctors and cardiologists like Atkins saying, look, if you want to get lean, don't eat carbs, just eat, you know, meat. And he said, cheeseburgers, double cheeseburgers, lobster Newberg.

Don't worry about the fat content. Just stay away from the carbs. And this outrage the rest of the medical community, because they were beginning to promote this idea that it was a dietary fat that caused heart disease.

And it's a reason people like me never experimented with Atkins, because who cares if you lose weight? If you have a heart attack, right? It's a trade off isn't really worth it.

So this saturated fat causes heart disease. Dogma became conventional wisdom. It was tested in multiple clinical trials. It never panned out in the trials, you know.

And yet people believed it anyway. But people well, again, it's the sociology and politics are fascinating. So researchers go to the Congress in the United States and they say, we want to test this hypothesis.

And Congress says, okay, we're going to give you $50 million to do the test. It's only seen as a success if the test turns out to it's a hypothesis can be confirmed.

So if you do the test and then you say to, well, it turns out we were wrong, which in any other science is a massive victory if refuted your hypothesis, that's that's when you have to do you have to get rid of all the wrong stuff, and then you're eventually only left with some ideas, the few of them that might be right.

And then you slowly do those experiments and you would have it down until there's only one theory left that you can trust. But in this world, if you came up with what was seen as the wrong answer to your hypothesis was incorrect, you not only failed the medical community and the public, you failed congressmen who had been so kind as to a lottery this money to do the study.

So there was an extraordinary amount of pressure, both the researchers who wanted to prove they were right, but who wanted to justify the investment. And again, despite that study after study failed to confirm, we pushed it anyway.

The public health people, the US, the agricultural Department, the USDA, the you know, you're pushing this idea that we should all be low fat diet. It was probably not just a coincidence.

And most of the processed foods and the miracle are high carb. So, you know, you're pushing agriculture, you're pushing brains and sugars as much as livestock.

We end up with an untenable sort of conflict between what you have to do to lose weight and what you're supposedly have to do to prevent heart disease.

And if we're talking longevity in doing this conversation 30 years ago, you're telling people to avoid fat and avoid salt and avoid saturated fat specifically.

And now, 30 years later, we're saying, you know, don't eat sugar, refined grains and seed oils depending on the strength of your belief system, the whole salt thing hasn't really there's not been a lot of evidence for that either.

Well, that's what got me into this. It's it's less press, but it's still also one of those things that got pushed and still out there. I mean, it's sort of again, as a nephrologist, I get this all the time.

And this whole idea that eating a low, low salt diet is good for you. It's there's no evidence, really to suggest that that's true. There's even evidence to suggest that.

Quite the opposite. Yeah, but this was just, you know, it was the nature of the sign. The whole idea of establishing dietary cause of product disease is basically a post-World War II conception.

Yeah. And this idea of, you know, using epidemiological studies, observational studies, these huge cohorts of 100,000 people to see what they eat and who's healthy and who's not.

As a post-World War II phenomenon, brand new sciences. And the people doing this, they weren't trained as like physicists or engineers. They didn't have they were, you know, they they were nutritionists and dietitians, and they had no real conception of what the real science is.

And they were making up the rules of these sciences as they went along.

## Why Calories Alone Donu2019t Explain Weight Gain

So let's talk about diabetes because you wrote another book, your most recent book, Rethinking Diabetes, which

was, again, the very insightful sort of, you know, huge amounts of information that I hadn't known before about diabetes, because that too is linked to obesity, obviously.

So weight loss, diabetes there linked. And both have a huge impact on health, mostly cardiovascular disease and cancer. But how does that sort of bad science play out in the field of diabetes?

Well, again, that's another long story. I mean, the book was written during the research, by the way. I wrote that book just because I thought diabetes is the most it's kind of a crucial disease in this discussion because, okay, you could argue with obesity.

It's not the carbohydrates, it's the calories. A calorie is a calorie, it's a calorie. But diabetes is a disorder of insulin dysregulation. Type two diabetes is in some resistance.

