

# **The Tim Ferriss Show Transcripts**

## **Episode 50: Peter Attia**

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Tim Ferriss: Mr. Peter, tell me about your very, very boring and repetitive breakfast.

Peter Attia: It usually starts with nothing, and then I usually do a second course usually because I'm a little hungry and I'll have a little bit more nothing. I usually top it off with a bit of nothing.

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Tim Ferriss: This is Tim Ferriss, and welcome to another episode of the Tim Ferriss Show, special holiday edition. I am celebrating this December with some reflection looking back at what I've done this year or not done and reassessing: trying to figure out how to do things that are bigger, better, badder in the coming year. And one of the most impressive people that I'm contemplating, thinking of emulating, in this coming year is Peter Attia, MD. He is the guest for this episode.

And while I sip on my Guayusa tea, which is from the Amazon, maybe a shrub tree - I have no idea - but it's something that's going to get you stopped in customs and perhaps have your body cavities searched if it's in a big gallon, zip lock bag. Don't ask me how I know that. He is one of the few people I have on speed dial for advice, and Peter's a very impressive guy. He is the president and cofounder of the Nutrition Science Initiative, NuSI, which can be thought of as the Manhattan Project for nutrition. I'm and advisor to that group.

He cofounded it with Gary Taubes who wrote "Good Calories, Bad Calories." But prior to all of that, he has an incredible range of experience. He worked at McKinsey & Company. Before that he spent five years at Johns Hopkins Hospital as a general surgery resident, where he was the recipient of many different prestigious awards, and he also authored comprehensive reviews of general surgery. He spent two years at the NIH, the National Institutes of Health, as a surgical oncology fellow at the National Cancer Institute, and he earned many different degrees. I'm not going to go into all of them, but MD from Stanford, mechanical engineering and applied mathematics degrees.

The guy's a stud, and he is also an ultra-endurance athlete. So he puts theories into practice. He also does horrible, horrible things to himself in the name of self-experimentation - just like me - that we will get into in this interview. So without further ado and it does get a little into the weeds - it gets a little dense in a few areas - and I'm for you to just bear with it. There are lots of gems here, so please meet Peter Attia.

Tim Ferris: Peter, welcome to the show. Thank you for joining me on this afternoon.

Peter Attia: Thanks for having me, Tim.

Tim Ferris: I have thought about our original introduction. I think it had to be through Gary Taubes. Is that right? Was that the first contact?

Peter Attia: Yeah, I think Gary introduced us about two and a half years ago.

Tim Ferris: To give people a little bit of context because there are many things I'd love to delve into, not the least of which is your medical and scientific pedigree, but also your similar DNA. That is, I feel like we're cut from the same cloth that we have a certain lack of self-preservation in self-experimentation or at least interest in pushing things to kind of or very clearly obsessive degree. So I want to jump into both of those, but the project that first brought us together was NuSI. So maybe you could give people just a little bit of background on what NuSI is.

Of course, I ended up becoming an advisor, but the Manhattan Project for nutrition as it has been called maybe you can tell people a little bit about it.

Peter Attia: Yeah, it's exactly how we got introduced, Tim. It's basically an organization that Gary and I founded in 2012 with the idea of trying to do nutrition science at a level that currently isn't doable in the existing funding and political is the wrong word but sort of risk atmosphere of how nutrition science is done. So fundamentally we argue that - and I don't think you'll find much push back on this - there are two main impediments to doing exceptional work in nutrition science. The first is that while in aggregate there's a lot of money out there in terms of doling it out to specific studies, it's shockingly low, and secondly and perhaps more importantly there really isn't an appetite to ask questions that are directly counter to the conventional point of view.

And so we felt that there are in fact alternative hypothesis about the role of diet and disease that do need to be asked. They could be wrong. It's entirely likely or certainly plausible that these ideas that we have could be incorrect, but they deserve a shot. They're certainly not our ideas, right. I mean, we're just sort of champions of them. And if the worst thing that happens is you spend time and resources demonstrating that what's currently believed to be true is true, that's a pretty limited downside when you consider that the upside is if you demonstrate the opposite that some of these things in fact are incorrect, the payoff's enormous.

Tim Ferris: Right. I think that oftentimes perhaps people view being a scientist as something that is restricted to a very rarified echelon of highly trained people. And, granted, there are who are very well trained for science, but it's really on some level about finding hypotheses worth disproving and sort of asking the uncomfortable questions.

So I think political is an appropriate term in a lot of instances, and to frame it a different way, the reason I was attracted to NuSI is that many of the studies out there are either very poorly designed. They conflate correlation with causation. You see this all the time. They're misinterpreted by media, and then they're oftentimes funded by very, very biased parties who sometimes have a profit motive. So the idea that you could raise whatever the dollar amount was but you had your anchor backer was the Arnold family, if I'm not mistaken.

Peter Attia: That's correct, John and Laura Arnold.

Tim Ferris: I suppose his background is as an energy trader, I believe, but please correct me if I'm wrong.

Peter Attia: That's correct.

Tim Ferris: You have this money coming in that is independent, unbiased, to fund studies designed and conducted by an eclectic collection of the brightest scientists in the US and probably beyond, many of whom disagree with one another.

Maybe you can just comment on that because I think it's a really important point.

Peter Attia: Yeah, when Gary and I started talking about this idea back in probably the spring to summer of 2011, we realized that we actually both had a pretty similar point of view on what the likely dietary triggers of metabolic disease were. In other words, why is it

that in one generation or certainly two generations we've seen an epidemic of disease that previously didn't really exist? We also agreed that the current state of the evidence - that which we found most compelling - was technically not really sufficient enough to change the rules of the game, to change the way medical advocacy groups and physicians and even the government would suggest people go about eating.

So we thought that what was missing was science, but to your point to get a whole bunch of people that agree with you that share your point of view to do the work is not a powerful way to go about doing it.

Part of the opportunity here is working with people who have different points of view about things. Not just because that's allowing you to draw from a broader pool, you're now actually access everybody as opposed to a subset, but because a really good scientist at the end of the day is defined by several things. But one of them is when confronted with conflicting data, they face it as opposed to sort of coming up with what we call ad hoc hypotheses, which are bolt-on hypothesis meant to support the original hypotheses in the face of evidence contrary.

Tim Ferris:

I want to give people perhaps a snapshot on your background because I think science can be intimidating, but ultimately if you look at a lot of the, say, amateur scientists throughout history, oftentimes the motives were very personal. So they lived in a place where smallpox was an epidemic, and they noticed that milkmaids didn't get smallpox.

And they hypothesized they could take sort puss or whatever from cows and inject their own families - I mean, these crazy fucking ideas that ended up having a high degree of validity. You earned your MD from Stanford. You have a mechanical engineering in applied mathematics degree from Queens University, and you have McKinsey. You have John Hopkins Hospital as a surgery resident, lots of awards and so on but - and we can dig into that a little further - what are your obsessions from a performance standpoint? And I was half joking earlier when I said lack of self-preservation, but it is kind of literal in the sense that it's possible to try to optimize health to the point where it's in your best interest to just kind of sit in a metal box and absolve yourself of interacting with anything in life.

And I think that you maximize your performance at the same time. So what are some of your obsessions in that realm at the moment or interests?

Peter Attia:

Well, growing up, Tim you know I grew up in Canada. So, obviously, hockey was sort of the most important sport of any good Canadian kid growing up, but actually pretty early on around the age of 13 my interest actually shifted towards boxing and martial arts. And that became really the focus of my life, and I never really did it in moderation. So even in high school I was sort of training six hours a day very, very hard. Even though in amateur boxing it's only three rounds, I was always thinking about the next step, which was being a professional. And, of course, at the time that's 12 rounds of boxing, so everything I did was geared towards. I had to run ten to fifteen miles in the morning not just four. I had to jump rope for 30 minutes not just 15 and had to spend this many hours sparring each day.

And so my foray into my care about the body's performance always came through the lens of performance. So it was how does the way I train or how does the way I eat impact my performance initially in a boxing ring? Now, at the time it was highly crude. In fact, I suffered from the issue that I'm sure a lot of 14-year-old boys suffered from, which we'd all kill to have that problem again, which is actually couldn't gain weight. I started my career at 127 pounds. By the time I was 16, I was a solid middle weight, which is 160 pounds. But as you may know from your experience, most people lived ten pounds above their weight class and then come down to it. But I was only four percent body fat, so I actually lived and fought at about 158 pounds. And to keep that weight on, I would eat about 6 to 7 thousand calories a day.

Just to give you an example of lunch because it was the one meal I can really remember, it was an entire loaf of bread, which is 15 pieces of bread. So that was seven sandwiches with a two-liter jug of orange juice, and then at the cafeteria I would buy a plate of French fries and like some other nastiness. And like that was lunch every day in high school, and yet I had a 27-inch waist and no fat on me. In part not just because I was exercising six hours a day, I think more importantly because we're very metabolically different when we're 14-year-old boys than when we're 40-year-old boys. So if you fast forward I don't know how many years, athletic stuff has always been important to me. The sport has shifted. By the time I was in my early 20s, the obsession switched away from boxing into other things, and more recently in me 30s the

obsession became swimming - ultra, ultra-long distance swimming.

Tim Ferris: How long is ultra-long distance swimming?

Peter Attia: Yeah, it's kind of a fuzzy definition. I think most people define ultra-long as anything over 16 miles, but I think that's somewhat arbitrary. It's sort of like one of those things like you know it when you see it. Like, is this one mile river swim –

[Crosstalk]

Tim Ferris: It's pornography.

Peter Attia: Yeah, exactly, right? Is this one mile swim across the river ultra-long? Not really. Is that 25-mile swim long? Yeah, that's ultra-long.

Tim Ferris: What's the longest swim that you've done?

Peter Attia: About 25 miles.

Tim Ferris: That is a long swim.

Peter Attia: So in my 30s - and this is now a different chapter in my life - obviously I'm not in school. I think at the time I'm working at McKinsey & Company in San Francisco. I'm still managing to spend an average of four hours - three to four hours - every day swimming because it's not linear. I spend eight hours a day on the weekends and then maybe only an hour and a half to two hours a day Monday through Friday, but I'm obviously burning a lot of matches.

And yet interestingly, my weight is getting higher and higher and higher. And I went from sort of being 170 pounds to 205 pounds, and the composition of that weight wasn't what I wanted. It wasn't like I was gaining all this muscle. I was gaining fat, and the blood tests showed that I was basically pre-diabetic. So all of the sudden the dietary strategy –

[Crosstalk]

Tim Ferris: What are the indicators that you looked at?

Peter Attia: You do something called an oral glucose tolerance test, which they draw your blood and then you drink this horrible, nasty drink of glucose. And then they measure your insulin and glucose levels an hour later and then again two hours later coupled with other

standard blood tests like your triglycerides and something called a hemoglobin A1C, which measures the amount of blood sugar that's basically sticking to your red blood cells.

