EP\_1252\_BENJAMIN\_BIKMAN\_(AUDIO)

**Benjamin:** [00:00:00] 70 percent of

**Dave:** all

**Benjamin:** calories consumed globally come from starches and sugars. And so the average [00:00:05] individual, because they've been told, is eating five or six times a day. It's all carbohydrate heavy. [00:00:10]

**Dave:** My biggest critics online are the dietetics. And I'm just like, guys, you've done a shitty job. [00:00:15] Look at the health of the country and look at people who eat hospital food.

Stop talking.

**Benjamin:** To the average person who wants to [00:00:20] lose weight, the traditional view is cut your calories. But if you're not addressing your insulin, that's just going to [00:00:25] promote hunger. Hunger always wins. Before you know it, you'll be right back where you were. Without insulin, you die.

**Dave:** I. [00:00:30] I've read studies saying that low insulin is four times [00:00:35] more deadly than high insulin.

If you do some research about insulin and Alzheimer's, you'd want to [00:00:40] be on a low carb diet, but you might want a little squirt of insulin every now and then in your brain because it seems to [00:00:45] help. What concerns me is if you take a little bit of insulin and you put it in a nasal [00:00:50] spray and you spray it up your nose, it's a way of treating Alzheimer's, so why is it when I [00:00:55] raise insulin my brain works better?

You're listening to the. Human upgrade with Dave [00:01:00] Asprey.

Today, we [00:01:05] are going to have an amazing conversation with Dr. [00:01:10] Benjamin Beekman or Ben Beekman, who is an absolute boss when it [00:01:15] comes to bioenergetics, metabolism, and the way the body actually [00:01:20] works. And as you might know, I kind of have a mitochondrial fetish I developed in the [00:01:25] late nineties because I had chronic fatigue syndrome and fibromyalgia and brain fog.

[00:01:30] And What 300 pounds of weight and I learned from people in their [00:01:35] 70s and 80s who are running a longevity nonprofit group and soon they asked me to run it. I'm the [00:01:40] only guy under 50. So mitochondria have been at the very center of biohacking. It's like you change the [00:01:45] environment around you. So you have control of your biology because your mitochondria listen to the environment.

[00:01:50] So now we have Dr. Beekman here to talk about what's really going on in there. [00:01:55] He's a fantastic teacher, very, very learned human being. And I'm [00:02:00] excited to share his wisdom with you today. Ben, welcome to the show. [00:02:05]

**Benjamin:** Hey, Dave, thanks so much. It's nice to be with you again. This is great. This is our second or third [00:02:10] interview.

I think it's our third. I think it's our third too.

**Dave:** Your YouTube channel, the stuff I've seen [00:02:15] from you lately on Instagram is so good. You're one of the few people in academia [00:02:20] who's just willing to say, Well, here's what the evidence says, even though it's not popular. And I just [00:02:25] so appreciate that. Do you take a lot of hits?

Like, do you have colleagues that are like, no, cholesterol's bad, [00:02:30] and then they're like, trying to not have lunch with you and eat vegan crap and

**Benjamin:** all that? Yeah, yeah, well, that's a [00:02:35] funny little kind of side comment, but yeah, I mean, one of the most sobering moments of my career came as a pre [00:02:40] tenured professor when I had a bunch of other professors try to get me fired from [00:02:45] the dietetics department, actually, in particular.

I was deemed a heretic of [00:02:50] sufficient vileness that they tried to get me removed. So yeah, it's, there have [00:02:55] been some naysayers, no doubt. Yeah.

**Dave:** Well, thank you for fighting the good fight. [00:03:00] And if I can just say, there are a very few number of functional dietitians. [00:03:05] All of the other dietetics or dietitians, they are the McNuggets in [00:03:10] hospitals people.

They're the school lunch people. And they are so brainwashed, or maybe just [00:03:15] downright evil, I'm not sure. They're the ones saying put, you know, corn syrup and [00:03:20] canola oil in baby formula. And it's. It's just not how you do [00:03:25] it, by the way. I'm not saying that that is in all the AP formula, but certainly around the world.

It is. And just that my [00:03:30] biggest critics online are like the dietetics, the dietitians people, and [00:03:35] I'm just like, guys, you've done a shitty job. Look at the health of the country and look at people who eat hospital food. [00:03:40] Stop talking. And then I just move on. Do you do the same thing? Or are you more polite?

Well.

**Benjamin:** [00:03:45] No, I think I, I mean, I definitely have to be a little diplomatic depending on who I'm talking with, but I [00:03:50] agree generally that while there are certainly dieticians and these licensed [00:03:55] individuals who challenged the, the prevailing thought, it is probably the most [00:04:00] dogmatic field I've ever encountered that, and if you [00:04:05] deem yourself worthy to step into that territory, you need to be [00:04:10] ready for the fiery darts because they come after you.