Type one diabetes is insulin deficiency. The insulin is responding to the carbohydrates on a fundamental level. I mean the glucose and the circulation stimulates the pancreatic beta cells to create insulin.

So we can put aside this whole calorie as a calorie thing because it's not relevant. And we can just ask for patients with diabetes. What's the proper way to control this disorder.

And how do we prevent, you know, in the past 60 years, the prevalence of diabetes in the United States has increased sevenfold. I mean, imagine any other disease increasing seven X in 60 years, and there'd be this public outcry.

And here there's none. It's just people say, well, we have drug therapies and the drugs and device therapies are getting better and better every year.

And and it's linked to obesity. So these are people just don't have the problem. They don't know how to eat moderation. So we can't have an outcry because it's it's a patient's fault right there.

They're eating too much or eating the wrong foods or whatever they're doing, but it's their fault. So it turns out when you look at the history. So the first sort of successful treatment of a diabetes case was in 1797 by a British physician working for the British military, who put an army colonel who, with diabetes, a guy, had lost a lot of weight.

He was thirsty all the time. He was peeing constantly. And all these symptoms of out of control diabetes. And this British doctor decides that, you know, look, the the way you diagnose diabetes, you look for sugar in the urine or back then they had their assistants taste the urine.

And if it tasted sweet, you knew they had diabetes. Ideally you had an assistant for that job. The the. Was interesting how many diseases were diagnosed by tasting the urine back then, but that's a digression anyway, he figures a urine sweet.

It's a carbohydrates are not being properly metabolized. So let me make this guy you to die without carbohydrates. It's basically fatty meat and rancid meat and some green vegetables.

And the guy gets better. Colonel Meredith gets better. He actually lives for 12 more years. And Rollo tries in another patient, a general who is someone gets better, but then falls off the diet, goes back home.

His regular doctor says, don't worry about you. Eat and he eats whatever he wants and he dies. So Rollo writes a pamphlet explaining what he's done, and he circulates the pamphlet all throughout the United Kingdom.

And he asks physicians back then I try it with your patients. Diabetes was so rare that a physician might see a patient once or twice in his lifetime and let me know what happens.

And few dozen physicians right back to him when he publishes the revised versions of his pamphlet with their

letters saying, you know, it works. I get people off carbs.

Their diabetes more or less goes into remission. And so through the 19th century, this becomes the standard of care. The diabetes is seen as a disorder of carbohydrate metabolism, and the idea is that they can't metabolize carbohydrates safely, don't eat them.

But because the patients usually whether type one or type two and they didn't differentiate back then, they would usually show up at the doctors only after having experienced significant weight loss.

You want to put weight back on them, so you don't want to. You want to replace the carbohydrate calories that they're not eating with something. And the one macronutrient that doesn't stimulate incidence creation is fat.

So through the 19th century, these diets became higher and higher and fat. And by 1900 doctors are saying, look, the the essence of a diabetic diet is butter.

And their patients, you know, their older patients, the younger patients who have type one diabetes cannot they they need insulin and they're going to die without it.

And they do. This diet will slow down the disease progression, but it won't stop them from passing away from pretty awful deaths. But the older patients, who we would now say have type two diabetes, did fine.

They lived as long as anyone. And there's some variations in therapy. A Harvard doctor comes along and does what I'm getting used to Harvard doctors doing, which is pushes a bad idea and it gets accepted.

And now they're starving all these patients, and you end up with these photographs of these kids who suffer from this disorder, type one diabetes, that makes them ravenously hungry.

And then they're treated by being starved. So it's a little bit breezy and they're emaciated. They look like they, you know, the concentration camp victims from World War Two.

And then insulin is discovered in 1921, the hormone insulin. And it's a miracle drug. One piece for the Atlantic earlier this year that how this affects how I see the drugs like will go.

And you know semaglutide which are their obesity drugs and also can be wonder drugs for the people who suffer with these disorders. So as soon as insulin comes in, you could give it to these kids.

The message to kids who are days away from dying or already in diabetic comas and revive them. And within days they're putting weight back on, and within weeks they're running around the wards, and within months they've gone back home.

