Diet Doctor Podcast
with Ben Bikman, PhD
Episode 63

Dr. Bret Scher: Welcome back to the Diet Doctor podcast. I'm your host Dr. Bret Scher. Today I am very pleased to be joined once again by Prof. Ben Bikman. Now if you've heard of Ben Bikman, hopefully you have, we did an episode with him, podcast episode number 35, and I got to be honest, he's just a great guy.

I really like speaking with Ben, talking to him, getting his opinion on things and hearing about all the research. And I think I try not to be too much of a... was it a boy fan or something or just a fan of his, because I want to push him and I want to make him clarify things.

Because he's so good at answering questions and so good at being clear about what we know and what we don't and still talking with passion and having the experience of being a researcher, doing the studies and therefore knowing how to interpret studies. So we spend a lot of time talking about insulin resistance and hyperinsulinemia because that's what he's been focusing on.

So to get to sort of his accolades, he's a PhD in bioenergetics and did a postdoc in metabolic disorders, he is an associate professor at BYU in the Department of physiology and development of biology and he focuses on insulin and ketones and their role in type 2 diabetes and obesity and metabolic disease and neurodegenerative disease.

So this is his area he knows more about this area than just about anybody in the world at this point I'd venture to say. He is also been very busy trying to reach people with his message. So he recently wrote a book, "Why We Get Sick?" As you'll hear, he's involved in two companies, Health Code and Insulin IQ, which he talks a little bit about at the end of the episode.

So, a very busy guy, a great guy, a very smart guy who really states things appropriately in terms of what we know, what we don't, and states it with passion so I hope you-- Oh, and humility... He is such a humble guy as well. Okay, I'll stop going on and on about how great Ben is and instead just let you get to the interview here with Prof. Ben Bikman.

My good friend Prof. Ben Bikman, it's a pleasure to have you back to the Diet Doctor podcast once again. Thanks for joining us.

Prof. Ben Bikman: Oh, Bret the pleasure is mine. It's great to see a friend and talk all things on metabolism.

Bret: Absolutely, we were joking before we got online, that every time I interview you, your biography is getting bigger and bigger, which shows you've been a very busy and productive man.

So it's good to see because we have to be honest, there's so much information out there coming from different sources and different people.

But I figure if you want to know about insulin resistance and metabolic health, you want to know about the guy who's got the PhD, who is doing the research, who is teaching the subjects and who probably knows more about the subject than most people in the world. And I got to figure at this point that that's you. I mean... and not to put you in an uncomfortable spot.

Ben: No, I am glad to be looked at as an authority, because I do think my sentiments do depart from others who speak about insulin resistance and act as an authority and I don't mean it in a derogatory way, but claimant authority in the realm of insulin resistance. And so I think I'm glad to be a dissenting voice in some instances with regards to insulin resistance; its causes, its consequences for example.

Bret: Yeah, interesting that you say dissenting voice and I guess there depends on which sort of camp you're talking about and it's sad that we even have to have camps when it comes to science, because science should be science.

But there is sort of a disconnect between maybe what the traditional or current medical community feels about insulin and insulin resistance and what some of the science says. So, is that what you would say is your dissenting view compared to the medical community or did you have something else in mind?

Ben: Yeah, well I am certainly a bit of a misfit in that regard and for example that's highlighted in my view of type 2 diabetes, where conventional medicine looks at type 2 diabetes as a glucose disease and I vehemently disagree with that sentiment and say we need to look at it as an insulin disease.

But even within the low-carb community there is some debate as to what are some of the fundamental causes of insulin resistance and so in that sense I guess neither view is dissenting because there shouldn't be a... you know, in science, or shouldn't really ever be a static consensus.

So I shouldn't invoke that term too lightly but I would say my views don't align with others who speak in the in the low-carb space about insulin resistance. And so I'm happy to have my voice be one of them that people will hear to add to the conversation.

Bret: All right, we hit the ground running right away talking about insulin resistance. So, let's rewind for just a second and tell us how do you define insulin resistance.

Ben: Yep this is an important point to start with because I think it helps really clarify where some people may see things differently. So, to me, insulin resistance as a term derives from the original observations from cell specific studies. So, to invoke the term insulin resistance is really invoking the perspective of the cell and some cells, like muscle cells for example, or maybe the very earliest and even fat cells these are cells that manifest with insulin resistance in an isolated cell culture.

We can expose the cells to different situations that actually make them insulin resistant. They're not responding to insulin very well. So, that's half of the definition as insulin resistance is used in a whole body situation or clinical situation. Then the other half is very relevant though and it's sometimes missed in the definition because insulin resistance in a human will always, always, always be accompanied by hyperinsulinemia.

That is the other half of the coin that cannot be dismissed, it cannot be overlooked or tossed out if we are coming up with a working clinical definition of insulin resistance. So again, insulin resistance is defined as some cells not responding to insulin and hyperinsulinemia. Those are the two sides of the coin and we have to understand both of them to understand why insulin resistance is so relevant to virtually every chronic disease.

Bret: Yeah, that's a really interesting point. So, it's not the cells not listening to insulin... but not all cells are the same, right? So, if you talk about insulin resistance in the muscle cell, versus the fat cell, versus the liver... so tell us some of the nuances there about how maybe you can have insulin resistance from one cell but not another.

Ben: Yeah, that's exactly right. There is very much a cell specific phenomenon here. You listed some that are known to become outright insulin resistance or very strongly insulin resistant and the muscle is a great example. When the muscle becomes insulin resistant virtually all of insulin effects are compromised. Including its inhibition of proteolysis or inhibition of protein breakdown, its inhibition of glycogen breakdown within the muscle and its stimulation of glucose uptake.

All of those things become compromised in the muscle cell. However there are other cells... an interesting example would be the thicker cells of the ovaries that do not become insulin resistant.

And so here we have a cell that in the context of the whole body is suffering because of the hyperinsulinemia which is kind of both cause and consequence of the insulin resistance, but here the insulin is acting with all of the sensitivity that it ever did and because the insulin resistant body is the hyperinsulinemic body now we have a thicker cell that is overstimulated with insulin and that leads to polycystic ovary syndrome because insulin inhibits aromatase, it inhibits the conversion of testosterone into the estrogens.

And of course it's a little-known fact that all estrogens were once testosterone in males and females. It's just the gonads make that conversion through aromatase. Insulin always inhibits aromatase... always. But because we have low levels of insulin in a healthy situation, the inhibition of aromatase and thus the inhibition of converting testosterone to estrogens is always very mild.