Tim Ferris: Is it fair to say hemoglobin A1C is sort of a running three-month average of your fasting glucose, or is that completely scientifically off?

Peter Attia: No, it's actually pretty close. It's not fasting. It's basically a three month running average of your aggregate glucose level.

Tim Ferris: Aggregate glucose, got it. Okay, cool, not to interrupt.

Peter Attia: Yeah, so anyway, basically, all of these tests were pointing in the wrong direction. I had something called metabolic syndrome, and, again, I think there's a lot of people that find themselves in that situation. To your question about what's the personal motivation, I think what pissed me off was - and I remember saying this to my wife - I said, "You know what pisses me off is I'm working too hard to be in this situation." It's one thing if you're sitting on the couch eating Doritos all day long, but my diet was actually much cleaner as a 35-year-old than that French fry, sandwich eating kid in high school. Obviously, it still wasn't the right diet, but the point is I was busting my ass to be fit and healthy and watch what I eat.

And, you know, frankly I just got aggravated beyond words. We joke about it now, but at the time I literally said to my wife, "I'm going to go get a gastric bypass." And she was like, "You are the most ridiculous human being that's ever lived. We're going to literally have to talk about our marriage if that's what you're considering at the weight of 205 pounds." I actually did go and see the top bariatrician in the city of San Diego, and it's kind of weird story because even though I was like obviously overweight, I was the thinnest person in the waiting room by a long shot, right. And it sort of put in perspective, like, "Peter, you think you've got problems. I mean, these people each weigh 400 pounds." And when I went up when it was my turn to go and see the doctor, the nurse took me up to the scale and weighed me. We got on the scale, and I'm like 210. And she's like, "Ah, this is fantastic. Like, are you here for a follow up?"

I'm like, "No, I'm here for," and so it was a real eye-opening experience, Tim, because frankly throughout my entire medical training, which was in surgery and then again in surgical oncology, which is cancer surgery, I had never paid attention to this problem

- never. If it didn't have to do with cancer, if it didn't have to do hepatobiliary surgery, I didn't care.

Tim Ferris: Orthorexia is used as a derogatory term, but I think you're very meticulous in your own testing. And perhaps even separate from NuSI, but you've introduced me to quite a few interesting tools or concepts, for instance, the idea of synthetic ketones. And maybe you could just comment on that as a taster for people, although taste might not be the way to put it. You can explain that, but this was an eye-opener for me.

And I remember hanging out with you having dinner not too long ago where you specked out sort of the chemical structure of beta hydroxybutyrate and a number of other ketone - I guess they'd be salts - right?

Peter Attia: They're actually salts or esters.

Tim Ferris: Or esters, right. But what are synthetic ketones, and why might people care about them?

Peter Attia: Well, I think that to explain it I probably have to spend a minute explaining what ketones are biologically or what we call endogenous ketones. So if you think back to what our ancestors were doing up until a few hundred years ago or certainly a few thousand years ago, we were basically often going 24 hours or longer without food. That was just the nature of how things worked, right. When you're in the hunter gatherer mindset, that's your life. The human body has only really evolved to store a finite amount of glucose, and there's only two places we store glucose. One is in the liver. One is in the muscles.

And it's only that stash in the liver that's accessible by the brain because the glucose that gets stored in the muscle can't leave the muscle. It circulates within the muscle. So we this organ - the brain - which weights maybe two percent of our overall bodyweight but probably accounts for 20 percent of our body's metabolic demand, and on top of that it ordinarily functions exclusively on glucose. And so you have this problem, which is you have an organism that is wildly dependent on glucose, and we can only store a fraction of what we need. We can only store about one day's worth.

Tim Ferris: About 400 grams, like 1600 calories?



Peter Attia: It really depends on the size of the person, but, yeah, that's probably about right for average. And remember most of that, by the way, it not accessible to the whole body.

Tim Ferris: Right.

Peter Attia: So the trick that we evolved was rather than make glucose out of protein, which is a pretty easy thing to do, the problem with that is if you want to make glucose out of protein you have to break down muscle.

And the last thing you want to do when you're out there trying to find your next meal is lose muscle at the expense of getting glucose for your brain. So what if there was a way we could get the brain to use fat, right? That's the problem that needed to be solved, and the solution was a beautiful one, which is we can break down fat of which even the leanest hunter gatherer had days and days if not months of fat on their body. What if you could break that fat down in the liver into another type of molecule distinct from glucose that the brain specifically could actually utilize as fuel.

And that's where ketones enter, and so what our bodies do when prolonged fasting occurs - and by prolonged I really mean it even begins at 24 hours of fasting - is we start breaking down our own sources of fat. We start making this thing you referred to beta hydroxybutyrate. Not to get too geeky on it, but beta hydroxybutyrate and another member of that family called acetoacetate, they exist in an equilibrium.

And these things get shuttled into the Krebs cycle, which I think your readers will be familiar with, and it basically becomes another substrate for making ATP. And George Cahill who is sort of a luminary in this field - passed away a few years ago - but George Cahill is one of the sort of leading godfathers in metabolism at Harvard University. He did some legendary experiments in the '50s and '60s where they had subjects that they would starve seven to 14 days and just measure glucose levels and ketone levels. And you'd think that after 14 days of not eating a person would be mentally foggy, not well, and it turned out it was just the opposite. After a couple day lull, and you know this personally Tim because you've done these long fasts, after a couple days of hell it's actually the reverse. You short of get sharp.

Tim Ferris: You feel amazing.

Peter Attia: Yeah, you feel unbelievable. And what Cahill showed was what fraction of the brain's energy was coming from those ketones. So that's relevant. That's starving, but look.

Outside of the odd, "Let's do a one-week-a-year fast," sort of thing, how does that play into something beyond that? Well, the other way you achieve ketosis though not to the same extreme is through something called nutritional ketosis, which is restricting the one dietary component primarily that restricts ketone formation and keeping at a minimum the other one that also restricts it. And those are carbohydrates and proteins respectively. And so if you eat a diet that has very little carbohydrate in it and only a modest amount of protein and the rest of it of course made up from fat, you can also generate ketones.

Now to your question, it turns out that you can drink or consume in some fashion - but they're all typically liquid - you can drink these ketone molecules directly. And that's what we call these exogenous or supplemental ketones, and they come in multiple different forms. They basically exist as a beta hydroxybutyrate ester, a beta hydroxybutyrate salt, and an acetoacetate diester. And I've tried all of these things, and I can safely say -

Tim Ferriss: Why don't tell people? Why don't you recount your first experience consuming these?

Peter Attia: Yes, so the first one I tried was the beta hydroxybutyrate ester, which a very good friend of mine sent me, and I had been told these things taste horrible. I had talked to two people who had consumed them before, and these are stoic dudes. Like, this isn't like a six-year-old kid. This is like stoic, military dudes who said, "Oh, man, that's the worst tasting stuff on earth." And so I knew that, but I think that piece of information was sort of like fleeting in the excitement when the box came. And so I tear open the box, and also there was a note in there that explained a somewhat palatable cocktail that you could mix - like how you could mix this with ten other things. And I just disregarded that, and I just took out like the 50 mL flask.

And I chugged it, and I remember it was like 6:00 in the morning because my wife was still sleeping. And all these thoughts go through your mind. So first all, you drink it, and it tasted like how I imagine jet fuel or diesel would taste. If you've ever smelled distillate, it's this horrible odor, and you can sort of imagine what it would taste like. This is what it tasted like, and so my first thought was, "God damn, like, what if I go blind? What if there's

like methanol in here? Like, what did I just do?” And then my next thought was just, “Oh my god, you’re gagging.

I mean, you’re really gagging. If you puke this stuff up, you’re gonna have to lick up your puke and this stuff. It’s just gonna be a disaster.” And so I’m like retching and gagging and like trying not to wake up the family and trying not to like spew my ketone esters all over the kitchen. And it took like 20 minutes for me to get out and do my bike ride, which was the whole purpose of that experiment.

Tim Ferris: Must have been a record setter.

Peter Attia: Oh, god, those things are unbearable. So until they figure out a way –

Tim Ferris: No, just to put that in perspective. I’m speculating here, but correct me if I’m wrong. I would imagine much like myself you’ve made a somewhat secondary career of choking down foul tasting shit, like gels on ryes or like all these protein powders, like the most hideous, ostensibly performance enhancing sports supplements. You get to a point where you’re like, “I’ve had bad stuff. How bad could it really be?”

Peter Attia: Yeah, I’m a generally insensitive person to bad taste. This took it to another level, and the funny thing is I’m such a glutton for punishment. I had the guy send me like half a liter of it after because I was determined to make it work. Like, I got to figure it out, and I would like try mixing it with this and mixing it with that.

And also I wanted people to try it with me. Any time I had a friend over, I’d make him dip his pinkie in to at least try like a sliver of it. And even some of the most stoic people I know were like, “Wow, you couldn’t design something to taste that bad.” So the good news is the acetoacetate diester actually can be put into a capsule. So if you’re willing to take 20 capsules in one sitting - each one the size of a horse pill - you can trade one problem for another. So you get rid of the taste, but then you have to gag down 20 capsules, which also tends to induce the desire to vomit. So I went down that path for a while and then really settled in on the beta hydroxybutyrate salts. To this day though I don’t use them that often because they’re pretty expensive and we’re not at a point in time yet where we have a great like supply of them.

Tim Ferris: What are the potential benefits or advantages of consuming these synthetic ketones?

Peter Attia: Well, the benefit that interested me the most, which came out of the research done by a guy named Richard Veech at NIH –

Tim Ferris: How do you spell Veech?

Peter Attia: V-E-E-C-H. Now it turns out, you may remember a moment ago I mentioned George Cahill at Harvard who is like a god, I never met George Cahill, but I've met Richard Veech. And Richard Veech actually did his post doc with Cahill, so he came from that sort of lineage of uber smart folks. And Veech showed in animal models that when you switched the substrate from glucose or even fatty acid to ketones you could generate more ATP - more mechanical work - for less oxygen. The difference was enormous in the animal models.

I also saw some unpublished data that looked at this in athletes - human athletes that is - and it suggested that there was a difference. So one of the things I wanted to do is do an experiment, which was, look, for a certain athletic event there may be an advantage to being able to consume less oxygen for a fixed power output. And that may not be true for all athletic events. Would that benefit a basketball player or a tennis player where you're stopping and starting and it's more explosive? I'm not sure, but I think it would address someone doing a long, steady state subthreshold effort.

Tim Ferris: Now subthreshold you mean below their anaerobic threshold.

Peter Attia: Correct. Below ventilatory and anaerobic threshold. So when we do marathon stuff, we refer to all day pace, which is typically about 65 percent of your VO2 max is a space that once you get really well tuned, you can probably hold that pace for 10 to 12 hours.