**Dave:** Well, they're very well funded by Big Pharma [00:04:15] and Big Ag, so there, there's that. You've been, I'd say, an outspoken voice on [00:04:20] insulin resistance and how it relates to disease, and [00:04:25] you're doing it both in a public sphere with your YouTube and you're doing it in your [00:04:30] book, you know, How to Not Get Sick. So, talk about insulin resistance.

What is [00:04:35] it, and where does it come from?

**Benjamin:** Yeah, insulin resistance is best [00:04:40] defined as a A pathology with two parts, so it's a two sided coin. [00:04:45] One side of the coin is the obvious side, and many people don't appreciate that there's another, [00:04:50] but that obvious side of the coin is the fact that the hormone insulin isn't working [00:04:55] particularly well at certain places of the body.

That is the insulin [00:05:00] resistance part, that some cells of the body, not all, some cells [00:05:05] insulin's effects. Now, all of this is compounded [00:05:10] by the other side of the coin, which most people do not appreciate. But if you are invoking the [00:05:15] term insulin resistance, you're invoking two parts, the one I just described, but then the [00:05:20] other, which most people overlook, which is hyperinsulinemia, or high blood insulin.

[00:05:25] There is no separating the two. That it does not matter if insulin [00:05:30] resistance exists in the organism, insulin levels are higher. The two [00:05:35] ideas, they are inseparable. So that is insulin resistance. And of course the [00:05:40] relevance of it is just the fact that it contributes to, well not only because it's so [00:05:45] common worldwide, it's the most common health disorder, but it's so [00:05:50] connected, and even in a causal way, to virtually every chronic disease.

All of these [00:05:55] What I like to call plagues of prosperity, while they do have individual noxious [00:06:00] stimuli that can cause them, they have a common one as well. And so the drum that I [00:06:05] try to beat so loudly is that there is a common origin to so many of these problems and it's [00:06:10] insulin resistance. So rather than.

Chasing these into [00:06:15] putting all of our efforts into chasing individual stimuli. Let's really put most [00:06:20] of the effort into addressing the most common thread that if we can pull that one [00:06:25] out, everything else just starts to crumble.

**Dave:** The number one cause of insulin resistance [00:06:30] is

**Benjamin:** Yeah, number one cause. So, more and more, Dave, as I've thought about [00:06:35] insulin resistance, which I do a lot, I've, I've come to the conclusion that there are [00:06:40] two paths to insulin resistance.

There's the, the fast insulin resistance, [00:06:45] and then there's slow insulin resistance. And, and that's a bigger conversation that [00:06:50] I don't know that we want to get into yet. But briefly, Slow insulin resistance is the result of [00:06:55] fat cells that have undergone significant hypertrophy. So that's the connection of [00:07:00] how fat tissue explains insulin resistance.

Now, don't assume that [00:07:05] that's everyone listening, that that just means only as we get really fat and obese. Depending on [00:07:10] the ethnicity, it can be only a little bit of fat gain and it's already resulting in larger [00:07:15] fat cells that are causing insulin resistance. So that's an insulin resistance that. Settles in a [00:07:20] little more slowly, and it reverses a little more slowly.

Fast [00:07:25] insulin resistance, which is the one to, I think, most explicitly answer your question, [00:07:30] is a result of stimuli that can literally trigger insulin resistance within hours. [00:07:35] But then they can be resolved within hours to days or maybe a week or so [00:07:40] and in that case The one that I believe is theme of all of these.

What is [00:07:45] the most relevant cause of insulin resistance? It is in that fast lane. The most [00:07:50] relevant is too much insulin And and so just to really bring this full [00:07:55] circle because it does kind of create a bit of a circle. I posit that as a [00:08:00] person Is eating a chronic constant carbohydrate consumption, [00:08:05] which is the global diet, 70 percent of all calories consumed globally come [00:08:10] from starches and sugars.

And so the average individual, because they've been [00:08:15] told, is eating 5 or 6 times a day, it's all carbohydrate heavy. The average [00:08:20] individual spending. Every single waking moment in the state of elevated insulin, never giving the [00:08:25] body enough time for the insulin to come down. As insulin wants to start to come down, they've bumped it [00:08:30] back up again.

And so it's that chronic insulin. That [00:08:35] starts to make the body somewhat deaf to the insulin and and that's and [00:08:40] then it starts to feed the cycle where Too much insulin is causing insulin resistance, [00:08:45] which is in turn creating more a need for more insulin Which is creating more [00:08:50] insulin resistance.

**Dave:** I've read studies saying that low insulin is [00:08:55] four times more deadly than high insulin Is that true?

**Benjamin:** [00:09:00] That's, that's remarkable. I could not believe that to be [00:09:05] true and would only believe it in the context of type 1 diabetes.

**Music:** Mm, [00:09:10]

**Benjamin:** that might be why. If, if the population is a type 1 diabetic population, [00:09:15] then I would believe that because it would suggest the person is underdosing to the point [00:09:20] of lethality.