But now in the case of the hyperinsulinemia we've taken that inhibition of aromatase to a whole other level and now we are inhibiting the production of estrogens too much, and now she doesn't have the estrogen peak with the menstrual cycle and thus we do not have a follicle that actually ovulates.

And so she doesn't ovulate and the ovaries just maintain all the follicles which become cysts and now she has a cyst loaded ovary or ovaries. So I highlight this just to highlight two very different cells that are each responding to this overall insulin resistance / hyperinsulinemic state.

And even within-- Bret, it even gets more complicated because we could look at say the endothelial cells, the cells that line the blood vessels and normally insulin will stimulate a mild degree of growth, a little bit of hypertrophy in the endothelial cells and it will stimulate the production of nitric oxide, which allows vasodilation which lowers blood pressure.

But in the case of insulin resistance and hyperinsulinemia we have the hypertrophy still happening, but now it's happening too much. Insulin is still stimulating hypertrophy of the endothelial cells, narrowing the blood vessel. But unfortunately the insulin induced production of nitric oxide is compromised.

That cell, that specific biochemical event is insulin resistant so now we have less nitric oxide production in the blood vessel that once would've dilated when it saw insulin, stays constricted. Those together combined with a few other variables explain why insulin resistance is so pivotal to hypertension.

Bret: Yeah, so those are a couple great examples of how it really is hard to just say insulin resistance and think it applies to every cell. But at the same time it does help simplify things because there is sort of this common thread that it's maybe insulin resistance in the muscle cell and the liver cell that triggers the cascade that then affects how different cells are affected.

Because PCOS is thought to be a condition of insulin resistance. But you just said, it really is a condition of hyperinsulinemia too much insulin while still being sensitive to insulin. So I think that's a very important differentiation that we don't hear a lot about.

Ben: Yeah, and Bret, there's one other point to this that I think is so important especially with people that are familiar to the general low-carb community. I do believe that the term insulin resistance is invoked incorrectly where people are saying that the adherence to a low-carb diet is causing physiological insulin resistance. I disagree with that conclusion quite strongly because you cannot have insulin resistance without hyperinsulinemia in the body.

Truly I am unaware of any instance of actual insulin resistance without hyperinsulinemia. And people are using the term physiological insulin resistance incorrectly. That is a real thing, physiological insulin resistance is real, but in both instances which is puberty and pregnancy, you still have hyperinsulinemia in both of those states.

Bret: Okay, timeout for a second.

Ben: Yeah.

Bret: You've got too much knowledge and you love to talk about things. And I didn't want to get into--

Ben: I go too fast.

Bret: That's on my list of things to talk about but now that I'm thinking about I think we just need to step back and say what is insulin, what does insulin do? Because I think most people have an idea of insulin and certainly in the medical community insulin regulates blood sugar. But it's got so many other functions that when we talk about hyperinsulinemia and insulin resistance that are so important and you already alluded to some in the blood vessel.

About how insulin doesn't just regulate blood sugar but it can affect the hypertrophy of the of the vessel wall and production of nitric oxide. So we're going to get into the physiologic insulin resistance and all the other downstream effects of hyperinsulinemia, but tell us some of the basic functions of insulin that we're going to need to know about as we discussed its contributions to other disease processes.

Ben: Yeah, good, so for better or for worse and maybe more for worse in conventional medicine, insulin is generally just viewed by nature of its actions on glucose. So glucose goes up when someone eats a starchy sugary meal. And hyperglycemia is dangerous. That's an even very rapidly dangerous situation given its effects on the kidneys and so thank heavens the body has a mechanism for very rapidly lowering that glucose level, preventing it from being dangerously high for too long.

And insulin is part of that... in fact much of that. And much of that is insulin effects on the muscles. 80% of glucose after someone eats a meal that has carbohydrates, 80% of that glucose will go into the muscle and almost all of that will be given the actions of insulin. So again for better or for worse insulin is typically viewed through the lens of its effects on glucose and its glucose lowering effects. However insulin has... I say that insulin basically controls fuel use in the body.

So whether the body is at its simplest storing energy or using energy, whether it's more inclined to store or whether it's more inclined to use and even waste energy, insulin controls which direction the human metabolism is leaning, storage or spending. And so that's more reflective I think, more accurate.

At almost every cell insulin will tell the cell what to do with energy. And the theme of it is taken nutrients and store them, build something from these nutrients or are more complex way to store the energy or nothing having to do with energy at all, telling it to make proteins for example, or make lipids that will be used in non-energetic processes, but safer structure, or hormones.

Bret: Yeah, so it's really a growth hormone in a way. I mean it's telling cells to grow. And that can be fat cells to grow but it can also be, you said, protein, so could also trigger muscle cells to grow. So we think of insulin as a... You know, nowadays there's so much talk about insulin as being bad. The effects of insulin are bad. But it's got some vital functions in our body for blood sugar regulation like you said and for regular human growth.

But like a lot of things in modern society, you know, good intentions, you can say, are led astray by our lifestyles and that's when you get in this concept of too much insulin. So now big part of the message is that insulin isn't just involved in type 2 diabetes but also has downstream effects on high blood pressure, cardiovascular disease, cancer, neurodegenerative disease. So, what is sort of the common thread that insulin has in its function that can lead to all these other diseases?

Ben: Yeah, so I think the only common thread we could approach is insulin telling cells what to do with energy ultimately and whether to grow or not. So, across each cell-- I would think that's probably as close as I could get to creating a common definition and in the case of say, like if we highlighted some of the pathologies you just mentioned, in the case of cognitive decline that is thought to be the loss of insulin functioning at the brain is thought to be part of the loss of glucose uptake, which is a very real quantifiable event in humans.

We can detect that in in cognitive decline whether it's mild cognitive impairment or outright Alzheimer's disease, we can detect actual reductions in brain glucose uptake and brain glucose metabolism and insulin does mediate some of the brain's glucose uptake.

The brain has insulin-dependent glucose transporters. Now it has an insulin independent transporter as well, but we know that the fact that it has GLUT-4, an insulin-dependent glucose transporter, does suggest strongly that insulin is relevant to at least some of the brain's glucose uptake. So that could be part of the pathology with the cognitive decline or the neurodegeneration.

And another example would be fatty liver disease where insulin appears to be pivotal and that is insulin resistance in the global sense, because it's not the insulin resistance per se at the hepatocytes that are causing them to store fat. It's the hyperinsulinemia, the other side of the coin.