Tim Ferris: Got it. Persistence hunter speed.

Peter Attia: Yeah.

Tim Ferris: Got it. Cool. Now ketosis is a really fascinating state, and we don't have to delve too far. Well, maybe to address a common point of confusion, people sometimes fear ketosis thinking of it as ketoacidosis, as observed in some people, diabetes. They're not the same thing as I understand it.

Peter Attia: That's correct. They're related in the way that a house fire and a fireplace are related. So when a house is on fire, there's fire

everywhere, and when you have a fireplace on, there's fire in there. So they have an association, but that's where it ends. Whereas in the fire place is like a well-controlled thing that actually adds some value, whether it be light or heat, the house fire is obviously not a controlled thing.

And therefore it's not a positive thing. It's a destructive thing. So ketoacidosis is indeed a very destructive phenomenon, and it is a situation that rises almost exclusively in people with Type 1 diabetes. Now I've read some really interesting case reports of people who are not Type 1 diabetic who have had ketoacidosis, but these examples of that are so rare that I think it's almost confusing to talk about them. So for the purpose of most people, ketoacidosis is a state that occurs usually when someone with Type 1 diabetes gets sick. There's usually a precipitating event, and what basically happens is a deficiency of insulin because a Type 1 diabetic requires exogenous insulin.

They have to be injecting themselves with insulin. So you have a miss match between the need for insulin and glucose. And what's basically happening is in the absence of insulin, which actually suppresses ketones, their ketone levels rise.

And once the ketones get kind of north of 15, 1-5 millimolars, and certainly any higher - 20 to 25 millimolars - you basically get into an acid based problem where the pH now starts to drop. So you get what's called metabolic acidosis in response to that. I feel like I must have taken care of 100 people with ketoacidosis when I was back in the emergency room. It's a life threatening illness, but the good news it's a very simple treatment. You give them massive amounts of IV fluid, glucose, insulin, potassium, and they're usually better within two days. Now ketosis, totally different.

Someone who has a normal working pancreas can't really generate ketones north of - I've heard of the odd person who can get up to 9 or 10 millimolars - but for most people even if they starved themselves for two weeks and certainly in Cahill's subjects over - gosh, I think his highest - 40 days in the longest Cahill cohort, they plateau at 6 millimolars.

Which is still a very high level, but it's nowhere near the level that produces acidosis. And that's because there's an auto regulatory feedback from insulin.

Tim Ferris:

I plateaued personally about six 6 millimolars when I was doing my seven day fast. So just to add something to what you said, for

those people listening if you go on the internet and you're researching ketosis and you see someone say, "Ketosis is very dangerous," and they link to an article, make sure that they are referring to nutritional ketosis not ketoacidosis. It's very commonly mixed up. The other tip I wanted to offer people who may not be very familiar with scientific terminology is a good mnemonic for remembering the difference between exogenous and endogenous is thinking of an exoskeleton on an insect, where their skeleton is on the outside. So "exo" typically means administered from the outside. You're not producing it in your own body, so endogenous versus exogenous.

I was hoping Peter that you could describe for people listening an experiment that you did which highlights your wonderful obsession and the metabolic chambers. And I thought if you wouldn't mind just briefly describing sort of the standardized experimentation that you did in the metabolic chambers. Most people don't even know probably what these chambers are or what they're used for, but I'd love to hear you describe that if you wouldn't mind.

Peter Attia:

So I guess going back to NuSI, one of the types of experiments that Gary and I felt were really essential to be funded were the types of experiments that examined what we think is perhaps one of the most important questions in all obesity, which is, "Is obesity simply a disorder where people consume too many calories, or does the type of calorie they consume play just as much a role?" In other words, there's no dispute that someone who's gaining weight is eating more calories than they're expending.

And we have the first law of thermodynamics to thank for that wonderful fact. It's just not particularly interesting. It's sort of like saying, "Hey, Tim, do you know why Bill Gates is so rich? He's so rich because he makes more money than he spends." Like, thanks, right?

Tim Ferriss:

Biography complete, right.

Peter Attia:

I'm Peter Greggs. Thanks for watching Biography. So it really irks me when we talk about obesity through the lens of it's an eating disorder. People eat more than they expend. Like, yeah, no shit, Sherlock. What I really want to know is why do they eat more than they expend. What is it that's driving that? And so one of the hypothesis is the types of calories we consume could actually be feeding back on the two systems that regulate what we eat and

what we expend being your appetite. In other words, are there some foods that make you want to eat more?

And secondly your - what we call - non-deliberate energy expenditure, meaning how much energy do you expend? So to your question about the chambers –

Tim Ferris: Is that the same as resting metabolic rate?

Peter Attia: Yeah, there's a little bit of confusion between all of the restings and basal metabolic rates, but I think the easiest way to describe it - that's why I use the term non-deliberate energy expenditure - it's sort of like, okay, you can go out for a run and burn 500 calories. And you can climb a flight of stairs and burn four calories, but what I really care about is - and what we know from studying people for forever is - the majority of the calories that the average person is burning is the calories they're burning when they're doing nothing. It's when you're sleeping, when you're sitting down, and so we measure that in a way. And we can also measure the total energy expenditure. So the question is can a question in macronutrient composition alter your appetite and your energy expenditure? Now, measuring appetite, we can talk about that. We could spend two hours just talking about how you measure appetite and appetitive behavior.

We'll save that for another day. On the energy expenditure side, there's really two ways to measure energy expenditure.

Tim Ferris: Sorry to interrupt, Peter. Just for those people who may not be deep in the nutritional world, so macronutrients - and, again, feel free to correct me, Peter, at any point - but you're talking about the distinction between protein, carbohydrate and fat principally. Is that correct?

Peter Attia: Correct. That's correct. I'm glad you brought that up. The reason we're asking that question is not because we think that that's the perfect vision amongst nutrients. I mean, there's a million different ways to distinguish between a piece of broccoli and a piece of bread, and even the macronutrient composition isn't the best one there. But what we're really interested in is that we have a pretty good understanding of how macro nutrients impact hormones and enzymes in the body and how those enzymes may regulate fat cell accumulation - the accumulation of fat within fat cells, adipose sites.

So this business of measuring energy expenditure is really tricky. And the two ways to do it, I've participated in both of those. One is a measure using doubly labeled water, and another one is a technique called indirect calorimetry. So anybody who's had a VO<sub>2</sub> max test has actually done indirect calorimetry. So if they've been on that treadmill or on the rowing machine or whatever, they've got that mask on them. It's actually sampling the amount of oxygen that they're using and the amount of CO<sub>2</sub> that they're generating. And some of your listeners may know that there's a ratio between those two. So the ratio of the CO<sub>2</sub> that you expend, which is called VC0<sub>2</sub> - the ventilatory CO<sub>2</sub> - over the ratio of oxygen consumed is called RQ - respiratory quotient, and there's a mathematical relationship that ties all of those into the amount of energy you're consuming.

So that's why when somebody does a treadmill test with one of those masks, we can tell two really exciting things about them. One is how much energy they use for that given task and, perhaps as interestingly if not more interestingly, where they got that energy from. Did they break down glucose, or did they use fat? So what if you want to do this for a long period of time, and you don't want to stick a mask on somebody? So then what you do is they build these rooms, these super-duper, NASA, airtight rooms with double-layered, gas-sealed doors and window and everything.

And the room has thousands of air sensors in it that sample frequently - generally about every 15 seconds - the concentration of CO<sub>2</sub> and O<sub>2</sub> in the room, and they then calculate on a minute by minute or second by second basis how much oxygen you're consuming as the subject in that room and how much CO<sub>2</sub> you're producing. And you'll typically spend 24 hours to 48 hours at a time in these lockdown rooms.

Of course, they're handing food to you through a doubly sealed door so even during the transfer of food or body fluids you're not contaminating the air in the room. And there's an engineering corps outside the room that's just getting all this data, and when the day is done, you can tell exactly how many calories you burned that day and where you got that energy from. Did you burn fat stores or glucose stores?

Tim Ferris:

You repeated an experiment in a number of these chambers, and you standardized the experiment where you were consuming exactly the same meals, performing exactly the same exercise routines. What were some interesting results or unusual data that came out of those experiments?



Peter Attia:

Well, first of all, the reason I wanted to do this was because one of the first studies that NuSI funded was relying on these sorts of chambers across the country and so at four different sites actually.

There aren't many of these things. I think there's only like maybe 15, 16 of these chambers in the United States. I mean, they're very expensive. It's a pretty costly piece of equipment. What we were basically asking subjects to go through because in these experiments we were asking subjects to spend two days a week inside a chamber for several months. So I wanted to at least get a sense of what that would feel like. I mean, does that drive you crazy? What's that like? The second thing I wanted to get a sense of was, as you alluded to, how reproducible are the results from once center to another - from one site to a site? And then of course there's just always this sort of morbid curiosity of what will we learn? I'm curious as to how these things change. I have no idea how much energy I expend in a day, nor do I have an understanding of how much of that is basal versus activity related.

And so I did this across three separate stints, and you were absolutely correct. So it was an exact, finite meal. Now I realized after the first day we did, we underdid the meal. In an ideal world, you match your intake to your expenditure. So a priori we had designed what the experiment was going to look like to try to replicate my life outside of a chamber. So down to the minute we knew everything that was going to happen. So I was going to go to sleep at this hour. I was going to wake up at this hour.

I was going to do this. I was going to get on the bike - the stationary bike - and ride it this many watts because it had a power meter. I was going to do this many pushups, this many plyometrics, this many sit-ups, eat the meal at this time, work on the computer for this time. So we did it the exact same way each time. And after the first day it was clear that we had only given me 3,000 calories, but my energy expenditure was about 4,300 or 4,400 kilocalories.

So I had a 1,400 calorie deficit, and, of course, I wanted to fix that the next time. But I was like, you know what? I'd rather we just stay reproducible and do it. So one of the things that I found interesting was how high my metabolic rate was at rest. So to do this in a chamber, you basically spend two hours wide awake doing nothing, laying down. And that produces a slightly higher metabolic rate than when you're sleeping, but it's the closest thing we have to a true resting energy expenditure.

Tim Ferris: Are you reading, or are you just staring at the ceiling for two hours?

Peter Attia: No, no. If drinking those ketones was the worst thing I've ever done and then almost dying getting an insulin suppression test was the second worst thing I've ever done, the third worst thing I've ever done is having to spend those two hour blocks doing nothing because you're not allowed to do anything. You can't sleep. If you even close your eyes, the dude is knocking at the door. You can't watch TV.  
You can't talk on the phone. You can't even check your phone if it beeps. I mean, it was really painful.

Tim Ferris: Okay, just had to ask because talk about monkey mind.