And as long as they take their insulin, they're living apparently perfectly normal lives. And the problem is one of the side effects of insulin is low blood sugar is almost impossible to figure out how to dose this drug.

Everybody was different. And you can be different at different times of day and different days of the week. And if you exercised, you needed less or more insulin than if you didn't.

And if you got a cold, you needed. I mean, this was an incredibly dangerous drug. And it turned out that the easiest thing to do was you wanted to prevent low blood sugar.

So you told these kids to eat carbs. So they these are kids who didn't eat carbs and adults who didn't eat carbs stay alive. And now you're telling them to eat carbs to avoid the possible fatal side effect of the cure, which is insulin.

And as time went on to the 20s, doctors said, look, you know, these kids don't want to be on diets anyway. And these kids have been diagnosed with this awful disease.

You don't want them to go their whole lives not getting the ice cream at their birthdays or cake. And so let's just give them more and more insulin and let them eat what they want.

And in fact, this was known as a liberal carbohydrate diet. And it got ever more liberal. And it started with kids and then it moved to adults. And by the late 1930s, this is sort of now the standard of care.

You might tell them to carefully consume the same amount of carbs every day to make it easier to dose their insulin to the carbs, but they're also eating carbs to make the insulin doses safe.

Yeah, and they get to eat like their family, so nobody has to go on a diet. And by the mid 1930s, it's clear that there is a new epidemic going on. And these all these kids, you might have been dead at age 12.

You've now kept alive for 10 or 15 years, are manifesting all the classic complications of diabetes, heart disease, kidney failure, the neuropathies a nerve failure, the amputations are going blind.

And many of these patients, it's happening very quickly say go from being apparently healthy to dead within 18 months. And the medical community is mystified.

They assume it's because of poorly controlled blood sugar, because that seems natural. And the older patients are also manifesting these same chronic conditions is just happening slower with them.

But they're all getting insulin. And what they don't even think about is it's they don't actually know what the blood sugar is doing alone, because they didn't have people staying alive without using insulin to control the blood sugar.

All they know is that the combination of the insulin doses, which are very high compared to what you're paying for, is secretes naturally, and the high blood sugar, because you're still doing a lousy job of controlling blood sugar, might be causing these chronic conditions.

## The History of Diabetes Treatment

And they struggled with these chronic complications. We still struggle with these complications today, 80 years later, 90 years later, the side effects of complications, consequences of living with diabetes or can be awful.

Most of the, you know, is in Africa the dialysis, the kidney failure, most of the amputations in America, limb amputations are from Naropa and poorly controlled by Betis.

And along the way they never said to themselves they meaning the medical community at large, never said, well, wait a minute. You know, we never tested this alternative approach.

And we're actually physicians writing about this in the 1920s just don't feed these people carbs. Yeah. And I think that there's yeah, back then it was also confusing because there was the type ones and the type twos, which are really two completely different diseases.

Yes, they both have high blood glucose, but you know, so they're like, that's diabetes. But that's sort of like saying, oh, you have a fever. Well it could be from, you know, Covid, it could be from the bacterial abscess.

It could be from 10,000 things. Right. Lupus whatever. And saying, oh, it's the same disease. We should all treat them the same. It's like, so this is it's just so stupid sometimes.

I mean, it's when you look at the history I can report in this book is not, as you know, it's not a diet book. It's not any particularly easy read. But the reporting was then writing was fascinating.

The they couldn't measure, we couldn't measure normal levels in the blood. Okay. So the assumption was if you have a what they call an endocrine disorder, endocrine is a fancy word for hormone, you know, some kind of glandular disorder.

We assume the problem is with the glance. So there's something wrong. And they would have, you know, they had hypothyroid conditions, too much thyroid being secreted and you had hypothyroid too little thyroid, but you couldn't actually measure the level of thyroid hormones.

You just knew that you saw these symptoms for one and these symptoms for the other. And the hypothyroid, too little could be treated by giving the person thyroid.