But in a, in a non type 1 diabetic, [00:09:25] I would Absolutely not believe those outcomes, but that's an important point, Dave, [00:09:30] because a lot of the confusion that people have with regards to even the insulinogenic effect of [00:09:35] macronutrients, like they will say fat causes an [00:09:40] insulin release and yet every single study that has ever shown [00:09:45] that was based on a type 1 diabetic population where because of their chronic [00:09:50] hyperglucagon levels, there is this sort of a a Unexpected [00:09:55] quirk of eating fat in a person with type 1 diabetes, but in a non diabetic [00:10:00] population, there's zero studies to show that fat has an effect on insulin.[00:10:05]

None. And so even in my own lab, a data, data we just have generated, we're about to [00:10:10] publish, I can state it authoritatively, dietary fat has no effect [00:10:15] on insulin, and there's never been a study that has ever shown it.

**Dave:** Thank you for saying that when [00:10:20] I write about fasting and intermittent fasting, I've [00:10:25] consistently said, if you have some butter and MCT oil and coffee or something like that, [00:10:30] it has no effect on insulin or mTOR, and A third [00:10:35] party actually went out and tested 300 different breakfast options to see their effect on blood [00:10:40] sugar and on insulin.

And coffee with butter and MCT was at the very top of the [00:10:45] list as having no effect whatsoever. That's why you can do it during a fast. True or false? [00:10:50]

**Benjamin:** Oh, Dave. Yes, you and I are completely aligned. In fact, to the, to the point that [00:10:55] I have, I've actually wanted to try and with your audience, you're going to make it happen.

I actually [00:11:00] think there should be some nuance within the conversation of fasting that I have more and [00:11:05] more. I consider there to be two types of fasts. One, which is a kind of true [00:11:10] classic Fast where you're not eating or drinking any calories. That I call a [00:11:15] caloric fast.

**Music:** Mm hmm.

**Benjamin:** And then what you and I are just describing is what I call a [00:11:20] metabolic fast.

There

**Music:** you go.

**Benjamin:** Which is what, what determines whether the body's in [00:11:25] At the level of the cell, all of the biochemistry, like the most famous effect being [00:11:30] autophagy, who determines whether autophagy is turned on or off? Well, it's actually insulin for the most [00:11:35] part. And this is why famous fasting slash starvation [00:11:40] physiologists like Dr.

George Cahill, who is a legend to me, he described insulin [00:11:45] as the hormone of the fed state. And in his Estimation, [00:11:50] which we ought to take seriously, he said that's insulin that really determines whether the [00:11:55] body's in a fed state, high insulin, or a fasted state, low insulin. Well, what if you're [00:12:00] eating in such a quirky way that you are putting calories in?

But you're keeping [00:12:05] insulin low, so ketogenesis is uninterrupted, mTOR is not turned on, [00:12:10] and there's no ceasing or inhibition of autophagy. As far as the cells are concerned, [00:12:15] you're still fasting. That's a, that's a pretty metabolically relevant [00:12:20] scenario, where I believe more and more As much as there are people who say we need to be doing protein [00:12:25] fasts, where you're just eating protein, I actually, during the fast, I actually think there's more [00:12:30] value in doing fat fasts, to be honest.

Now, there's no studies that have compared those two, so I don't mind if people disagree [00:12:35] with me, but for all the reasons you and I have just outlined, I actually think a fat [00:12:40] fast is superior in its outcomes than any other form of fasting.

**Dave:** [00:12:45] Having tested them all on myself and had hundreds of thousands of followers over the last ten years, [00:12:50] I find the same results, and people get really mad.

Like, well, in the mice studies, [00:12:55] it was just water. It's because they didn't test the fat thing. And then I funded some [00:13:00] research at the University of Washington around exclusion zone water in cells and how [00:13:05] certain types of lipids, like C8 MCT, make mitochondria work better by [00:13:10] changing water. Like, very esoteric stuff.

That's Dr. Joe Pollock's work. And maybe that's why. [00:13:15] But what I do know is the two types of fasting you're talking about, like a caloric fast [00:13:20] or a metabolic fast, I I characterize them as there's [00:13:25] a spiritual fast where you're okay to be maybe at a lower level performance [00:13:30] and you're, you're going to push yourself and be introspective and then there's a working fast where it's the middle of the week.

I [00:13:35] have a job and kids and stuff to do. So a little bit of fat makes the fast completely painless and you [00:13:40] get the metabolic benefits because I couldn't do what I do if I was never, you know, if I was always [00:13:45] just fasting on water, I think it'd be miserable.

**Benjamin:** Yeah. Yeah, uh, in fact, because [00:13:50] you just invoked spirituality, I will just add a hearty amen.

I completely agree with everything you just [00:13:55] said.

**Dave:** I always look for alignment and areas where we maybe see things [00:14:00] differently because, look, I have a perspective. I could be wrong, and you know a lot, [00:14:05] so let's find the areas where, okay, that makes sense, and then the areas where maybe I'm full [00:14:10] of crap. What concerns me?