Peter Attia: Oh, yeah. No, it was crazy. So, yeah, it was interesting. It gave you a sense of how useless some of the tables are that we use to typically try to understand metabolic behavior. So if you put all of my statistics into a table - right - so: I'm this tall; I weigh this much; I'm this old; I'm a guy; I exercise this much; I have this percent body fat. If you put all of my data into a table and I've done this with the most elaborate regression models that exist, they all basically say my resting energy expenditure is somewhere between 1,720 and 1,760 calories per day.

Tim Ferris: That's a pretty narrow range.

Peter Attia: Yeah, it is. Yeah, yeah.

Tim Ferris: And in fact in the chambers?

Peter Attia: And in fact in the chambers it turned out to be between 2,100 and 2,250 on three occasions.

So I am definitely burning 400 calories a day more than anybody would predict, and I would argue similarly that I take care of patients in my medical practice in whom their metabolic rate is infinitely slower than what is predicted by those tables. And so I think tables are directionally probably reasonable, but at the end of the day, you realize that the individual variation among people is significant. And, of course, one of my questions is how much of that is macronutrient dependent?

Tim Ferris: Right. It raises a question. You may have thought about this. You probably have thought about it before, but looking at say blood

ranges - the normal range for various biomarkers when you have blood tests performed, whether that be hemoglobin A1C or total cholesterol or anything else - and how those numbers change over time.

And what I've just become so fascinated by is looking at how difficult it is to find someone who is living hard - living and playing hard, whether they're an athlete or just someone who's a type A personality CEO type - who isn't constantly out of range on at least a handful of things. And the questions that I've started to ask are if we're not able to get the nutrition we need ostensibly as reflected in these tests, how the hell did our ancient predecessors ever pull it off, number one? And does that in fact mean that they didn't, and it's a flaw in the test. Or is there rather than not getting enough of something is modern man exposed to various types of contaminants or estrogenic compounds in shampoo or who the hell knows that create those deficiencies?

It's not that we're not getting enough. It's that there are things that are causing deficiencies. Or, again, just the tests are influenced by lobbying and all these other things, and they're just not reliable. How do you even start to think about that stuff because thankfully there are doctors I work with who can look at the tests and say, "This is slightly out of range. I wouldn't worry about it at all for these following reasons." But for people who go to a doctor once a year or get an annual checkup and get prescribed drugs for anything that's out of range just based on that one snapshot, I mean, how do you even start to think about this stuff? I know that's a long question, but it's something that's been bothering recently.

Peter Attia:

Yeah, it's something I think a lot about. I became most interested in this a couple of years ago. Well, to back up for a second, I have my own centrifuge and blood draw kit at home and stuff like that because I'm doing blood work a lot on myself and on anyone who is unlucky enough to walk in the front door.

But one of the things I noticed was just the huge variability in my lipoproteins. These are the molecules that carry around cholesterol and triglycerides in our body. Conventional thinking would be, hey, if you're not taking any drugs and/or your diet's not really changing much, why would those things vary? And yet I had a dental abscess I was sort of ignoring because I was too busy and it was sort of getting pretty bad, and eventually just had to get the tooth yanked out. And I noticed that the changes in my lipoproteins were enormous.

And so I started realizing, hey, obviously inflammation absent any other change is changing these things. And then I got this idea which was I wonder how much exercise is impacting it. So I did an experiment with the help of actually the lab that does all the acae because this was such an exhaustive thing. I couldn't do it on my own.

So basically I did super comprehensive like ten-tube blood draws on myself in three days but sandwiched around similar activities. So I would do an immediate morning fasting one. Then I would do an immediate hard work out then immediately do one after and then immediately do one three hours later. And there are some things that you should expect to vary a lot due to that kind of variation. You should expect your glucose levels to change, probably your triglycerides, probably your insulin levels, right.

There are other things that you'd think, "Wow, if they change and we're prescribing medication to people based on those such as CRP, LDL cholesterol, LDL particle number, these sorts of things, we maybe need to ask ourselves a question." So sure enough the changes were amazing. I mean, I couldn't believe what a hard workout would do - how much crappier it made you look on paper.

The first time I ever did this, actually, Tim was in 2005 when I swam from Catalina Island to LA. And I had my friend Mark Lewis, who's an anesthesiologist, draw my blood like ten minutes before I got in the water on Catalina Island and then ten minutes after I got out of the water in LA ten and a half hours later. And it was a real epiphany for me because I had developed something called systemic inflammatory response syndrome, SIRS, which is something that we typically see in hospitalized patients who have horrible infections or who have been in really bad trauma: gunshot, car accident, that sort of thing.

So my platelets went from a normal level to six times normal. My white blood cell count went from normal to sort of - I don't know - five times normal. All of these huge changes occurred in my blood that you wouldn't distinguish me from someone who had just been shot.

And in many ways, you could argue, look, that's sort of what's swimming for 10 and a half hours is, right, and maybe the changes we're seeing are due to these things. So I guess I would say this. I've always been hesitant to treat a patient for any snapshot, no matter how bad it looks, because I don't know. For example, I saw

a guy recently who on his morning cortisol level, it was like five times the normal level. So you might think, wow, this guy's got an adrenal tumor, right? But a little follow-up question realized that at 3:00 that morning, a few hours before this blood draw, the water heater blew up in his house. So the normal level of morning cortisol assumes a guy sleeps through the night now that he has to de-flood his house.

[Crosstalk]

Tim Ferris: Deal with a household emergency.

Peter Attia: Exactly, right? So, again, a silly example but I do think the sort of big picture matters a little bit more than the immediate value sitting in front of you.

Now to your first question Tim, I don't know that the values we deem normal have any bearing on what our ancestors would have looked like. I mean, I think this is a knowable question. I just don't know the answer. I do believe that there's somebody out there that could tell you because there's a handful of aboriginal populations that I think still exist in Australia, for example, where we can get a pretty good idea of what our ancestors probably look like. And I would be interested to know how far outside of quote-unquote normal are they.

Tim Ferris: Definitely. And it's not to say that perhaps the paleo ideal is not the target we should shoot for, but it makes me wonder, if you were in Iceland and you're getting four hours of daylight for three to four months, do you really have the same vitamin D level as someone who's like equatorial?

I don't know, but probably not I would guess. And does it really matter if that's your ethnic background? You know, the identification of uncontaminated indigenous populations has been of interest to me recently because of the Lyme disease and the long term antibiotic use and trying to research fecal matter transplants - shit swaps as they're scientifically known - and realizing how hard it is to find a population that has not been chronically administered antibiotics, whether in childhood or in adulthood. In Tanzania apparently there are a handful of places where researchers are flocking, and there aren't that many.

Peter Attia: That's really interesting. I had not thought of that.

Tim Ferris: It's fascinating. And I was actually going to do a fecal matter transplant.

As a side note, you can do the reverse enema, but there are people now who are also creating capsules that are frozen. I think they're typically frozen and then swallowed. The reason I decided against doing an FMT as they call them is that I spoke with a gastroenterologist who felt like if you were to create a spreadsheet of our known risks and the known potential benefits, you might decide to do a fecal matter transplant. But how many pathogens, how many different types of communicable diseases have we not identified that might be transmitted in a fecal matter transplant.

And their hypothesis was probably a lot. I mean, we think we've figured out things like hepatitis, but god knows what else is in there. So if you don't have to do it, if you're not dying of Crohn's disease or who knows, if it's a total optional thing like of like you and your bariatric band, then probably not a good idea to do a fecal matter transplant.

But this leads me just as it relates to the blood testing - oh, just as a side note, I'm not a doctor. I don't play one on the internet, but maybe Peter you could share your thoughts on this and just as a cautionary note. I've so many friends here in the US and elsewhere who, again, they have one or two checkups a year. If anything's out of range, they're prescribed medication, and the doctor will do their best. And they're constrained by a lot of things, but they'll do their best to trend.

And they'll look at previous blood tests, and what astonishes me is that the day of the week and the time of the day of the draw is not standardized. So, for instance, I have friends - male friends - who are now prescription testosterone. They've been given supplemental testosterone because their testosterone was 200 points lower than a previous test.

And if I look at their blood tests - not as a doctor just as a friend, obviously, out of curiosity - I'm like, "Well, wait a second. You had one test at 8:00 in the morning and another one at 11:30." And when I've done my own - I've done so many different blood tests - there's a very clear peak that then drops off really quickly for me depending on the time of the day. And certainly like your friend with this his water heater disaster, if you do a test on say a Thursday after three or four days of no drinking and then a test on Monday morning after a weekend of binge drinking, your values are inevitably going to be kind of all over the place. So just the importance of standardizing time, I think, for any type of trending is really important for people to keep in mind.

Peter Attia: Completely agree.

Tim Ferris: So, Peter, you sent me something very interesting a couple of days ago and the email that turned into a manifesto.

You sent me a 20-some page document that I do think you should turn into something about your thinking on basically hacking lifespan and performance and how to choose the appropriate blend of the two for yourself. I was hoping maybe you could comment on perhaps the common misconceptions or just sloppy thinking around life extension if we could start there.

Peter Attia: Yeah, so I don't know what, why, it occurs, but I guess once we hit our 40s where I'm comfortable now and maybe it's having kids - I don't know - but something sort of changes where we become a little bit more interested in longevity than we do performance. I think my days of sort of trying to be the best at anything are long behind me.

I do still compete in at least one sport competitively. I do sort of at least with myself give a damn about how I do, but the reality of it is nobody actually cares. Right, like, it's not like Pinarello is sponsoring me, and if I don't have a good race this weekend, they're going to drop me. Like, nobody cares. I don't think my wife could tell you within like 200 percent my times.

Tim Ferris: This is for the 20-kilometer time trial?

Peter Attia: Yeah, exactly. I mean, it's just like who cares, right? Nobody cared except me. But this was all catalyzed as you said through an email that a friend sent me over the summer. So it was a good friend of mine who's probably 50 years old, super-duper stud in all manners of life, very, very successful guy, does iron man, half iron man all the time. And I think he had just done sort of like his 20th half iron man or something like that.

And he said, "You know, Peter, I feel like I'm 50 now. I don't know that I need to do these anymore. I really want to start thinking about how I should shift my exercise towards increasing my longevity as opposed to fixating on performance of an athletic event." And so I responded to that email with a very long email, and that email - thanks to Evernote - turned into basically a collection of all of my thoughts on this. And that's actually what I sent you the other day because I knew if anybody would like it you would get a kick out of it.

Tim Ferris: Loved it.

Peter Attia: Yeah, so it's really been an obsession of mine for a long period of time. I think it's just crystalized into something that's getting more deliberate focus, which is how do you balance the desire to live longer - to live more years on this earth - with the desire to enjoy them and perform well? Because you can come up with all of these sort of thought experiments that are idiotic.

And I include one in this piece, which is if someone said to me, "Peter, you can live to be 150 years old in perfect health, but you have to sit in a dark room, alone, and never see your family again." Would you do it? I mean, not a chance, right? Like, I'd rather die in five years than have to do that.