So once again highlighting the fact that when we talk about insulin resistance in the context of health and disease, we need to understand that that is a loaded term that encompasses the hyperinsulinemia which is not discussed. And indeed when I'm mentioning insulin resistance I am

putting the two together in one term.

Bret: Yeah I think it was so interesting in your book how you mentioned that even textbooks from 20 years ago listed the brain as an organ that had no response to insulin and insulin had absolutely zero effect on the brain. And now that's completely false and it's that old saying that 50% of what we believe is false, we just don't know what 50% yet. That turned out to be one of them. And now it's opened up this whole new field of neurodegenerative diseases possibly being insulin and glucose related.

And when it comes to diseases like that though I also want to say or ask you what is the level of evidence in your opinion that we have linking high insulin levels as being a causative factor in a number of these disease states? Is it associated, is it causative? Where do we stand now and where do you think are headed from that standpoint?

Ben: That's a great point to make. And I would want anyone to know lest I be accused of kind of having a one track mind... I'm not attempting to say that insulin resistance is the only variable say for example causing Alzheimer's disease. I would never be so bold as to say that. So, in humans there are strong correlational evidence, which is probably about as good as we can get with humans, that we find that insulin resistance is one of the strongest predictors of Alzheimer's over a 10 or 11 year period.

There is one very specific study that the title starts with something like, "insulin predicts Alzheimer's disease over 11 years". So in humans there's strong correlation; in fact one study from Finland found that fasting insulin had a higher statistical significance to Alzheimer's risk than age did. By that I mean the P value was stronger with insulin than age.

So pretty compelling. In rodent models we know that if we start manipulating the insulin receptor or the GLUT-4 glucose transporter, we cause neurodegeneration. We start to cause cognitive decline in the animals. So there's a much more causal link which is the reason we use animals at all. It's to do causal mechanistic research that we just can't do it humans.

Now adding to that, my lab... we have this in the second round of review right now specific to Alzheimer's disease where we study gene expression from human brains. So, of course postmortem, people who died without any evidence of Alzheimer's disease and people who died with... as confirmed as we can with Alzheimer's disease.

And we did a wide genome analysis and found that there were fundamental reductions in almost every gene involved in glucose uptake and glucose metabolism in the brains with dementia. Now I cannot say that that's a result of insulin resistance and I wouldn't try.

But I would say it lends more evidence to this kind of energetic deficiency of Alzheimer's at the origin of the disease. But interestingly when we looked at genes involved in ketone uptake, in ketone metabolism, there was no significant differences across either the brain's with dementia or the healthy brains.

Bret: Interesting, very interesting. So, I think that's a very good overview of sort of where we stand for insulin resistance and dementia. So when you say the level of evidence is similar for hypertension and for obesity and for cancer or do we have a different... are some sort of more proven than others?

Ben: Yeah, I think so, insofar as you can... in a human situation with type 2 diabetes... Well, we

know in humans that if you cause insulin resistance, insulin stimulated glucose uptake is compromised. So I think in the case of type 2 diabetes we can say that the mechanism has been established in humans. In the case of... what was the other disorder you mentioned?

Bret: Hypertension.

Ben: Hypertension, yeah, very well. Established hypertension, as a consequence of insulin resistance has been really well-documented from the 1980s with Gerald Reaven's group so that's a much more established causal relationship to the point that they often will say that if it's primary or essential hypertension it's generally going to be actually a result of insulin resistance. So, with Alzheimer's it's probably more we're relying on animal data to establish causality.

With other disorders like hypertension and type 2 diabetes we have much more causal evidence even in humans. Yeah, I think it is so interesting that hyperinsulinemia, insulin resistance are correlated and potentially causative of so many different diseases, which originally... at least me as a physician the way I was trained and thought was that they are all separate.

Alzheimer's is very different from heart disease and high blood pressure, which is different from diabetes, which is different from cancer, but really there can be sort of an underlying unifying mechanism that at least makes them more likely to happen, but yet here is the disconnect between having the evidence to say that's the case and the medical community sort of accepting that.

So in your assessment... you're not a physician, which probably gives you an advantage in this setting, why do you think there's a disconnect there?

Ben: Yeah, what an excellent point. And in fact, Bret, it's an interesting thing to be this the PhD biomedical scientist claiming to have insight into clinical practice when I have actually no experience in the clinic. And my hope, as an answer to this question in all humility... the advantage that the PhD has is that the PhD scientists gets paid to ask questions and to find answers to questions.

The MD or D.O. physician does not get paid to be curious and ask questions the MD and D.O. gets paid to see patients. That is the mechanism of compensation. And so I get paid to be curious so I can just kick my feet up, look out the window and ask, well, how does insulin resistance cause hypertension? And I can go in and find the five distinct mechanisms that explains this connection.

But it's not fair for me to say, well, an MD... you should know this. I'd be talking to my doctor, my physician friends and say well, of course, you should know this. No, they shouldn't because that's not their job. They don't get paid to be curious. And I don't mean that in a derogatory way. The mechanism is simply... you get paid to see patients, so the hospital would say, see your patients.

And if the physicians were to say, I'm going to take an hour off right now and just do some pub med searches and find some answers to some biomedical questions, that's not going to get them very far. And so I think the disconnect, to answer the question, is maybe in fact is reflected in what you said earlier where there's knowledge gaps...

I don't remember exactly what you said, 50% of what we think we know will be found to be irrelevant or outright wrong in a few years from now and I think it's happening. I think that there is a growing awareness.

And this is my central thesis of the book and even my professional goals... it is to help people understand that... to varying degrees, not 100% probably in any situation... to varying degrees insulin resistance is a fundamental part of most chronic diseases and the sooner we acknowledge its role the sooner we can detect it as a problem and perhaps as a causal problem to whatever the disorders we have in mind and the better we can treat it.

And that is most explicitly evidenced in type 2 diabetes which isn't unsurprising because that's the disease that is most explicitly a consequence of insulin resistance. But our ongoing obsession with glucose means that over the years of the patient's life, their insulin is climbing, climbing, climbing, reflective of the insulin resistance, but it's enough to keep the glucose in check. So because conventional medicine is only looking at the glucose in the context of metabolic health, the hyperinsulinemia is missed.

And it is only 10 or 20 years later when the bodies become so resistant to its own insulin, that now the glucose is climbing and then we detect the problem. So if we shift the paradigm away from glucose onto insulin, we first of all detect the problem sooner. Because we can detect the elevated insulin much, much sooner before the glucose starts to climb.