Is if you take a little bit of [00:14:15] insulin and you put it in a nasal spray and you spray it up your nose, [00:14:20] it's a way of treating Alzheimer's. It's also a really potent cognitive [00:14:25] enhancer. So why is it when I raise insulin? My brain works better. Yeah, yeah.

**Benjamin:** So [00:14:30] I have to speculate a little bit, Dave, just because I want to and I want everyone listening to know if whenever, [00:14:35] whenever I mentioned I'm speculating, it's because I don't know of a specific study that I can rely on [00:14:40] to answer.

So, what I suspect is happening is that with the intranasal [00:14:45] administration, you're able to get that insulin absorbed directly through that. Upper kind of [00:14:50] wall of that nasal cavity. It's so this is insulin is not getting systemic. Right. In other words, [00:14:55] it's not getting into general circulation. You are just kind of shooting it right up to the [00:15:00] brain, especially the hypothalamus, which is, you know, right in that lower part or just above that [00:15:05] nasal cavity.

And it could be, now I'm, I'm theorizing, it could be that as the [00:15:10] insulin perfuses directly up through that layer, the epithelium of the nasal cavity [00:15:15] and up, it just directly opens the doors and allows a rush of glucose to come in. It would [00:15:20] be my speculation. Now, It wouldn't be contributing [00:15:25] to the elevated insulin induced insulin resistance, again, because if I had you on a [00:15:30] table and I was measuring your blood insulin levels systemically, like from a, your, your [00:15:35] arm vein we wouldn't get any, it'd be no So, No change.[00:15:40]

There's no change in insulin at the level of the entire body. There's no [00:15:45] systemic alteration. This was such a modest and such a direct administration. [00:15:50] I suspect it's just moving right up into the hypothalamus, opening the doors, because [00:15:55] some of the brain's glucose uptake is dependent on GLUT4, [00:16:00] which is an insulin, itself an insulin dependent Glucose [00:16:05] transporter, whereas other parts of the brain, including in the hypothalamus, it has [00:16:10] other, other doors, other glucose transporters that are just always open.

The moment glucose goes [00:16:15] up in the blood, it will flow into that cell, whatever the cell may be, the kidney or the liver, [00:16:20] but then parts of the body, Including the hypothalamus will have some of these [00:16:25] insulin dependent doorways where insulin has to come and knock now the door opens and glucose comes [00:16:30] in so it could just be that you are just bypassing any systemic [00:16:35] circulation and going right to the brain saying hey brain I want you to open those doors and really pull in [00:16:40] a bunch of glucose.

**Dave:** That makes good sense to me. And. I've always been a [00:16:45] fan of having exogenous ketones present and doing a little bit of nasal insulin. [00:16:50] So I'm like, alright, neurons, you've got your ketones, glial cells, you've got your [00:16:55] glucose, let's go. And that is a pretty darn good state of high performance. [00:17:00] I just don't know how often it would be safe to do it, which is why I don't agree on it.

Yeah, I

**Benjamin:** don't either. I [00:17:05] would, because too much insulin will cause insulin resistance, I do think [00:17:10] that'd be one reason I'd say to use it judiciously.

**Dave:** Like once a week, kind of.

**Benjamin:** Yeah, oh yeah, [00:17:15] that'd be, I think, perfectly fine. But then second, there would be, there's another part of me that just [00:17:20] thinks, well, is, is that, is that epithelium, that layer of the nasal cavity, [00:17:25] it's not natural for it to see insulin.

Would there be some long term effect? [00:17:30] Am I stimulating too much growth? Am I gonna make the epithelium start to get a little thicker? I, [00:17:35] this is of course entirely speculative. Oh yeah. But if it's like on the order of once a week, like you're saying, [00:17:40] that would have, that's such a modest Bolles is such a modest frequency that that [00:17:45] wouldn't have any of these effects.

I'm confident

**Dave:** this is so fascinating, and I know we're getting a little bit nerdy [00:17:50] on things and at the same time, there are a lot of people who [00:17:55] are 50s, 60s, 70s. They're getting early onset brain stuff. [00:18:00] And if you do some research about insulin and Alzheimer's, you'd want carb diet, but you might want a little squirt [00:18:05] of insulin every now and then in your brain because it seems to help and All the [00:18:10] metabolic stuff that you talk about and I talk about if someone's listening to the show today, [00:18:15] and they're saying, Well, how do I know if I have insulin resistance?

How can you tell with lab tests? And how can you tell with [00:18:20] symptoms?

**Benjamin:** Yeah, yeah, let's start with symptoms just because that's going to be something people can rely on a little [00:18:25] more. Well, immediately. with regards to symptoms, if you have just been told you [00:18:30] have high blood pressure, insulin resistance is the most common cause of [00:18:35] what's just called idiopathic or just run of the mill insulin resistant of run of the mill [00:18:40] hypertension.