Tim Ferris: There's also been some evidence in nematodes - I'm not sure in mice models or anything - that show that avoiding ejaculation can extend your lifespan - similar idea.

Peter Attia: Well, we know castration can.

Tim Ferris: Oh, fantastic.

Peter Attia: So, yeah, castration has been shown in most animal models to increase longevity.

Tim Ferris: It solves the volitional ejaculation issue.

Peter Attia: Yeah, yeah. So there has to be a balance, right? Similarly, I think there's pretty good evidence that there are certain dietary strategies that could really increase longevity, but if they come at a cost that detracts from wanting to live, they might not be worth it.

So that's one macro theme. The other macro theme is we do need to distinguish between, because these are sometimes orthogonal, what I would call cellular health and what I would call organism health. So I think there are things that matter on a cellular level. There are things that can delay aging of the cells within our body, and those aren't necessarily things that produce the best output of the organism. And so I suspect that the solutions to this thing, which I have lots of thoughts on, are not going to be simple.

There's going to be a few 80/20 pieces thrown in, and a little bit of bootstrapping, and a little bit of empirical feedback. I think there's gonna be some people that respond more favorably to some things



versus others. And I do think there's some really exciting pharmacologic plays that are out there that need further study but I think could sort of move the needle.

Tim Ferris:

So just conceptually I think people will find a number of things interesting that I'll just highlight here. One is the thinking of life extension or death avoidance. So I think death avoidance is sort of an interesting phrasing. But then also having a framework, like you said two issues, one's defensive; the other's on offensive. But delaying dying and optimizing living, right? And put another way, getting sort of 80 percent of the long term benefit while still getting - let's just argue - 60 percent of the short term pleasure. I just think it's written in a very compelling way.

I had a couple of very, very specific questions for you. People might find this amusing, so I'll just say it. I added a word to a line here. You have a line that is, "Streamlining this a bit further, once you've arrived into your 40s, your 50s, and assuming you're not a smoker or heavy drinker, you don't do IV drugs or engage in... dot, dot, dot."

And I put you should put, "You don't do bad IV drugs," in parenthesis since I do IV glutathione and all sorts of stuff. The question I had for you is related to IGF1, and I'd love for you to explain IGF1 to people. But I've talked about very openly and I've written about for instance after reconstructive shoulder surgery I had a number of doctors supervise use of anabolic agents including testosterone or an Androlone plus human growth hormone and other things. And certainly injecting exogenous growth hormone, twice a day, six days a week or whatever it might be - sort of low dose, high frequency - makes your IGF1 levels much, much, much higher than they would normally be.

And I'm curious because it appears when we're talking about cancer that decreasing IGF1 could be beneficial. What are your thoughts on the risks of growth hormone use for instance?

Peter Attia:

Yeah, so let's put this in the broader context. So as you said, there's really two pieces to longevity. The first is delaying death as long as possible. We call that the defensive plays, and then the second one is enhancing life - the offense play. On that defensive play, there are basically four diseases that are gonna kill you. In other words, if you're 40 years old and you care about this, you're probably not going to die in a car accident because you're out of that demographic. You're less likely to die of X, Y and Z. It turns out that when you look at the mortality tables there's an 80 percent

chance you're going to die from cardiovascular disease, cerebrovascular disease, cancer or neurodegenerative disease, period.

And, again, I think it's really important. If you remember nothing else, remember this. If you're in your 40s or beyond and you care about this issue, which immediately puts you in a selection bias category, there's an 80 percent chance you're going to die of those four diseases. So any strategy towards increasing longevity has to be geared towards reducing the risk of those diseases as much as is humanly possible.

Tim Ferris: Not just to do a recap, so cancer, we've got that. Most people are familiar with cancer. Cardiovascular disease, I think most people at a very, very high level are familiar with that. Could you just briefly define cerebrovascular disease and neurodegenerative disease?

Peter Attia: Yep, so cerebrovascular would be stroke, and there's two ways you can have a stroke. One is through an occlusion; the other one is through bleeding usually due to elevated blood pressure and things like that. And then neurodegenerative disease as its name suggests is degeneration in the brain. The most common cause of that is, in fact, Alzheimer's, dementia, and Alzheimer's is one of the top ten causes of death in the United States.

So what do those four diseases in common? They have a lot of things in common. The most obvious is they're metabolic diseases meaning they're not infectious diseases. They don't kill you quickly. These are all disease processes that build up over time. The second thing that jumps out at us is that these are diseases that did not exist in populations that failed to see Western influence. So I'm not here to say that the west is bad and we've done a bad job because I think great things have come out of everything, you know, Western civilization. However, some bad things have come out of Western civilization. So as it comes to my interest around diet, I think the western diet - the traditional western diet - is problematic.

And I think evidence of that is that if you look at societies that haven't consumed western diets or look at the introduction of western diets to these societies you're going to see all of a sudden these metabolic diseases become the dominant sources of death. Now, if you dig a little bit closer, you realize that there's a really interesting phenomenon in the anti-aging literature that cropped up a long time ago, and it is still talked about just as much. And it's the idea of caloric restriction. Now I'm positive that you have

written and or spoken about this at some point, so I won't belabor the point.

But the idea is that in anything from fruit flies to mice to rhesus monkeys, when you restrict calories, most of the time though not all of the time you appear to increase longevity. And you appear to increase longevity by delaying the onset of those metabolic diseases. Now the million-dollar question - though I have a strong point of view on this - the million-dollar question is, "Is that effect coming due to the reduction in a number of calories, or is it coming as a result of a reduction in a subset of the type of calorie?"

Because if I said, "Tim, we're gonna take your diet, and we're gonna reduce it by 30 percent." Well, technically, I've reduced how much fat, how much saturated fat, how much protein, how much carbohydrate, how much sugar, all these things. And so is it any one or combination of those things that have been reduced that's driving the increase in longevity? And if so could you get the same benefit without restricting calories and just restricting that agent, or is it the aggregate?

Now I'll tell you why I believe the answer that it's a specific set of macronutrients and not the number of calories though I could be entirely wrong because the experiment I'm about to describe is highly flawed. When I say flawed, I mean it's not apples to apples comparison, so two very famous studies that were done, one at the Wisconsin and one at the NIH using two different strains of rhesus monkeys, so right out of the gate you've got two separate problems with them.

At the NIH study they took the rhesus monkeys and they fed them a calorie restricted diet of sort of what I would call whole foods, meaning like real monkey food, like the food that they would eat in their environment. And these animals did in fact experience a slight increase in their survival. The monkeys in Wisconsin were given like a laboratory made pellet of their food and obviously at lower caloric level. The problem is they hated it, and they wouldn't eat it. And the only way they could get the monkeys to eat it was to add a bunch of sugar to it. And so those monkeys went ahead on a high sugar, calorie reduced diet. It had 28 percent sucrose in it.

Tim Ferris: Oh, god.

Peter Attia: And it turned out those animals did not experience a survival benefit. Now that doesn't prove anything because these were two separate experiments. They weren't controlled. But it does suggest

to me that there's something about highly refined carbohydrates and sugars and potentially protein, though it might be for a different reason, that seems to raise insulin, which we know, and by extension raise insulin like growth factor.

And we know that IGF is driving not just aging but it's also certainly driving a lot of cancers though not all of them. So my thoughts on exogenous - again, exoskeleton - exogenous growth hormone would be I just wouldn't view it as a great thing to do other than if you had a medical need as you described, certainly recovering from an injury, things like that. I certainly know people that clinically are deficient in growth hormone and in whom it's a good thing to manage.

But it certainly does concern me a little bit that I know that it's sort of the drug of choice of athletes today because it's still undetectable from a performance enhancing standpoint relative to say testosterone or sort of the typical things that athletes are getting busted for. But I personally think it's a little disconcerting that people would be pumping themselves full of growth hormone for their 20s and 30s and 40s or whatever because I do think in the susceptible individual that could be problematic.

So not ubiquitously but in the susceptible person I think it could be a problem.

Tim Ferris: Just thinking about it, ironically, the people who are most inclined to use it as non-athletes - i.e. people who are getting older and want to improve their youthful vigor or restore some of that youthful vigor - may be the riskiest population to start administering exogenous GH because they may have sort of precancerous or cancerous - what would they be called - cancerous cells that have not yet metastasized or anything like that present already and for just like 40 or 50 years of accumulated DNA damage and so on. Don't know.

Peter Attia: Yeah, no. It's a super interesting clinical question because that's another very interesting population. So, for example, I'll give you an example of a population that does really well high doses of anabolic steroids and growth hormones. And that's patients with HIV wasting.

Tim Ferris: Yeah, definitely.

Peter Attia: So you talk about a group and I've seen these people recover. Like it just blows my mind. Like it's so amazing to see someone on

heart therapy and anabolic agents and growth hormone, and you think to yourself, “This guy has HIV? Like, there’s no way.” So you might say, “Well, look, is there an increased risk in that person’s life from taking the growth hormone?” and the answer is, yeah, maybe, but compared to what? I mean, look what we’re rescuing them from. So that’s an obvious case. That’s a no-brainer. To your example Tim, you know, someone who’s 80 years old, maybe their greatest risk isn’t cancer at that point. Maybe it’s a broken hip, right? Maybe it’s a fall, and we know this. An 80-year-old that’s gonna fall and break their hip has as very significant mortality whether it be directly from a pulmonary embolism or indirectly.

That’s a huge risk for an 80-year-old. So that’s the nuance to this, right, is we can’t just sort of say, “This is good. This is bad.” It’s gotta be, “This is context specific, and we’ve got to be able to make tradeoffs.”

Tim Ferris:

So speaking of context specific and different activities or interventions - whatever you want to call them - not always being 100 percent good or very seldom being 100 percent good, 100 percent bad, I wanted to chat about something that came up in this manifesto. And that is that there appears to be evidence that heavy amounts of aerobic, especially subthreshold efforts, activity, may result in right sided cardiac dilatation it looks like - stretching - which may be the driver in paradoxical rise we’re seeing dysrhythmia.

Perhaps you could just explain that for the lay audience that’s listening to this but the idea that what we think is exactly what we should be doing, or that some people might think, could be doing us in fact harm. And the question I put in the margins there was, “Subthreshold, what percentage max heart rate are we talking about?” Because later in the piece there’s a discussion of walking, which gets the greenlight. I’d love to hear you expand on this a little bit.

Peter Attia:

Yeah, so it’s been generally sort of not acknowledged that readily, but if you actually go back and look through the literature, which I did about three months ago, I wanted to look at the literature of atrial fibrillation, which I’ll explain in a second, in athletes. So why did I want to do this? Because I basically started noticing every person I knew was getting A-fib, so people who were older than me, my peers, patients that were coming to who were stud athletes.