And then second by looking at the insulin we treat the problem better especially in the context of type 2 diabetes, the patient would come in, they've had years of hyperinsulinemia and now they have hyperglycemia. And the typical physician might say, we just need to lower that glucose by any means even to the point of pushing the insulin up even higher, because there's no regard for the insulin, there is no respect. Insulin is like Rodney Dangerfield that gets no respect.

So we push the insulin up even higher and it works; it lowers the glucose. But a meta-analysis published just a few years ago that evaluated all available data on insulin treated type 2 diabetics, found no evidence of improvements in any clinical outcome. In fact they found evidence for adverse events. And what tends to happen-- in fact we know... the more insulin we're giving to a type 2 diabetic, when we give them insulin, their risk of dying from heart disease triples and the risk of cancer mortality doubles.

And they typically gain about 10 to 20 pounds within just the first six months. And so when we put a type 2 diabetic on insulin therapy pushing their already hyperinsulinemic state to super physiological insulin, we kill them faster and make them fatter. But, Bret, there's so much... unfortunately there's tricky language that is used to describe type 2 diabetes because people will say despite the hyperinsulinemia, once they start to see the glucose climbing the wording is clever.

They will say insulin becomes deficient or insufficient. And that's a relative term because it never goes to zero... never. I mean if it does, that's type 1 diabetes so it's the wrong problem. So insulin over the course of the person's life in type 2 diabetes, it will start normal, it will go very high and in some type 2 diabetics it will come down a bit.

But it's still multiples higher than it was before. And so the wording is dangerously ignorant or deliberate where they say insulin is insufficient... It is... to keep the glucose in check, even though they have a lot... there isn't enough to keep glucose, they are so insulin resistant they can't keep the glucose in check.

So then a physician is justified they feel and pushing the insulin up even higher again making them fatter and sicker because it's not a glucose disease. Because even though they have normal glucose levels, once we put them on insulin therapy, we are killing them faster. It's because it's a disease of too much insulin and that paradigm shift is essential to truly understand type 2

diabetes.

And to varying degrees the same thing applies for other disorders like polycystic ovarian syndrome, or Alzheimer's, or fatty liver disease and so on.

Bret: That was a very passionate and strong statement about the role of insulin in type 2 diabetes. I think that's such an important point you made though. The way the wording is used is so important because it totally changes your mindset and the framework. And let's be honest; insulin is lifesaving. If your blood sugar is too high, insulin can be lifesaving. But it kind of goes on this concept, some is good, more must be even better. Like if we can use a little bit, then why can't we use a lot?

I mean for a long time it was kind of unknown that these higher levels of insulin were harmful and it was thought that was just because your risk of hypoglycemia is higher. But it wasn't the thought that just having too much insulin stick around for so long itself was harmful. And I think that's where the tides is sort of shifting and where your passion message is really important.

So I wanted to just get to maybe a little bit of the details about type 2 diabetes because there's also this concept of beta cell failure where the beta cells and the pancreas... some people call burnt out diabetes, where the pancreas can't keep up with the insulin production and so that's where the words of being insufficient are.

But isn't there a point where the pancreas literally can't put out enough insulin even to match sort of normal physiologic levels for people who... even their blood sugar was under good control? Or are you saying that doesn't happen?

Ben: Yeah, well this is... I want to be cautious here, because I think I know the sum of all evidence, but I'm a humble scientist. So to my knowledge and if it's actually type 2 diabetes not an autoimmune destruction of the beta cells namely type 1, the beta cells are never gone in a type 2.

And in fact there is instances to show that as insulin production might have dropped in type 2 diabetes, there's beta cell reversal with type 2 diabetes, as the person incorporates dietary changes such as fasting for example. They note what they call beta cell reversal. In fact that's how some of the manuscript titles, that's some of the terms used in these manuscript titles, this reversal of beta cell loss.

So it is not this irreversible destruction of beta cells like we have in true type 1 diabetes. It might be a reduction in insulin production where it starts to slow back down, but to my knowledge it is never gone. We've never started this to wage war on the beta cells, insulin production might've gone down... And I don't know the reasons for that but I would say that's fine. Lower your glucose load coming into the body, so that lower level of insulin production is now more than sufficient to meet the glucose load coming in.

And so I can't speak to the reasons why, in fact I'm not sure anyone can, why is insulin coming down, why is it sloping downwards in some, not all, in some type 2 diabetics, which is amplifying or accelerating the hyperglycemia. I don't know why. I would say it does not get to zero, not even close. It stays higher than it was before. So if that's a problem it's only a problem insofar as the person is continuing to probably demand a high insulin load by consuming starches and sugars.

Bret: Rights, so here now we get the sort of the cause of insulin resistance and hyperinsulinemia. So is it too simplistic just to say sugar is the cause, carbohydrates are the cause? I mean

that's a lot of what you see online. Now, there's part truth in that, or maybe oversimplification? So tell us about that.

Ben: Yeah, so that's a great question. And I'm glad to share my insight into this because it might not align with others in the low-carb space. I believe that there are primary and secondary causes of insulin resistance and it is not as simple as just carbohydrate consumption and people will want to invoke the Kitavans and say, well, these are people who ate a high carbohydrate diet and had no instance of insulin resistance and type 2 diabetes.

I think that's true, I think that's accurate, that's fair to note. It's also I think accurate to note that we don't eat carbohydrates the same way more sort of ancestral diet cultures did. If we were all only getting our carbohydrates from tuberous vegetables, I don't think it would be a problem. Even if we're getting our carbohydrates from just baked potatoes. I don't think that's going to cause insulin resistance in an otherwise healthy person.

So I wouldn't want someone to think I'm claiming it's that simple, but of the primary causes, I call these primary, because if I'm actually growing... because there's evidence to show that in isolated cell cultures, the three causes I'll mention in a moment will directly cause insulin resistance.

And also in human studies even, not to mention rodent studies, all three of these will directly cause insulin resistance rapidly. So maybe I'll mention them in sort of inverse relevance. So the first one would be perhaps stress where we know the cortisol and the catecholamines are insulin antagonists.

So if cortisol is elevated over a long term including artificial cortisol like through prednisone or dexamethasone these cortisol analogs, to control inflammation, which will be my next point, but if cortisol is elevated, the body will become insulin resistant rapidly. And we see this in humans, we see this in animals and in cells. So it's just reflective of the insulin antagonistic actions of the stress hormones.