So if you have hypertension, that's definitely a [00:18:45] knock in favor, a check in favor of you having insulin resistance, high blood pressure, [00:18:50] most common manifestation. If you have a family member with type two diabetes, diabetes, [00:18:55] You're on that spectrum as well, very likely, you're much more likely to [00:19:00] have insulin resistance.

And maybe the final one of the symptoms before mentioning some clinical tests [00:19:05] is, is the skin itself, where there are two [00:19:10] distinct skin, Manifestations that are a direct result of insulin resistance. [00:19:15] One is called acanthosis nigricans. Then the other one is [00:19:20] skin tags. Skin tags is a little more obvious where it's just like a teeny little, like [00:19:25] mushroom stalk.

Like, it's not a big rounded mole. It's a teeny [00:19:30] little bump. People are probably thinking of it correctly. Right now, you can imagine there's these, [00:19:35] just almost like a little mushroom of skin. Mm hmm. And you tend to get them around the neck. You can [00:19:40] also get them around the armpit, usually anywhere where there's going to be a skin fold or a wrinkling [00:19:45] or crinkling of skin, you can see these skin tags.

And then the more [00:19:50] complicated term I just mentioned, acanthosis nigricans, that's also at those same [00:19:55] locations, like the, around the collar of the neck where we all have a little bit of a [00:20:00] skin fold, and then around armpits and groin, etc. The skin can get a little darker. [00:20:05] And now, depending on pigment of skin pigment, that may be easier or harder [00:20:10] to see the natural pigment of the person, but the skin will also start to have a [00:20:15] texture and appearance of like crinkled tissue paper.

So if we took a tissue paper, crumpled it up [00:20:20] and then opened it back up, that's kind of how the skin might look. So this darker [00:20:25] crinkled skin and skin tags, that is proof positive [00:20:30] of insulin resistance. And then just if you're wondering, that's also. Imminently [00:20:35] reversible, as the insulin resistance goes away, the skin resolves in that [00:20:40] regard.

So skin is kind of a, a window to the metabolic soul in that regard.[00:20:45]

**Dave:** I [00:20:50] remember when I was about 13, I started growing those skin tags. I had [00:20:55] hundreds of them, they're around my neck, armpits, and they used to sometimes get a little irritated or they'd bleed and Yeah. And [00:21:00] one day I was, I'm done with this and I took like little fingernail scissors and I just cut all of them off because they have a tiny little [00:21:05] stalk that don't really bleed when you do that.

Yeah. And it just frustrated me. I had no idea what it was. I thought maybe they're [00:21:10] warts and I'm familiar with that as a sign of insulin resistance and there's a lot [00:21:15] of listeners right now going, Oh my God, I started growing those. They went away when I [00:21:20] went into ketosis for the first time, and when I did the whole bulletproof diet thing, I just, I don't have [00:21:25] them, and I didn't have to cut them off, they just disappeared.

**Benjamin:** That's right, yeah, so the nice thing about the [00:21:30] skin in this regard is that it is so dynamic, you know, we are growing and sloughing [00:21:35] skin literally every moment, and so as, as the insulin levels start to come down, [00:21:40] That hyper excitability of that layer of skin cells, because insulin basically [00:21:45] starts to stimulate this kind of sporadic growth.

Insulin likes to grow things. That is not [00:21:50] inherently bad. We need it. Without insulin you die, so lest anyone thinking Dave and [00:21:55] I are painting insulin as the villain, No, but the poisons in the dose, if you will, where we're [00:22:00] talking about when it's gone beyond normal levels, insulin's resulting is kind of frenzied [00:22:05] growth of these skin cells.

But again, the nice thing with the skin, it gets sloughed off [00:22:10] so readily, literally all the time that once the stimulus to keep growing goes [00:22:15] away, I mean, give it like just week, just a couple of weeks, and they're likely going to [00:22:20] start literally just You'll notice them smaller, smaller, and they're gone as they just wear [00:22:25] off.

And now the clinical tests, just for the sake of time and, and not [00:22:30] getting into too much depth, I'll just mention two. One is just getting [00:22:35] insulin itself measured this is, I believe, one of the greatest mistaken approaches [00:22:40] of conventional clinical care, despite all of its problems, we have a [00:22:45] glucose centric paradigm of metabolic health, and the conventional clinician is [00:22:50] content just once a year measuring someone's glucose levels, and glucose is a [00:22:55] late, signal that while glucose levels are staying normal over [00:23:00] years and the person's developing hypertension and erectile dysfunction and migraines, it's [00:23:05] insulin that's been the canary in the coal mine metabolically.

It's getting higher and [00:23:10] higher and higher. So look at your insulin. I generally outline three ranges. [00:23:15] If it's six micro units per mil and lower, that [00:23:20] is that's really good. Solid sign, absolute green light,

**Dave:** less than six.

**Benjamin:** Yep. And then if it's [00:23:25] seven to mid teens, I'd just say seven to 17, then it's sort [00:23:30] of, you know, Hey, possible problem now.