I mean, obviously, they weren't all getting atrial fibrillation, but enough of them were that I was sort of like, "What in the heck is going on here? So atrial fibrillation is a rhythm of the heart where the atrium - that's the smaller collection chambers in the top of the heart to be contrasted with the ventricles that have to do the big pumping. The atria, which only transmit blood into the ventricle, if you get beats that are generated there that are not what we would call a normal sinus beat - like there's a pacemaker within the heart that generates the normal, regular beat that you can feel if you put your hand on your wrist or your chest - an atrial fibrillation is an irregularly irregular beat.

And it often gets kicked off with what we call a rapid ventricular response. So it can sometimes start being so quickly that even the ventricle picks up that beat, and all of a sudden you've got this horrible heartrate.

Atrial fibrillation is a very dangerous beat the wrong patient. So it's very dangerous in people who are older or who get it for the reasons of ischemia, meaning lack of blood flow to the heart, because not only does it lead to demise directly but it can predispose you to forming clots within the chambers of the heart that then get lodged up in the brain. So older patients who are atrial fibrillation, even younger patients who are on it where they stay in atrial fibrillation long enough, are usually put on very aggressive blood thinning medication.

So the question is why are all these athletes getting it, right, because it seems disproportionate? So if you look at the population as a whole and say, "How many 40-year-olds have A-fib?" the answer is like none. But if you look at cyclists, it's like 10 percent. If you look at runners, it's a little bit lower. So I basically started looking through all this literature.

And a couple of things come up. So there's a guy by the name of James O'Keefe who's a cardiologist at the university. I think he's at Wisconsin, and he's a great athlete himself and a cardiologist. But he's also been interested in this for a long period of time. He's got some ideas, which suggest that long amounts of sort of pretty exhausting cardiac stuff - the stuff you'd experience if you were riding in an iron man or something really hard or running a marathon really hard - it actually creates a stretch in the heart.

Now, we know that's true. That's how we increase cardiac output. But it does so at a level that the right side of the heart can't compensate as well as the left. So the left side of the heart is very

muscular because it's needed to pump blood against the entire resistance of the body. The right side of the heart is actually not muscular because it only needs to pump against the lungs, which are a very low resistance system.

Tim Ferris: Which side has the higher musculature?

Peter Attia: Left.

Tim Ferris: Left.

Peter Attia: So the left ventricle, if you took a normal heart out of the body and did an autopsy or somebody or took a heart out of a sheep's body or something, you'd see this very thick muscular left wall, and then the right one collapses. You barely see it. And, again, it's because in normal physiology one of them is pumping against like 120 millimeters of mercury. The other one's pumping against like 20 millimeters of mercury. When you exercise, when you really, really throw down the hammer, you have to increase cardiac output. Meaning, how do we measure that?

We measure that in liters per minute. How many liters per minute of blood are going through your body? And so if I'm sitting here right now talking with you, Tim, you know my cardiac output is three, four, maybe five liters per minute. When I'm in a race, when I'm doing an all-out threshold, subthreshold effort, I'm at 25 to 30 liters per minute. And I get that not just by beating faster, which gets me part of it, but by taking bigger beats each time.

Tim Ferris: Stroke volume.

Peter Attia: And that's the stretch. You got it. That's the stroke volume. And so what O'Keefe has argued and certainly showed in animal models and there's a ton of epidemiology that certainly suggests it, although I don't think we're ever going to have a clinical trial that can demonstrate this one way or the other, is there's a subset of people for reasons I don't know who are susceptible where chronic, right-sided dilatation over and over again leads to distortion of the right side which leads to atrial fibrillation or worse fatal dysrhythmias.

We do see it. Fortunately, it's quite rare, but nevertheless it's tragic where we see these athletes dying suddenly. And this is not what we typically think of as the sudden athlete death, which is actually massive left-sided hypertrophy. That's a separate cause, but I think we're seeing more commonly young athletes dropping dead from

what looks like a heart attack only to find out their coronary arteries are pristine.

Tim Ferris: If someone is not a professional athlete, like you said they're not gonna lose a million-dollar sponsorship deal by changing their form of exercise, tentatively what would be your recommendations for perhaps things to minimize or ration a bit or things to do more of?

Peter Attia: Well, look, this is where I'm certainly the most hypocritical in all of the things I think about.

Tim Ferris: You're doing precisely.

Peter Attia: Yeah, exactly. And I wrote about this. I'm very transparent about this, right, which is right now I look at my approach to cycling as completely illogical. Like, there's no upside in it, and there's just downside. And that says nothing about the risk of getting hit by a car and all the other stupid things that unfortunately happen. Um, but for whatever reason, it's scratching an itch within me that is giving me so much pleasure that every year I just sort of say, "Well, one more year, one more year."

And I don't know. Maybe I'm waiting for that first bout of A-fib, and then I'm gonna be like, "I'm done." And hopefully the A-fib goes away. So I think what I'd want to reiterate is, look, you got to balance your own happiness and sanity with sort of your desire to live as long as is humanly possible. And I think that if I couldn't ride my bike, I would just be very unhappy even if it gave me an extra five years of life. At some point that might not be true. That said, for people that I take care of who are like me, I don't try to talk someone out of doing this, right.

What I do though, Tim, is nothing breaks my heart more than seeing that person who's struggling to lose weight who thinks that they need to be running 20 miles a week. And it's like they have no desire to do it. Their knees hurt. They hate it, and they're not losing weight. And I'm like, "Well, I've got great news for you. You don't ever need to run another step a day in your life because there's no value in that."

There is value in exercise though, and I do think that the most important type of exercise, especially in terms of bang for your buck, is going to be really high intensity, heavy strength training.

Tim Ferris: And that's for mitochondrial density?



Peter Attia: I think it's mitochondrial density. I think it's also just general glucose disposal. I also think it matters as far as aging us, meaning so many of the injuries we get as we age are kind of not just orthopedic like as in, "Oh, I have neck pain or knee pain," but a result of our inability to be strong.

Tim Ferris: It is sarcopenia? Is that the fancy way to say it?

Peter Attia: Yeah, I mean, that's a more extreme sense, but, yeah, it's sort of like you're going out to play ball with your kid, and you get hurt doing it. It's not that you're hurt because of anything you did that you didn't have the athletic ability to do.

It's been that you've been a bit deconditioned at the muscular level. I'll give you an extreme example. If you look at the best athletes in - I don't know, pick your favorite league - the NBA, the NFL, you look at them at their peak, and then you look at them when they leave. What's the difference? What's the difference between LeBron James today and LeBron James ten years from now? I don't think anybody disputes. In ten years he will either be retired or a fraction of what he is today. What's the difference? Is it athletic skill? Will he have less ball handling ability? Not a chance.

The difference will be strength. He will be weaker. On a strength to bodyweight ratio, he will be weaker in ten years than he is today. So I'm not saying that his strength is what makes him a great athlete today. His great athleticism is due to a number of factors. But I'm saying that the thing that deteriorates as we age is basically our power to weight ratio. And so I like to see people, whether they are athletes -

[Crosstalk]

Tim Ferris: So not absolute strength. You're talking about relative strength?

Peter Attia: Yeah, I think it's power to weight. I don't think that we necessarily are going to increase or strength. Look, I'll never be as strong as I was when I was a powerlifter in high school. I mean, at a weight of 160 I could deadlift 500 pounds and squat 425. I have no desire to do that again, right? But the point is I think I can still increase my power to weight ratio now, and I can keep there. And I can do it using muscles that are also a huge part of glucose disposal and metabolic health.

Tim Ferris: Just a quick question, a couple of quick questions, the first is do you think if you look at the careers of boxers like Mohammad Ali

and there are other boxers like George Foreman. now, George Foreman remained strong for a very long period of time.

And he was able to come back out of retirement and compete because his style didn't necessitate speed whereas you have say an Ali where for any number of reasons speed seems to be the first thing to go. Is that a reflection of a loss of strength in some sense, or is that a loss of neurotransmitters that just give you better sort of conductivity from the brain to your periphery or other?

Peter Attia: Yeah, so it's a couple of things. Ali's case is complicated because I think there's a huge appreciation in the neurology community now that dementia pugilistica, which is the brain damage that we see in boxers which is not what happened to Mohammad Ali. Of course, he got Parkinson's disease, but of course it's hard to make the case that boxing didn't accelerate that. maybe he would have always gotten Parkinson's.

Tim Ferris: Tyson's another good example because his style was so predicated on speed.

Peter Attia: Yeah, so speed is power. So speed requires strength. There's no such thing as speed without strength.

And it's funny because boxing's a sport I love so much. I used to. I couldn't care for the sport now, but at the time most people don't appreciate how hard Ali hit. Just got back and watch Ali versus Cleveland Williams and tell me Ali couldn't like knock a fire pole over. So, yeah, styles make it, absolutely. Actually, Foreman's a bit of an exception. Usually brawlers turn to burn out first in boxing because brawlers are typically more one trick ponies. I almost attribute George Foreman - remember he had two careers. After Ali beat him, he then lost I think to Jimmy Ellis in 1977 and then retired and then like literally just went off the face of the earth and the shows up ten years later kind of reinvented. It's sort of an amazing story.

Tim Ferris: Made \$120 million with the George Foreman grill and came back a new man. It was an amazing story, yeah.

Peter Attia: Yeah, so I think you're right. I think that strength is basically everything that I describe as follows.

It's firing of the transmitter in the brain. It's the neuro fire. It's the impulse down to the motor end unit, and it's the contraction. That's what I mean by strength, and I think that's the element that

deteriorates. Now the question is - the million-dollar question is - why is it deteriorating and what can we do to delay the deterioration? I don't know the answer, but I believe the answer is it deteriorates due to lack of use. We basically stop training it in a way that we once did. Now I think that there's something inherent that's causing the deterioration. Meaning, I just think there's some aspect of aging that is slowing that down, but I think we can delay it with a specific and certain type of training that replicates the switch if you will.

Tim Ferris:

No, definitely. It brings to mind a rather embarrassing experience that I had with you where we went to the gym together, and we did a bunch of glute medius exercises.

And for those who don't know what that means just imagine the side of your hip basically. I know this is vastly simplified, but imagine you're then laying on one side like a Suzanne Somers or Jane Fonda. We weren't doing exactly clamshells, but we were doing a lot of glute medius exercise. And it was agonizing. I just remember kind of rolling around on the floor.

It was quite a show for everyone, but it really made me realize that as a noncompetitive athlete at this point sitting down oftentimes in chairs that are not very ergonomically set with kind of my knees splayed out or crossing my feet under a chair or whatever it might be, my glute medius had become so weak. As soon as I did literally three or four of that workout, I felt like my hip stability, my knee stability, my ankle stability, the entire sort of chain from the floor up was improved.

And I felt - one could argue - younger, but it was simply from conditioning muscles that had become deconditioned from too much desk monkey work.