And this can be from something even as seemingly benign as sleep deprivation, which causes an acute cortisol spike which is likely part of the reason why the body is insulin resistant the following day. So it's a very acute phenomenon. The next cause is inflammation and we see this in autoimmune diseases and in acute illnesses and infections.

The body becomes insulin resistant when inflammation is up and we can introduce pro-inflammatory cytokines into people through infusions and into rodents and sure enough we cause insulin resistance immediately. And the last one is one that some don't agree with, but the evidence is simply too overwhelming, including some from my own lab, which is hyperinsulinemia itself.

And this is where carbohydrates would come into play. The chronic consumption and frequent consumption of high loads of refined starches and sugars which would basically ensure that a person has elevated insulin every waking moment of the day. And so hyperinsulinemia itself is a cause of insulin resistance and this can be done in isolated cell cultures and in human and rodent models.

So even at physiological levels as this has been done in human studies, it's not like pushing their insulin up to super physiological doses is all what you have to do... it doesn't have to be that extreme. So those are the three what I consider primary. And then the third or the fourth rather I call it a secondary... not to diminish its relevance, but because I think it's more of a multi-cell

phenomenon overall in the whole body and that is the excessive consumption of linoleic acid, as I outline this in the book.

But linoleic acid is one, the most commonly consumed fat in the human diet now, which ancestrally it never was, although we always ate some amounts of it because it exists in natural fats as well, but now that we get most of our fat from soybean oil and corn oil and canola oil, we're getting much, much higher, thousands of times more of this fat than we ever did in human history.

And linoleic acid makes fat cells insulin resistant by forcing hypertrophic growth rather than hyperplastic growth. So I am getting into the weeds a little bit here, but suffice it to say that as linoleic acid is altering the growth of fat cells making them insulin resistant, that insulin resistance spills into other cells subsequently.

Now I don't know, I can say that in muscle cells when I... even fat cells in an acute term, if I incubate them with linoleic acid they do not become insulin resistant as acutely as the primary causes, or what I am calling primary causes.

So, I'm making some assumptions here that I don't need to make with the primary causes. We know that linoleic acid affects the growth of fat cells in a way that would make them insulin resistant and when fat cells become insulin resistant I consider it the first domino to fall.

Bret: Yeah, I think that's really interesting and I like how you are sort of careful about how strongly you say that and how you label it as secondary rather than primary, because there is sort of a difference in the level of association there. And we have to admit, in humans doing any type of nutritional study and trying to boil it down to one food group or one type of food is so hard, because we don't eat one type of food; we eat a diet and we eat a certain amount of calories.

And the effects something may have in a hypocaloric diet versus a caloric excess diet may be totally different how that one macronutrient or that one specific type of fat affects you. Whereas in the cell culture though, totally different. Because there you have complete control.

And I think that's something that's nice about what you bring to this picture because you do have knowledge of both sides of the story, the sort of the nutritional epidemiology and the studies that you do and therefore you can even better interpret. So I think that's well said.

Now you did mention about evolution and about how we ate in evolution and I want to touch on that, but also a briefly touch on what you call your "plagues of prosperity", which is a term I've heard you using before which I really enjoy, because again it comes down to the hyperinsulinemia and insulin resistance and may have to do with what we eat, but it also has to do with how much and when and how much we move our bodies and how our sleep is and how our stress is and the things you mentioned as the causes.

So I assume when you talk about "the plagues of prosperity" is not just food, right? Just like the entire lifestyle.

Ben: Oh yeah, I think so. I do love alliteration. So the "plagues of prosperity" was just my attempt to sound clever and yet try to be accurate. Which was my attempt to highlight the relevance of our modern environment and its role in causing diseases that our ancestors would have had no concerns with.

Now cancer is a more sort of a primordial disease that I think has always happened but the like heart disease and diabetes and Alzheimer's disease, the rate at which we're seeing these, I think it's safe to say it's unheard of in human history. And so yeah I do think I consider the biggest problem to be the type and frequency of eating.

The type of foods we are eating and the frequency with which we're eating them and namely that these are foods that are high carbohydrate and high-fat. And that is not something that happens much in nature. In fact to my knowledge the only real source of foods that would've been high carb and high-fat would've been breast milk. It's not something that we get much of or mammalian milk. Other than that, carbohydrates is really its own class of food. And rather other foods come protein and fat together.

Bret: Yeah, I think that is a great point about... so many things in modern society have changed and one of the biggest one is that combination of carbs and fat. And so when it comes to the etiology of insulin resistance a lot of the science that has been done show that it's an increased accumulation of fat within the muscle cells. A

n increased accumulation specifically they say of saturated fat within the cells which leads many to think, okay, eating fat must therefore cause insulin resistance. So how does that play into what you just said about the combination of carbs and fat?

Ben: Yeah, I love that you're bringing this up partly because I've actually explicitly published papers on the effects of saturated and unsaturated fats on insulin resistance. And so it's something I can speak to, with, you know, a fair degree of authority. So fat, it depends on the fat, whether it's relevant or not. So for example if a muscle is taking in saturated fat and it's using that to turn it into triglycerides, that is totally inert.

So triglycerides have no effect on muscle insulin sensitivity. And we see this very well. It's very well confirmed and refuted, the idea that triglycerides are contributing to insulin resistance even if the triglycerides are made up of saturated fats, as part of their fats. But however there is some truth to this idea that fat is contributing to insulin resistance and once again it depends on the type...

And by that-- Or now I guess it's the appropriate time to mention a molecule called ceramide, where ceramides are a type of fat that a cell will make in response to inflammation, in response to cortisol and in response to chronically elevated insulin. And it will take a saturated fat, palmitate, which is the most abundant saturated fat... even if we are eating zero fat, the saturated fat palmitate will still be the most abundant fat in the blood because of the liver producing palmitate.

When insulin is high the liver is... its production of palmitate will be the main contributor to the saturated fat in the blood. So regardless, even the muscle cell will take a palmitate molecule and will take an amino acid and it will start to create ceramides. And ceramides are known disruptors to the insulin cascade, to the biochemical events that are mediating the insulin pathway.

So when people are citing or mentioning that the reason-- many plant-based advocates will do this. They'll say saturated fat causes insulin resistance and so that's a reason to avoid any sources of saturated fat.

And I hear that and think that is a person who has taken an ounce of the truth, including some of what I have published myself, finding that saturated fat is necessary for the backbone of ceramides, but they then confuse the issue by claiming that it's the dietary saturated fat that's

necessary in that event when in reality someone can be eating no dietary saturated fat and still be producing ceramides from the saturated fat that the liver is pumping out very readily from glucose and insulin.