I, I, as a scientist, I don't like being [00:23:35] a little soft in my wording. I like to be very clear and definitive. Insulin is a [00:23:40] hormone and every hormone has its own ebb and flow. [00:23:45] So the reason I kind of give a bit of an if with that range is if someone had their insulin [00:23:50] measured and it's 11 or 12, they would say, oh.

Shucks, I'm busted. Yet that [00:23:55] might have been just a peak moment where your insulin might have been [00:24:00] kind of peaking up as it has a natural circadian rhythm to it. And so if [00:24:05] it's in that middle range, you might be all right. All the more reason to rely on the second [00:24:10] one I'll just mention in a second. But you might not be.

And then if it's high teens and [00:24:15] beyond, absolute red light. Even the normal volatility of insulin shouldn't be getting that high in a, [00:24:20] in an insulin sensitive person. So high teens into twenties, warning, you're [00:24:25] insulin resistant. Red alert. Now, the other metric is valuable [00:24:30] because it's not as volatile, and it is surprisingly [00:24:35] accurate, or a good predictor, a poor man's method, and that is the triglyceride [00:24:40] to HDL ratio.

So just take your fasting triglycerides, [00:24:45] divide it by your HDL, and that's going to be really good. If that number, there is some [00:24:50] differences across the ethnicities.

**Music:** Right.

**Benjamin:** So, the [00:24:55] general rule is 1. 5. If that ratio is above 1. 5 [00:25:00] That's that's that's a that's a red light. That's a warning now in some [00:25:05] other subsets of populations around the world like East Asians, Japanese Korean, that [00:25:10] number ought to be actually closer to one.

So it's a little more strict. So it triglyceride [00:25:15] to HDL ratio of one is your cutoff lower the better. And then in in [00:25:20] blacks, and I think Hispanics, it can be a little higher where the normal cutoff is going to be about 2. [00:25:25] 0.

**Music:** Okay,

**Benjamin:** so it's going to be around that one to two range. Transcribed if that, if [00:25:30] it's higher than that, warning, metabolic dysregulation.

**Dave:** Does it change for men [00:25:35] and women?

**Benjamin:** Yeah, that's a great question. No, no, because those numbers are pretty static across the sexes. [00:25:40] It's funny you bring that up though, because the one lipid that isn't is free fatty acids. [00:25:45] Right. If anyone ever measures free fatty acids, which is Not the [00:25:50] same. You know, triglycerides are what the liver is making, or what you're eating.

Free fatty [00:25:55] acids are 100 percent a product of what the fat cells are breaking down. [00:26:00] And women, due to estrogens, most gals don't appreciate this, but a woman at any moment [00:26:05] is burning 40 percent more fat than her male counterpart. What? And you see this. That is not fair. You see this [00:26:10] in her free fatty acid levels.

Now, Dave, before we fellas Claim [00:26:15] defeat here and say, we've been robbed. She's also putting in more fat at [00:26:20] any moment too. And so because she's eating more

**Dave:** fat or because her, no, no, not at all. Just because

**Benjamin:** [00:26:25] of sex hormones. Okay. So the, so estro, estrogens are both stimulating the [00:26:30] breakdown and. The building up so females just have a much much higher rate [00:26:35] of turnover in their fat cells, you know, so so ends up because the [00:26:40] the overall dynamic is she will have more fat on her body than her male counterpart, which [00:26:45] she's supposed to.

It's by design. because she carries the metabolic [00:26:50] burden of reproduction. She needs to have this kind of metabolic insurance [00:26:55] before she ever commits to the metabolic marathon of reproduction, but she's also [00:27:00] turning it over at a lot faster. So she, her free fatty acids will be normally [00:27:05] about 40 percent higher than what you see in a guy.

But the other lipid measurements are generally going to be in [00:27:10] the, at least those ones are going to be generally in the same range.

**Dave:** What I have found [00:27:15] 99 percent of the time when people go on something like the Bulletproof diet, which [00:27:20] is clean, cyclical keto, the right kinds of animal fats controlling the [00:27:25] type of protein toxins, all the stuff that I've been teaching for a long time, HDL goes through the roof and [00:27:30] triglycerides drop.

Which is exactly what you want for this ratio sure is and [00:27:35] when I went to my my first longevity doctor when I was maybe [00:27:40] 29 There's a long time ago, and I came in [00:27:45] with that pattern. He just looked at me said well you're Your lipids are [00:27:50] technically disordered, but really good. Because, you know, my [00:27:55] HCL was like, how did you get it so high?

And why is, why is your triglyceride low? And it was because of [00:28:00] the idea of intermittent fasting and things like that.

**Benjamin:** Yeah, you're doing it right. That's the right way to do it. And it is a consistent [00:28:05] finding that you, you start to control carbohydrates. Triglycerides are going to come down, because it's [00:28:10] mainly the elevated insulin that is spiking the production of triglyceride [00:28:15] rich lipoproteins from the liver.

So, as carbs go up, triglycerides will go up. As carbs come [00:28:20] down, triglycerides will come down.