So the assumption with insulin was it was all insulin deficiency because we could treat it by giving them insulin. And there was some talk of insulin resistance because he's older or heavier, patients with diabetes needed massive amounts of insulin to lower their blood sugar, but nobody really knew what that meant.

And not until 1960 is there a technology, a test and assay that allows you to measure hormone levels in the blood. So they're the very first paper by this team, yellow and Burse and very first paper they talked about insulin resistance because patients with type two diabetes are overweight.

Older patients with diabetes and obese patients have high levels of insulin and high levels of blood sugar, so that they're not insulin deficient. They actually have too much insulin.

And as you said, when talking about hormonal disorders and every other field, I'm reading textbooks from German textbooks, which I get translate ChatGPT or translate form, and I'm reading them from the 1920s and it's, you know, you've got, like I said, hypothyroid and hypothyroid, you've got hyper pituitary diseases and hyperspeed.

They hadn't discovered growth hormone, but then you've got typo genitalia. I knew that too much sex hormones created this disease. Too little created that disease.

But if you didn't actually see the symptoms, you just assume the symptom was high blood sugar. Then nobody ever questioned why is there one disorder? Did not until the 1960s.

Do you learn type two is too much insulin. Type one is not enough. Type two is insulin resistance type. Well, what's interesting about that is that that whole idea, like you're saying that these sort of when you see everything through a belief system, right?

So even through now you hear this now, which is, I find stunning type two diabetes where you can measure insulin levels, you know, they're too high. Everybody knows they're too high.

But it gets called in the medical literature as relative deficiency is like, what the hell is not doing its job right? It's not doing it. So it's too low actually, even though it's too high.

Yeah. And that justifies right. The treatment justifies give them more insulin. Yeah. Exactly. Which I think. Yeah. And then you, you do clinical trials and to the diabetes communities they actually do these trials because in obesity research they won't do it.

They know it's all about calories. So they're not going to test it. But the diabetes researchers have done clinical trials clinical testing their fundamental beliefs.

And they've refuted almost all of them. And now where they lose credit is they don't say. I mean, one of my favorite quotes was from a guy at Harvard who, in the New York Times in 2013 is quoted saying, you know, they do this huge study because fundamental belief in diabetes, if you could reduce weight type two diabetics, you will have less what are called macro vascular complications, heart disease, stroke and huge study called look ahead $200 million.

Get these people to lose weight. They get them to lose weight. There's no difference in heart disease completely demolishes the fundamental belief system in the field that gone back 100 years is the professor at Harvard very well respected?

Diabetes is quoted in the New York Times saying, we have to have an adult conversation about this, and they never do because it's what do you do? Like I said, nobody wants to accept the fact that they're wrong about these things.

But then when you're demonstrated that you're wrong, what do you do about that? Particularly if you're a clinician, you know, a medical doctor, if you're scientists, you could say, well, let's do some more tests, and then you're accused of being in it for the money because let's do some more tests means give me more funding and I'll do my studies.

But what do you do with that? And what they do with it is they just kind of ignore it. They just they write some commentaries and some some opinion pieces, and they move on and go back to doing what they've always done.

And if they wait long enough, you'll get a better drug, which is what's happened. Yeah, I think that happened to and I'm going to have to wrap this up.

But also in 2009 when the accord the advanced study. So this idea that everything bad was due to the high blood sugar. So therefore do whatever you have to do.

Get that blood sugar down. You're going to do better intensive insulin therapy. Give them more insulin. They'll do better. In fact they did worse. It wasn't one study.

It was multiple studies showed this. So that I remember being, you know, you know, I was in practice by that time. And I think this change I was reading these paper like, this changes everything, like everything about treating diabetes, because that's a huge disease for me as and and what changed in the treatment of diabetes?

Nothing. They didn't recommend any changes. I was like, My God, like what is happening here? And this is an obvious issue. It's just, look, there are two levers you could use to manipulate blood sugar.

You could use diet or you could use drugs. You made your levers, minor levers like exercise and sleep. But those are the major levers. And so we've demonstrated that drugs don't help.