Bret: Yeah and then there are also studies showing that if you measure blood levels of saturated fats it doesn't necessarily correlate with the saturated fats you're eating. So along those same lines.

Ben: Yeah, that's exactly right.

Bret: And I think that gets back to the point also what I mentioned earlier about, how the number of calories you eat and like you're saying the combination of carbs and fat, that creates a whole different atmosphere that may or may not make it more likely for fat get deposited in the cells and trigger insulin resistance. So, it goes far beyond just one type of food, which I think is so important.

Okay, but now let's get into this concept of physiologic insulin resistance because when someone eats low-carb, and we're going to get into ways to combat insulin resistance in a little bit, but one of those happens to be eating low-carb.

So people claim that when you are eating low-carb you do have some signs of insulin resistance physiologically, but that it's a good thing because it's physiologic insulin resistance and not associated with the elevated insulin levels. But now I think it sounds like you've got some issues with that, at least with the definitions of it, so I'd love to hear your take on it.

Ben: Yeah, I do. I think that the glucose intolerance, that's the term I want to use... the glucose intolerance that is manifested in long-term adherence to a low-carb diet doesn't fit any description of insulin resistance. There is neither actual insulin resistance at the muscle and there is also no hyperinsulinemia.

And me mentioning this is based on... well, the first observation is that insulin goes exquisitely low in someone who's adhering to a low-carb diet. So we knock off the hyperinsulinemia immediately. But someone could then return and say, but still now you've just gone to the fundamental insulin resistance at the muscle cells, which is why the long-term low-carb adherent eats the bagel now and now their glucose goes higher than it did before they ever went low-carb. But that's not true.

And George Cahill decades ago found this phenomenon where he was fasting people for days, you know, weeks and that is low-carb... I mean that's fasting, they are very much ketogenic, very much in ketosis. And when they injected them with insulin, these human patients with insulin, their glucose dropped immediately.

They had an immediate and exquisite response to the insulin suggesting that this long-term ketogenic state was not causing any insulin resistance. So I think by any definition the body is not becoming insulin resistant in response to adaptation to a low-carb diet. I do however think what we see is for lack of a better term, a reverse or an inverse metabolic inflexibility. And now you probably already know where I'm going with this, but in typical metabolic inflexibility or... Let me even start more basic.

A healthy person is metabolically flexible where they eat a mixed macronutrient meal and they go into sugar burning mode, you know, blood sugar, blood glucose. And then they fast for a few

hours later and they shift over to relying more on fat for fuel. So that's a metabolic flexibility. People with insulin resistance because of the chronically elevated insulin are stuck in sugar burning mode even when they are fasting.

So it's been hours or a day later and they are still burning more glucose than you would've seen in a healthy person who would've shifted the fat burning. So it's like they're stuck in glucose burning mode unable to burn the fat as well as a primary fuel. Long-term adherence to low-carb diet I believe puts us in the reverse situation.

It causes this reverse metabolic inflexibility where the person has shifted so strongly to fat burning that now when we load the system with glucose we have a bit of a delayed glucose burning and I cannot speak to the mechanisms. I mean it doesn't change the reality that we do see that they are compromised and shifting back to glucose, so I think it's fair to create this term of reverse metabolic inflexibility.

But I cannot speak to the mechanism. I don't know why it would be happening. Because we know that they are exquisitely sensitive to insulin when you give them an insulin load. We've done this in animal models where they've been adhering to a low-carb diet. Give them an insulin load and their glucose drops.

They are very, very insulin sensitive. So I don't think it's fair to mention insulin resistance in any way. I think it's more accurate to either say that they have a glucose intolerance, an acute glucose intolerance, or reverse metabolic inflexibility. And a lot of this is probably just semantics, people when they'll be hearing me and they say, that's what I meant when I said insulin resistance... but if so then it's too inaccurate.

We cannot be mentioning insulin resistance when the two fundamental sides of the insulin resistance coin are nonexistent in long-term adherence to a low-carb diet.

Bret: Yeah, I think that's a good point because then you are saying the solution sounds a lot like the problem, which can get really confusing when you're still using that term insulin resistance, so I think that's a good point to eliminate that. Even if we are still talking about the same thing. And there's some theory that it's an evolutionarily beneficial thing to have this glucose intolerance, adaptive glucose sparing, whatever you want to call it.

Ben: Yeah. Both good terms.

Bret: Some people call it physiologic. Because if your muscles are using fatty acids, then you want to save the glucose for your brain, so you don't want your muscles to be burning the glucose. So they are resistant to glucose not resistant to insulin you could say. So it's like a lot of things, it's a potentially beneficial adaptive mechanism for our ancestors that our modern lifestyle has turned into a detriment and contributed to a lot of chronic diseases.

Ben: Yeah, well said.

Bret: So, now that we've laid the groundwork for how pervasive a problem it is and how significant of a problem it is, you said, I want to get the quote right, "How we live can both be the culprit and the cure for insulin resistance". So it's, you know, not all grand, we can do something about this and it's actually probably not all that complicated to do something about it.

And one of the first things you talk about in your book is exercise and the importance of exercising. And we hear time and time again now that you can't outrun a bad diet and exercise by itself

isn't the best thing for weight loss, but could exercise by itself be a major contributor to healing hyperinsulinemia and insulin resistance?

Ben: Oh yeah, I think it could. I do think that the person would be still ignoring what I would contend as the more relevant variable. But no question the advantage of exercise that that cycling of contraction and relaxation will start pulling in glucose independent of insulin.

So, you can take a profoundly insulin resistant quadriceps and you start contracting it and it will engage those glucose transporters that will pull in glucose that normally insulin must be present to engage but independent of insulin. And so it creates an immediate mechanism to lower the glucose following the exercise and thereby allowing the insulin to come down. And the moment the insulin comes down, insulin sensitivity starts to improve.

So yeah, I'm a huge advocate of exercise there's no question. It can have a direct effect on improving insulin resistance. However just to highlight the relevance of diet, not to diminish the exercise, because that was the point of this bit of the conversation, there's a study that found that when you finish exercise, and I mentioned this in the book, if you finish an exercise session with a carbohydrate rich meal, you undo the insulin sensitizing effect of the exercise.

And that's tragic because most people will go to the gym, they get done the gym and then they go get a smoothie or they drink a bottle of Gatorade because they're told... you are training for a marathon, you got to eat all these carbs to train for the marathon. And they might be able to finish the marathon but paradoxically they've gained weight during the course of training for it and that shouldn't be happening.