Peter Attia:

Yeah, I mean, those are motor end units that hadn't been firing, and glute med's one of my favorites, which is why it's just a huge part of what do. But the same is true of most muscles that control lateral movement. I'm all for deadlifts. I think it's probably the single best exercise you could do. If you could only limit yourself to one exercise in life, it'd be a hex bar deadlift. But you gotta be a little bit careful because most sort of exercises like that are typically working muscles in a forward plane. And most athletes are actually weakest in the lateral plane, and so we've always got to keep in mind that very strong glute med, very strong tensor fasciae latae, very strong vastus medialis, completely essential for knee-hip alignment and longevity of performance.

Again, that can be like literally gardening and walking and doing the stuff that I think we most realize we want to be doing when we're in our 90s.

Tim Ferris: So I want to ask you a couple of rapid fire questions. Then I know you have to get running, and if people would like to perhaps sometime hear a round two, definitely let us know guys. I'm twitter @TFerris, T-F-E-R-R-I-S. What's your Twitter handle, Peter, if you have one?

Peter Attia: Yeah, @PeterAttiaMD.

Tim Ferris: That's easy. And I'll put all this in the show notes, guys, but the rapid fire questions. So the first one I want to ask is do you meditate, and if so what type of meditation do you prefer?

Peter Attia: Oh, sorry. Can I not give you a rapid fire answer?

Tim Ferris: Okay, let me switch the order. Yes, I've planted the seed on that one. The first one is, what book have you gifted to other people the most?

Peter Attia: Probably, "Mistakes Were Made, But Not By Me."

Tim Ferris: What is that book about?

Peter Attia: Actually, and the other one is, "Surely You're Joking, Mr. Feynman!" Those would be a tossup.

Tim Ferris: Oh, great book, great book.

Peter Attia: So you know "Surely You're Joking." My son, by the way, he was born three months ago, his middle name is Feynman. So he's got big boots to fill. "Mistakes Were Made, But Not By Me," is a book about cognitive dissonance, and it's one of the few books that at the moment I finished it I not only reread it but I bought it for about ten people. I think that the authors, one of whom I've become very close friends with and she is now actually an advisor to NuSI as well, her name is Carol Tavris. One of the things the authors do such a great job of is really getting at the psychology of why it is that we are simply not wired to acknowledge mistakes when we make them, look for weaknesses in our thinking.

And I just think that, you know, how do I make sure I go through life without becoming too sure of myself? Because on some levels

I am sure of myself, but at other levels I have to realize, like, what can I do to make sure I'm not missing something that could allow me to do a better job? And so I think it's a fantastic book.

Tim Ferris: Very cool. And that author's name is Carol Tavris, T-A-V-R-I-S.

Peter Attia: Correct.

Tim Ferris: And the other author is Elliot Aronson for people interested. I'll put this in the show notes as well. Okay, so meditation, you don't have to give a rapid fire response. That's why it's called rapid fire questions. You could take a half hour to answer.

Peter Attia: So when I think about the pillars of longevity, what are the things - what are the levers - you have to pull to live the longest, most productive, high performing life imaginable?

It involves fixing your nutrition. We've talked about that. Changing your exercise - we've talked about that. Fixing your sleep - we have not talked about that, but that's important. Using the right supplements - we've not talked about that. Modulating hormones as necessary - we've not really talked about that. Fixing anything that needs to be fixed, on top of that pharmacologically, and I'm a huge proponent of pharmacotherapy under the right setting. The final component is managing your stress. So as a guy who's thinking about this, I became really interested after actually reading Dan Harris's book, which the title is blanking on me, but it's "Ten Percent Happier" actually is the title of the book.

Tim Ferris: Yeah, that's the one.

Peter Attia: And I loved the book, and I read it. And I've read a lot about mediation, but it was the first book I read where I thought, like, "I can relate to this guy." You know?

Tim Ferris: I think the analogy he said is when he was trying to learn to meditate it was like being dragged behind a boat trying to waterski.

Peter Attia: So true. So to make a long story short, that made me decide it's time to get off the couch and try this stuff. And what I did is I spent about three months really working hard at what I think is the second of sort of three types of meditation that exists. So the three types being focused attention, the second being open monitoring, and the third being transcendental. And so this open monitoring or mindfulness approach, which was that which at least for Dan Harris turned out to be the one that worked the best for him, I

worked my tail off on it. And I think I got some benefit, but to be honest with you, three months in, which maybe wasn't enough and I'm sure there's somebody listening to this podcast who's like a guru at it and is like, "Oh my God, you're committing this common fallacy of like, blah, blah, blah," it just was too hard for me.

It was like I'm okay drinking the ketones once a while, but I can't drink them every day. And it's like I just couldn't suffer through this.

Tim Ferris: It's the existential equivalent of the ketones.

Peter Attia: Yeah, yeah, yeah. So then I was talking to a mutual friend of ours, Dan Loeb. And so Dan and I were talking about this, and I'm hanging out with Dan for like a day. And guy like in the day that we're hanging out this one day, he meditates twice for like 20 minutes.

Tim Ferris: For those people who may not know, one of the most preeminent and successful hedge fund managers in the world.

Peter Attia: Yes, and also just a super cool, funny, thoughtful guy who obsesses over all the same stuff, which is why we all hang out. So Dan was doing like 20 minutes of transcendental meditation a day, and so, you know, we started talking about it. He said, "Look, I gotta introduce you to my guy." And so he introduced me to his guy in New York, and this guy really put a lot of things in perspective for me, right.

He's like, "Look, it's not that there's any method that's good or bad. I think the key is finding the one that works for you." This guy happens to teach transcendental. He's made a pretty good case why I may find the most benefit in that.

Tim Ferris: What was the case?

Peter Attia: The case is basically when you look at focused attention and open monitoring, for people who tend to be really, really restless it's hard because they have to focus on something that by its very nature is against what they do. He knew a lot of about me, and I think guys like me and Dan Loeb are sort of similar in that sense. I mean, I have a hard time sleeping because I'm trying to think of 200 other things to do. I think the reason he thought transcendental or automatic self-transcending meditation would be valuable is

that it would allow thinking. I think he described it as allowing the thinking mind to experience a quieter level of thought.

And he's like, "I think for you, Peter, that could be more beneficial than trying to just be present." So I think what I would say, Tim, is I'm in the early stages of exploring that. I am committed to figuring out ways to reduce my level of stress because if I want to get serious about doing all these things that we're all obsessed with, I think you've got to take a full and comprehensive approach to it. I mean, this is questions about me, but tell me your experience on this because I know you must think about this a lot.

Tim Ferris: Sure. I had a very similar experience exploring meditation, and the only meditation that really stuck for me was transcendental meditation. There are aspects of many of the organizations that teach TM that bug the shit out of me. With any subculture you can have a certain degree of sort of culty vibes, and I think that's true for many, many different groups.

But as a technique I found it extremely secularly useful as a way of giving the mind a warm bath was one of the expressions that I heard. Are you at the moment repeating word or a mantra? Is that the general technique that you're using, or are you doing something else?

Peter Attia: No, no, no. I'm not even there yet actually. I'm still doing my very slow Peter Attia sort of reading about it, find out who the gurus are, yeah.

Tim Ferris: Yeah, so I found generally speaking the reason transcendental meditation has been the most successful is it's kind of like - what's the expression - it's a terrible system, but it's the best one we have.

I don't think that TM is terrible, but it has had a stickiness for me beyond everything else that I've tried. I think that for me there are a couple of reasons for that. Number one, I was held accountable, so I paid for a teacher. And I had to meet with that teacher once a day for four days and meditate two times in between sessions and then report back on those sessions. And there was a risk of embarrassment and a sunken cost that I think facilitated my practice.

That was number one. It wasn't just up to me, and there was a cost - a social cost and a financial cost - to not doing it. The second is they made the game - this teacher made the game -winnable. So I think that meditation is often very uncompromising in the same

way that let's just say 100 percent pure paleo or 100 percent strict veganism, they might be effective for certain people, but 99 percent of the folks out there will never stick with it for more than two weeks because they're too prohibitive.

And you can't win. It's hard to win - inconvenient to win. Whereas with TM, it's like, look. If you sit down and you meditate for 20 minutes and you say your mantra or your word two or three times and the rest of the time your monkey brain is just running through your to-do list, that's okay. You have meditated, and that was a successful session.

So setting a frame through which I could look at mediation and not feel like I had failed if I was thinking about some stupid, you know, offhanded asshole comment that some guy made to me in a salad line in college for like ten minutes or whatever the hell I'm like thinking about, you know. And then when I've tried to meditate before, I'm just like, "I am terrible at this. I can't believe I thought about Transformers 3 and like Megan Cox for like 15 minutes."

Like I'm terrible at this, and I would get really frustrated. And I would quit, and this teacher was like, "No, it's fine. You're still deriving a lot of benefit from sitting with good posture and breathing deeply even if your mind is going crazy and kicking and screaming. And feeling like I was winning, I feel like this should make sense to you because we're both so competitive.

Peter Attia: No, no. It completely does, and I'm glad to know I'm not the only one that really sucked at open monitoring.

Tim Ferriss: I was terrible. So for me I have mediated regularly. I continue to experiment with, for instance, yoga type calisthenics with particular types of breathing rhythms that I'm interspersing with the mediation. But typically if I meditate, for me the magic number is about 20 minutes, if I can sit for 20 minutes. And I think a lot of it quite frankly has nothing to do with what's going on in my head.

It has everything to do with sitting still - completely still - and breathing deeply for 20 minutes. And for me having a noise to repeat, and I don't like using the word mantra because it sounds too woo-woo new-agey but having a sound to repeat incessantly - and they would hate me to describe it this way - but it drowns out a lot of the banter in a way that trying to concentrate on an imaginary candle flame cannot do for me. And so for me I found it very, very helpful, and without fail if I do it for a week straight -



first thing in the morning, you have to do it like as soon as you wake up, push yourself up and lean against a wall and do it, or it'll start to fall away - I find it tremendously helpful. And I deal with things in a much more relaxed, effective way. So that's my very long two cents.

Peter Attia: No, that's great to hear. So, yeah, it's a chapter that I must get to.

Tim Ferris: Switching gears to the purely materialistic, I'm curious. What is the best \$100 you've spent recently - \$100 or less? What is the item, the service, the anything that you've spend \$100 or less on that has been most worthwhile or helpful or enjoyable in recent memory?

Peter Attia: Actually, this is kind of funny. About a month ago my daughter who is six, we'd try maybe like once a month to go and do a daddy daughter date. So we'll go downtown to a hotel. Go out for dinner. The one thing is for a six-year-old, she has a remarkable palate. So if it's not like sushi or esoteric Indian food, she doesn't want it. So I can actually eat food I want to eat. And then we'll go back to the hotel, watch a movie, go to sleep. It's really a time I cherish. So we were walking back to the hotel, and one of those rickshaw guys came up with a fully lit up bike.