**Dave:** I have, uh, a confession to make about an [00:28:25] experiment I've been running for the past Probably a year. [00:28:30] I am down to about 5 percent body fat. I am like crazy, crazy [00:28:35] lean. And this is on using, you know, [00:28:40] 26, 000 clinical grade bioimpedance things and lots of different measures.

And I [00:28:45] used to weigh 300 pounds. Isn't that amazing? What a

**Benjamin:** testament [00:28:50] to just how adaptable and dynamic the human body is.

**Dave:** It [00:28:55] blows me away, and like my new book cover, I'm on there with my shirt off, and I'm like the fat [00:29:00] computer hacker who would never, I didn't want pictures of me when I was fat. So I'm still kind of stunned by [00:29:05] it, but here's the problem.

5 percent might be below [00:29:10] ideal for longevity. I think closer to 8 is where I want to be. Like, [00:29:15] I'm really having a hard time putting on fat, and I have half of a large bath towels [00:29:20] worth of extra skin from when I was obese. So, I went in a couple weeks ago, I just [00:29:25] posted about this on, online, and they removed a passport size piece of skin [00:29:30] from each side of my face and neck.

This isn't from aging skin, this is obese [00:29:35] skin. Probably postnatal aging, I don't know. And I've been healing really fast, but they're [00:29:40] saying, Dave, we need some fat to put in with stem cells in your face to get the volume back. [00:29:45] So I have been pounding the carbs, Ben, like 400 [00:29:50] grams of rice and honey and fruit and watermelon, and I have not put on any fat.

[00:29:55] They found 20 cc's when they like completely emptied my butt of all the fat they could find. Oh my gosh. So how is [00:30:00] it that I'm not getting fat on 400 grams of carbs? I'm doing 200 grams of animal protein a day. I'm only eating good [00:30:05] fats, but what's going on?

**Benjamin:** Yeah, yeah, well, so I [00:30:10] can't state definitively, but generally there are going to be two essential variables that [00:30:15] determine the growing and the shrinking of the fat cell.

Now, your audience is savvy enough to know that there is more [00:30:20] than just one variable, which is the traditional view being that it's just purely calories in, calories [00:30:25] out. That is so easily disproven. No, right? No, but, and I'm not [00:30:30] saying, like, just so everyone knows, I'm not saying calories don't matter. But [00:30:35] that is the only message anyone ever hears that people think that's what [00:30:40] I'm saying.

And if I kind of claim any authority, and I, I'm loathe [00:30:45] to do this, I've such a general disregard for higher education and terminal degrees, like a [00:30:50] PhD, which is ironic, I know, but I'm surrounded by PhDs. And so I've seen [00:30:55] how Dumb. Many of these people are, and I hope I'm not one of them. And I don't [00:31:00] necessarily just mean my colleagues here.

I love them all dearly. But the, my, if I'm going to [00:31:05] make any sort of stake a claim here, I have a unique PhD is, and you noted this in [00:31:10] the introduction, it's very uncommon. There are only, I think two institutions [00:31:15] on the planet that grant a dissertation, a PhD in [00:31:20] bioenergetics. Bioenergetics is a particular niche of, of, of [00:31:25] research that combines the metabolism, biochemistry, and physiology, [00:31:30] but with a heavy focus on thermodynamics, and, and the [00:31:35] relevance of thermodynamics in living systems, because less Anyone [00:31:40] has forgotten thermodynamics was originally posited as a view [00:31:45] to make the better steam engine.

That it is in the realm of physics and [00:31:50] it is an odd, uh, or it's an awkward fit to try to [00:31:55] fit principles of physics in principles of biology. I actually think [00:32:00] it, I'll make this kind of a bold claim. I consider the [00:32:05] introduction of calories into biology as part of what got us here. [00:32:10] Where we got it wrong.

It, it's so thoroughly distracted us from, [00:32:15] from I think what matters most that it actually brought us to where we are. I think the whole [00:32:20] war on fat in part was born because of this improper [00:32:25] introduction or invocation of, of thermodynamics. Now having said all that. [00:32:30] Calories matter, and those carbons need to be accounted for in some way.[00:32:35]

But a cell, like especially a fat cell, it needs to [00:32:40] know when it's time to eat and when it's time to break down. And the [00:32:45] fat cell wants to be And everyone, pardon me if it sounds like I'm being silly. I am, after all, a professor who [00:32:50] teaches 18 year olds, so I have to sometimes be a little juvenile in my description of things.[00:32:55]

But the fat cell needs to know that it's playing nicely in the entire neighborhood of [00:33:00] the body. That it needs to know, okay, what are the demands of the brain right now? What are the demands [00:33:05] of the muscle? There's no direct nerve that's connecting them. It's [00:33:10] hormones that generally will tell the Overall orchestra when it's the [00:33:15] woodwinds section to play when it's the brass section or when is it time for the muscle [00:33:20] to be needing energy you couldn't you don't want muscle to [00:33:25] be exercising and pulling in energy to break it down at the same time fat is taking in energy to [00:33:30] store the body.