Bret: Yeah, I can tell you how many workouts in the 80s and 90s training for triathlons I finished by a smoothie. When you said it, I was, "Oh, I did that". You have this concept you're doing something good for yourself. I definitely want to get into the nutrition because it's such a big part, but exercise, I liked how you covered exercise in the book, especially when you said friends don't let friends skip leg days, that was great.

So this concept of resistance training, because people want the minimum effective dose. If I only have limited amount of time where am I going to get the biggest bang for the buck and in the book you made a case for resistance training. But for a lot of people, they don't know how to get started maybe with resistance training, or the concept of exercise to failure.

What does that mean? What does that feel like? How we do it? So what kind of advice would you give somebody about the best exercise and the best method of exercise to combat insulin resistance?

Ben: Yeah, that's great. Mentioning the minimum effective dose that is the perfect way to describe it, how can we maximize the benefit. In answering this question I would say that my sentiment on what is the best exercise, really is the one you'll do. So if someone hears us talking about doing resistance exercise and this older lady is thinking, "I'm not going to do that.

And so I may as well not do anything." No, if your exercise is going to be going on a walk, then go out on a walk. Insofar as a person is willing and able to incorporate some resistance, it could be something as simple as sitting in and out of a chair, just while you're watching a show... you're watching a show in the evening just sit and get up and out of your chair repeatedly, just keep

going until the muscles are uncomfortable.

If a person is comfortable getting down on the ground just do even knee push-ups. Not even doing push-ups, but just holding a position until it starts to get hard and the muscle start to burn. That is a good enough starting point. Because if we can give that stimulus to the muscle that we are doing something hard and we need you to grow, the bigger the muscles get the more easily we are disposing of glucose and clearing it from the blood, helping lower glucose, helping lower insulin and we're addressing the problem.

Bret: Yeah, I think that's a great point, that this sort of vision of pumping big iron in a gym can be a definite detriment to people because they think, I can't do that so why get started. That was a great example. But then, again you hear this term like exercise to failure. That's sort of like that's what you need to do, when a lot of people maybe don't understand what that even means.

Ben: Yes so that was... when I mentioned in my answer a moment ago just kind of go until the muscles are burning, in some people that would be failure and that would be at least the first version of what failure would look like. Like oh, my muscles are getting uncomfortable. That might be enough for the first several days and then eventually when I say going to failure it's this idea that now you're doing those chair squats until you just can't get back up.

You know, you kind of went down and you're trying to get back up and that was the end of it. And that would look the same in push-ups. You're doing those knee push-ups on the floor during commercial breaks or something, you've done them until you're pushing, pushing, you can't do another one... Ah, you flop to the ground.

That's what failure would be when you cannot repeat the activity again. And that's a mentally exhausting way to work out. So I do mean what I said a moment ago where a person doesn't start with that, they go to the point where it's just a little uncomfortable, the next day they go a little further and before they know it at any age, they find that they can go to failure. And that's when you really will start to get more substantial muscle gains.

Bret: Yeah, I think that's great. And also in the book you really emphasize compound movements and using the larger muscle groups and I think that's important. It's sort of its own topic of how to best exercise for insulin resistance. But you covered it very well.

But like you alluded to, if you just talk about exercise you're probably ignoring the bigger factor which is nutrition. And again it'll be easy to say that low-carb and keto diets are very good diets for reversing insulin resistance. That is I think true and very well proven in the literature. But is that the only way?

Ben: Excellent, it is certainly not the only way. And even someone... It little pains me to say this because I think the diet is incompatible with human survival, but someone could even go on a plant-based vegan diet... What I'll say is anything that departs from the typical standard American diet is going to improve insulin resistance. And that is why we do have human evidence to suggest if you take people and put them onto a low-fat plant-based diet, their insulin resistance will get better.

Absolutely, there are studies out there to confirm that. Independent of monitoring any macronutrients, intermittent fasting has been shown conclusively to also improve insulin resistance, so that's one that is totally disconnected from any kind of macronutrient manipulation. You don't even have to worry about your macros. You're just fasting for these periods of time.

But then what I think what led me to be more of an advocate of a low-carb diet when it comes to insulin resistance is the studies that compared directly the low-fat diet, that if you take a person on a standard American diet, put them on a low-fat diet it gets better. True enough. But what happens when you compare the low-fat diet with the low-carb diet? The low-carb diet will outperform the low-fat diet.

And that to me was the eye-opening realization. It's a handful of years ago when I was... my view on insulin resistance was clarifying to the point I was more acknowledging the role of insulin itself as a cause of insulin resistance. Indeed as a result partly of my own experiments in my own lab, my evolution then became... well, if I'm finding that insulin is part of the cause of insulin resistance what is the best way to lower insulin?

And of course it's an easy step you say, well it's cutting the one macronutrient or controlling the one macronutrient that actually elicits a substantial insulin spike and that is carbohydrates. And indeed like I mentioned a moment ago it bears out, it plays out this way in the clinical studies. To my knowledge of all the dozens of studies that had put humans onto a low-fat diet or low-carb diet not one has ever shown a significant improvement in the low-fat diet beyond low-carb, not one.

And in contrast there are dozens that have shown significant benefits beyond the low-fat diet from the low-carb diet. So there's a clear winner there in my mind. I shouldn't say my mind, I shouldn't qualify it like that. The evidence is clear, a low-carb diet will outperform a low-fat diet.

But again, to be diplomatic and scientifically objective, there are numerous interventions that will improve insulin resistance when a person is starting from a baseline of the standard American diet.

Bret: Yeah, I think that's very well said. It summarizes the literature, and it's open and honest about the different ways to do it. So, each person has to find their path that is going to be most sustainable for them, most enjoyable for them that is also going to impact the insulin resistance.

And if you talk head-to-head, if the low-carb diet is so much better than the low-fat diet, but you love a low-fat plant-based diet and that's the diet you're gonna stick to, okay, you're probably going to do better off than that, than you otherwise would have following a standard American diet.

And then there's still other things you can do like we talked about with exercise. And then you talked about the "plagues of prosperity" and the sleep and the stress, focusing on these things. So we have pretty good science to back up by giving attention to your stress, paying attention to your sleep and improving those will independently improve insulin resistance as well?

Ben: No, I have not seen any study that has ever done that. So we only have the inverse, showing that sleep deprivation and stress is causing insulin resistance. But taking someone who is insulin resistant and then the intervention is improving their sleep, does that improve insulin resistance independent of any other variable... I've never seen that paper if it's out there.