And I mean I just normally like would never even think about like hopping a ride on one of those things, and I could just see this look in her eye that was like, "Wow, like, his bike has lights all over it." So it's like, let's hop on. And so this guy gave us a ride back, which probably cost \$20, so not even \$100. And, believe me, it's \$20 more than we should have spent to just walk home, but the look on her face was worth every dollar I have. So, I mean, I just got a little cheesy and cliché because old dads are like that, but that's the best \$20 I've spent in a long time.

Tim Ferris: Cool, no, that's a good answer. I don't want people to have this image of you as a really nice guy though, so I'm going to ask another question, which is, "When you think of the word "punchable" whose face is the first one that comes to mind?"

Peter Attia: It's funny. I'm a pretty spicy, ornery guy. It's generally not directed towards individuals. I'm sure if given enough time, Tim, I could come up with ten, but there are sort of things that happen that I wish I could punch. Like I'm completely disturbed by the system of healthcare delivery in the United States, and if there was someone I could punch for that, I would. But I don't know who to punch. There's no one person. I'm completely just torn in pieces

by the utter state of nutrition science in this country. And yet I don't think knocking one person unconscious would fix it. Of course, by the time we're off this call, Tim, I'll remember 20 people, and I'll text them to you.

Tim Ferris: Perfect. I can put those in the show notes in order of preference. Maybe an easier question or a less loaded one, when you think of the word successful, who is the person who comes to mind?

Peter Attia: There are a couple of people I've met with who I've just gotten to know that just really define success for me. One of them is a friend of mine. Actually, it's the guy who emailed me about wanting to sort of think about transitioning from iron man to other things. His name is John Griffin. He's also a very successful hedge fund manager in New York, and John and there's a couple other people I know like John, another guy by the name Dennis Calabrese who's the president of John and Laura's foundation. We've become very close friends. These are guys that I just view as successful because they've done something I want to be able to do so badly one day.

So it's actually something my brother and I talk about a lot, which is how do you balance trying to be successful in the career that you set out to do and at the same time be as exceptional in your personal life? And so my brother who is a very successful federal prosecutor has four kids under the age of five, and he thinks a lot about being as good a prosecutor as he is being that good a father. And, in fact, this longevity manifesto that I've written, he's actually writing his called the Fatherhood Manifesto. And so in many ways that's what success is to me. Success is, "Do your kids remember you for being the best dad? Not the dad who gave them everything, but will they be able to tell you anything one day? Will they be able to call you out of the blue, any day, no matter what?"

"Are you the first person they want to ask for advice? And at the same time can you hit it out of the park in whatever it is you decide to do as a lawyer, as a doctor, as a stockbroker, as a whatever?"

Tim Ferris: One of the perennial challenges it would seem. Very cool. Well, I want to give us just a few minutes in closing, and for those people listening links to resources, books, etcetera will all be in the show notes. So that's at [4hourworkweek.com](http://4hourworkweek.com) - all spelled out - and just click on podcast, and it'll take you right to a whole list of episodes and the newest one will be at the top, namely this episode probably. I wanted to just give a bit of info on one of your current

initiatives, and I'm involved. But perhaps you could talk about the fatty liver project.

Peter Attia:

Yeah, it something I'm super jazzed about. So fatty liver disease is a disease that many people probably haven't heard of, but it's kind of a remarkable epidemic. It's a condition that was either barely existing 20 years ago, or if it did exist, it was so small we didn't know it. In the year 2000 about 1 percent of liver transplants in the United States were the result of this condition - non-alcoholic fatty liver disease - which is a form of liver failure that looks just like alcoholic cirrhosis except there's no alcohol involved.

Today about 10 percent of liver transplants in the United States are a result of this, and Ron Busuttil and other experts have suggested that by 2020 this will be the single most common cause of liver transplant in the United States. And that by 2025, 5,000 people a year could die waiting for livers in this country as a result of that. So believe that there's a nutritional component to this. That's why NuSI is working on this.

And after we spent the better part of four months interviewing 38 gastroenterologists and hepatologists around the United States and meeting with them over the course of two weeks, we gathered from them that there were three likely hypotheses for what was driving this disease. So the first is over abundance of calories, so just too many calories and then you get fatty liver disease. The other was an overabundance of carbohydrates, and the third hypothesis was an overabundance of sugar, specially fructose - the simple sugar within table sugar and high fructose corn syrup that makes it really sweet.

Tim Ferris:

Or agave nectar - 75 percent fructose.

Peter Attia:

Correct. Sure, agave, honey, things like that. So we said, look, here's all the data. Every week a new experiment comes out that sort of waffles one way or the other, and it was like the more we talk to these guys the more we realize there were simple things that weren't even know. Like, it's not actually known what the natural history of influx between NAFLD and NASH.

So those are just two technical terms that describe the state of when you just have fatty liver versus when you have something called steatosis. So we realized that there needs to be a really crucial experiment - a relatively small experiment by the way, certainly by NuSI standards but a very crucial experiment - that just answers the question very simply, what happens when you

change the diet of a child who has biopsy proven fatty liver disease? And to do this you've got to be able to feed the child every meal, every day.

And because you're not gonna lock them up in the hospital, you have to be able to feed their family every meal of every day because you can't have a kid eat one thing and have their siblings and their parents eat another. You've got to feed their entire family, and so this very small initial experiment that's going to pave the way to what the longer, larger experiments do basically has to take these kids and for two months feed them every meal and then check based on their MRIs how does the amount of fat in their liver change.

And you do that in parallel with a group that you're not feeding anything to, and that's the control group that you're going to see the natural evolution between NASH and NAFLD. And so this experiment is about \$1.2 million. Again, it sounds like a lot of money, but actually for a major nutrition science experiment, that's actually a pretty low cost. And, Tim, a couple of months ago you called me and said, "Look, I've got this idea, which is I want to sort of kick off a campaign, and I want to give you a bunch of appreciated Twitter stock. Where do you think it could go?" It was sort of perfect timing because I thought, "I could go exactly to that study because you had 20 people that kick in \$50,000 of their favorite appreciated stock, you've all of a sudden funded this thing."

And the way I look at it is fatty liver disease, we're going to look back at this one day, and we're going to look back at people who played a role in helping figure this out kind of the way we look back at other things that have changed the game in biomedical science. One of my favorite examples is certainly the development of the birth control pill, which we just take that for granted today. It's important to remember that in the '50s and '60s - certainly in the '50s, I'm sorry - post World War II, late '40s and early '50s, that was a completely taboo thought.

Nobody was going to fund research in oral contraceptives - nobody. And it actually took a woman by the name of Catherine McCormick who was a philanthropist to come along and say, "This is a really important thing. I'm gonna fund this myself." And she funded it to the tune of about \$20 million, and by the early 1960s we had the first working version of oral contraceptives. And if you look at the literature of the number of women that occupied

the workplace before and after OC's came out, it's like this unbelievable graph.

And so it's easy to take that for granted today, and I hope that one day we take for granted that people don't have fatty liver disease. But as it stands today, seven million children have fatty liver disease, and 40 million adults in the US have fatty liver disease.

Tim Ferris: What were the numbers again?

Peter Attia: Seven million children in the US and 40 million adults. Those are the CDC numbers, which are the most conservative.

Tim Ferris: Jesus.

Peter Attia: And I like to use those. I've heard other estimates north of 60 million Americans with fatty liver disease. But if we go with the most conservative, it's seven million kids, 40 million adults. The study we're talking about, Tim, this pilot study, is gonna be done in kids because I think the urgency's even greater there.

Tim Ferris: No, definitely. Part of the reason this study is of great interest to me, number one, like you said I think it's an astonishingly inexpensive target for an experiment that could really create sort of a phase shift and spur many developments and a lot more research in the right direction.

So, I mean, I would imagine there are all sorts of implications once you've identified - hopefully identified - causal factors or lack of causality related to certain things with fatty liver disease that then opens the door for new studies related to different types of visceral fat and all sorts of metabolic conditions. So this is very exciting to me just as kind of a lead domino, and for those people listening - and you wouldn't be hearing me say that sentence if you weren't listening, so welcome to the show - but I think that the research can be very expensive.

And this is sort of a really attractive, minimum effective dose sort of like throwing a rock at an avalanche to start it. So what I'm going to do in the post that accompanies this podcast on the blog, you can go to [4hourworkweek.com](http://4hourworkweek.com) and click on podcast, or you can go to [4hourworkweek.com/podcast](http://4hourworkweek.com/podcast).

They go to the same place. And click on this episode, and I will give you all information related to how you can learn more about this. It's very exciting to become citizen philanthropists as it

relates to science. I've had the good fortune of being involved with quite a few studies over the last several years, and it's really exciting to feel and in fact be part of something like that. So is there anything else that you feel, Peter, people should know about this that I'm leaving out?

Peter Attia: No, you've done a great job, Tim. I think your blog post will reiterate some of these points, and we're super excited. We agree. This is about dominos, and I certainly want to know once and for all what the dietary triggers of this are.

I'm not sort of quiet about my own hypotheses, but, boy, we certainly need better than best guesses to change policy around what we do and don't want to see people eating a lot of.

Tim Ferriss: Yeah, absolutely. And just as a side note to those people listening who want to become more involved with giving to causes - or organizations. When people hear the word "cause," I think sometimes it sounds like you're doing it to get a pat on the head and feel good about it. I do things like work with NuSI because I care about results, and I judge NuSI and other organizations I work with in the same way that I would judge a very lean, for profit startup. I mean, I want to see the metrics. I want to see the inputs versus the outputs, the costs.

For those people interested in getting a taste of that, consider if you have stock that that can be a much more efficient way to go about it because rather than just say selling \$100 worth of stock paying 30 percent or 30 to 40 percent in taxes or more and then giving the remaining, say, \$60 to a nonprofit or an organization, you can donate the stock - directly transfer that stock - and then they get the benefit of and you get the tax benefit of that \$100.

And, obviously, talk to your accountant and so on, but this is something that I feel stupid for not realizing earlier. I can basically in some cases nearly double my impact just by giving appreciated assets as opposed to post tax dollars. So something to think about. Peter, any final words? Where can people learn more about you? Obviously, NuSI.org. People can check out NuSI, N-U-S-I.org.

How about your blog, any other places that people should look for you?

Peter Attia: Yeah, my blog I don't write that often just because my day job takes up too much of my life, but the blog is [eatingacademy.com](http://eatingacademy.com). There's a lot of stuff on there about ketosis and exercise and

science and nutrition and things like that. And that's about it. I'm not a big guy. I don't hang out that much in the ether.

Tim Ferris: Well, I think that's probably partially why you get so much done. You're not busy clicking on cat videos and god knows what else. Well, Peter, it's always fun to hang out. I hope to see you in person again very soon, and thanks so much for taking the time to be on the show.

Peter Attia: Thanks very much, Tim. This was a blast and happy to do it again.