A natural question to ask is, well, what if we pull the other level? What if we use diet to lower blood sugar? As we know, we did that for 123 years before insulin was discovered, and it seemed to work.

That's just a natural question to ask. But these people, they, you know, they also have when you hold an incorrect belief system and it's constantly being challenged by evidence, you develop sort of a defense immune system to defend yourself against these, these, these infectious nuggets of evidence.

And so you develop all these ways to think about the problem that convince you you're still right, even as the evidence is saying you're wrong. So one of these belief systems is nobody follows a diet.

Why would we do that? Why would we tell people to eat this way when we know they're not going to follow it? And then you could have a conversation. I've had conversations like that.

How do you know people are not going to follow it? And, well, it's because it's wants to eat like that. And I said, well, yeah, I'm going to acknowledge to you that that I do because I find that I could sustain a 30 pound weight loss, you know, I mean, I seem to be very healthy for the guy who's getting on in years, and I want to stay healthy.

And they'll say, oh, you're one of those keto people. And now they can dismiss you because of the there's it's just a and again, you can't blame the people like you actually allow the evidence to change your mind are few and far between.

Yeah. You need not only personal experience. Ideally it's personal experience. A lot of the people who think like we do now do it because they said, okay, who knows, maybe it's fun.

Character knows what he's talking about. Let me try some intermittent fasting and then give up carbs and see what happens. And they get healthier. And then it's like, oh well that was interesting.

Now I care or they try it on their patients and their patients get healthier. But if you're like, if I'm talking to a lean marathon running physician, it's like, I'm never going to get through it.

Yeah, it's like a wall. You know, they just can't break through that wall. I think the and the academics are even harder because they've got this, you know, they've got this idea that they're the smartest people in that subject in the well, and they've got all this feedback, right.

They've moved up in their careers, they've become professors, they got accolades. It's like, who wouldn't? You know, since we're running out of time, we should tie this back, right?

Yeah. So let's just say I think we're just over, actually. But let's let's just tie this back quickly to the longevity story and then and then wrap it up from there. Right.

So longevity you want to prevent delay chronic diseases. That's part of it as long as you can if not indefinitely. This alternative hypothesis, these chronic diseases are triggered in effect a predisposition.

It's a genome, you know, the genes triggered by the environment. The environment has changed. We've added sugar and refined grains. And if like I said, if you believe the seed oil hypothesis, sugar, refined process, grain seed oil, we know now several hundred clinical trials that if you remove the car, if you tell people don't eat the sugars and the grains and the starches, don't drink caloric beverages for the most part, which are inevitably sugar, you know, beer, then you'll get healthier.

We see that happening with people, and you can't. I mean, I don't believe you can. I can promise these people they'll live longer again.

## Diabetes Trials, Insulin, and Longevity

That was the scary thing about the diabetes experience with type one and insulin is, you know, you clearly wonder drug comes in, saves these kids lives.

And then 15 years later, either all hell breaks loose, you never know. But you could make a very good argument that if you want to, the first step to maximizing longevity is removing these foods from your diet.

I completely agree, because if you think about, you know, the two main causes of death, like by far and away in the United States, cardiovascular disease and cancer, both huge, very, very powerful links to both obesity and diabetes.

Diabetes much more strongly because of course, that's a much more serious health condition. But you see, like there's tons of data showing that there's obesity associated cancers, including extremely important ones such as breast cancer, colorectal cancer, pancreatic cancer.

So very, very strong link between obesity and cancer, very, very strong link between diabetes and cardiovascular disease. So clearly, since those are the two sort of dominant diseases of our day, right.

You know, 100 years ago we'd be talking about, I don't know, TB or something like that. Right. But you know, of, of this modern era, those are the things we have to focus on.

And, you know, I thank you for sort of bringing your hard science hard nosed coach to a lot of these fields because it's opened a lot of people's eyes and bleeding mine.

And, you know, I think that's, you know, thank you so much for being here. And thanks for having such a great conversation about this. I really enjoyed it. Again. Thank you Jason.

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