Bret: Yeah, and that's interesting, but it's also the "what's the harm". What's the harm of paying attention to your stress and paying attention to your sleep. It's only going to have other proven potential benefits from blood pressure and anxiety and depression and other downstream effects. So, one of those things you probably should do anyway and it's likely going to affect insulin resistance as well.

Ben: Agreed, yeah.

Bret: So at one point you also had mentioned fasting and time restricted eating, which is another really interesting topic that we're sort of I think on the threshold of just an explosion of scientific evidence of what's going to come for this. But again it comes back to that question of what is the minimal effective dose. Which I know, we don't know the answer to, proven scientifically, but I really want to get your opinion.

What you think, where you think that threshold is for fasting or time restricted eating that's really going to make a difference that is worth spending the time. Because when we talk about, look, trying to focus on your sleep is a good thing because it's going to make you feel better and it's got positive outcomes... fasting, let's be honest, is not always that much fun.

Especially if you're talking about a three, four, five day fast. So where is that bar for you that you think people should aim for?

Ben: Yeah, that's... what a great question! I think firstly it doesn't appear... It appears that there are multiple good right ways to do it and I say that because if I recall in one of Jason Fung's recently published papers looking at intermittent fasting, these case studies he published, I think the patients were using multiple forms of intermittent fasting including like the five days eating, two days fasting, one day eating, one day fasting, so I think I believe if I'm remembering that study correctly they found significant dramatic improvements in insulin sensitivity in people with type 2 diabetes that were using multiple strategies.

So I don't know that there's anyone's strategy that is necessarily much better than another, so that once again might be an answer of find the one that works for you. But the minimum effective dose... I can only speculate. And I would speculate that I think there's probably something around that 15 to 18 hour window where insulin does come down and now the person is getting into ketosis.

And I don't say that because of the ketones, that the person needs to be getting ketones in their blood. I say that because I think that represents a shift in fuel use that if someone has been fasting for around 15 to 18 hours or so then they are likely getting into the first sort of hints of ketosis and that itself is reflective of this energetic shift where we have shifted from sugar burning into fat burning which itself is a function of insulin having lowered because insulin dictates which fuel is being used.

So I'm speculating a little and so there might be some wisdom to the 18-6, which is a pretty common time restricted eating protocol, but if someone gets closer and closer to one meal a day fasting, my fear starts to grow that during that eating window they're going bonkers.

And in some people that version of time restricted eating where it's like a one meal a day or just a few hours in a day... I say this because I've seen it happen... where it basically turns into a time restricted binge purge cycle, where they start to eat in that window and like I said they go bonkers.

They stuff themselves with these terrible foods and they are eating all of this right before they're going to bed and now they sleep terribly, their body temperature is higher, their glucose is higher, they're digesting the food, they are comfortably, they sleep terrible, they regret it, they have remorse and they say, I'm going to do it again and I'm going to do it better tomorrow.

So they fast through breakfast, they fast through lunch. And there's this sort of punitive response

to the binge the night before and they do it all over again. And so I think it's so important... I think intermittent fasting is very effective, but the person must have a plan with how they end the fast.

They need to have a very well structured and very disciplined... to not let it become a binge purge cycle and sort of a glorified version of an eating disorder. So the meal, how a person ends a fast is probably more important or at least as important as how long they are fasting.

Bret: Wow, so well said! Yeah, because I have seen it time and time again even with two meals a day. If someone was eating 1500 cal and three meals a day, they go to two meals a day and the psychology of it is all of a sudden they are eating 2200 cal in those two meals and you're not doing yourself any favors.

And you're describing it in an even more extreme example of that. And so well said... a plan... you have to have a plan of how you're going to fast and break the fast. And I think that's a great way to just sort of wrap it up because it really does show the way just... I really enjoyed talking to you because you're so passionate, you're so intelligent, but you're also very measured and you see things from different perspectives and you want to be clear about what we know and what we don't and that's why I think your voice is such an important voice to listen to and why I think your book is a fantastic book for that same reason.

And so this is my way again of saying thank you for coming on and thanking for all you're doing. And since your biography has been growing and you're involved in so many different things, now, where can people turn, because you have so much more knowledge so much more information that I want people to be able to learn from, where can they learn more from you and hear more about you?

Ben: Well, Bret, seriously thanks again, what a fun opportunity to reconnect now that we can't go to in person conferences these days I don't get to see my buddies in the community. So, it's great to connect with you. I am moderately involved in social media and I would want people to know at the outset is never pictures of me doing things.

When I first got on social media about four years ago it was simply to overcome the inherent hurdle in research where I would make a cool finding, I publish it in a paper and no one ever reads the paper. It just sort of slips into irrelevance. Is the curse of the scientists. So I got involved in social media simply and totally as a vehicle to share a metabolic insight.

And some of which I highlight in the book and so of course you mentioned the book. Anyone who is curious about it, go get a copy anywhere books are sold, that's "Why We Get Sick". But social media I'm mostly involved on Instagram these days and my handle there is BenBikmanPhD.

I'm still somewhat active on Twitter but I find that I'm sort of retreating from Twitter more and more as it's just becoming a very different place and not a place I care to be in too often these days. So also I'm involved with a low-carb coaching platform, called Insulin IQ and we've developed a very good program online. This is simply just a coaching platform.

Anyone curious about this if you find like you need help... in fact we're partnering with Diet Doctor to help give content here, but they can learn more on Insuliniq.com And we have fun live sessions each week where we talk about research and answer questions. And I'm also involved in the development of a low-carb shake.

So as anyone knows once you go low-carb one of the beauties of that is that you're forced to eat

more real food because there aren't a lot of convenient low-carb options that are well-built. So, this was an effort for me to only for my own sake make low-carb a little more convenient and that was the development of a low-carb high-fat high protein shake. And I think it's the best out there. Anyone who wants to learn more I won't elaborate more here go to gethlth, and health is spelled H-L-T-H gethlth.com to learn more.

And I provide blog posts there and eventually some video content as well. So, that's how to connect with me and I love connection, guys. Come find me on social media, ask me questions. It's a fun way to connect with people and a very genuine sort of transfer of information. If I know something that someone deems beneficial I'm happy to share.

Bret: Wonderful, wonderful. I highly recommend people look you up and all you are doing and thank you again for all your work and your contribution and thank you for your voice.

Ben: My pleasure. Thanks again, Bret.