And it's overall balance of metabolism wants [00:33:35] to balance out the two parts of what is metabolism. Anabolic versus [00:33:40] catabolic. Insulin is the hormone that sends that signal. So, this is my really [00:33:45] long winded way of just saying, I have right now down the hallway in my lab, [00:33:50] fat cells growing in Petri dishes.

Like literally right now. I'm not even being hyperbolic here. [00:33:55] Those fat cells, when we first plate them and they're sticking to the cell, to the bottom of [00:34:00] the dish, They are in a bath of tons of calories, [00:34:05] tons of fat and tons of glucose, everything a fat cell wants in order to grow, [00:34:10] but it stays small.

Until we do one single thing, which is [00:34:15] add insulin into the culture. The moment insulin comes into that little culture media, [00:34:20] we call it, or that bath, now the fat cells would say, if they were part of the greater whole, [00:34:25] Ah, it's time for me to eat. This is my signal that it's time to store [00:34:30] energy rather than break it down.

And now, if we look at those fat cells just four to six hours [00:34:35] later, They're actually thicker, chubbier. If we look at them six hours later, still, they're bigger [00:34:40] again. So all of this is just to say that there must be a [00:34:45] stimulus that tells the fat cell to grow, which is elevated insulin. And then there must [00:34:50] be sufficient calories to fuel that growth.

You cannot have [00:34:55] one without the other, and just to really put a fine point on that, if a person had high [00:35:00] insulin, and low calories, they will die. Because they would become [00:35:05] hypoglycemic, and there would be no ketones being produced, because the high insulin would be inhibiting [00:35:10] ketogenesis, and then the brain would have been deprived of its two fuels, and the body would [00:35:15] shut off.

The brain would shut off. Now, in contrast, if insulin's low, [00:35:20] and calories are really high, What happens then, now the person is burning up [00:35:25] almost, they, they burn to death, where their metabolic rate and their ketogenesis is [00:35:30] unstoppable because there's nothing to tell the body to stop burning energy, and so [00:35:35] they die from ketoacidosis and hyperglycemia.

This is [00:35:40] a scenario which is so real that, and it works, where you can eat as much as you want. [00:35:45] People with type 1 diabetes, some of them have learned and are so [00:35:50] tempted that they can deliberately underdose their insulin level [00:35:55] and be into a state of ketoacidosis and massive hyperglycemia and [00:36:00] feel miserable and wretched and yet they will be as thin as they want.

Just everyone [00:36:05] imagine the temptation. You know, they don't have to vomit up their food. They don't have to [00:36:10] starve themselves through traditional anorexia. They can eat everything they want. They can [00:36:15] enjoy the sensation of eating it and swallowing it and digesting it. Which has its, [00:36:20] all of its own gratification.

And all they have to do is not poke themselves with a [00:36:25] needle and inject their insulin. That's a condition called diabulimia. And all of this [00:36:30] is just to say, there are two parts. The fat cell must be told. To store insulin or to [00:36:35] store fat via high insulin, but it needs sufficient calories to fuel that [00:36:40] growth.

It's one thing to tell the fat cell, let's grow and the fat cell has to grow.

**Dave:** So low [00:36:45] insulin and high food intake equals thin [00:36:50] and lots of energy, but I guess they feel like crap.

**Benjamin:** Oh yeah. I mean, they, yeah. In fact, you can read case reports [00:36:55] where the, the people will say, like, I, I felt, I feel like I'm dying, but.

I want to stay [00:37:00] thin.

**Dave:** Wow. Yeah, that's unhealthy. Oh, I mean,

**Benjamin:** just imagine.

**Dave:** Yeah.

**Benjamin:** Yeah.

**Dave:** [00:37:05] I did notice, when I was testing out the edges of the recommendations before I [00:37:10] published the Bulletproof Diet, and I published that in 2014, I published it online in maybe 2013, I went for [00:37:15] about a year. And I was eating 000 calories a day on [00:37:20] purpose.

More than I wanted. Like, forcing myself to do it. Eating no [00:37:25] carbs. And I was sleeping less than 5 and usually less than 4 hours a night on purpose. [00:37:30] Like, I'm going to set myself up to get fat. I'm going to prove that I don't get fat the way I should. But [00:37:35] I kept losing weight. I grew abs for the first time.

On crazy amounts of this. [00:37:40] I know my insulin was low because I had it measured. It was actually pretty darn low. I don't remember the number off the [00:37:45] top of my head. And that probably explains why I didn't feel like crap until kind of maybe [00:37:50] halfway through I started getting cortisol effects where I would wake up dozens of times a night.[00:37:55]

My sleep quality went down. You start getting reductions in testosterone, [00:38:00] thinning of hair, and all of that. And I stopped it, but I meant to do it for a month to just make fun [00:38:05] of the calorie people. And I ended up doing it for a lot longer because I'm actually in a really [00:38:10] strong state of high performance.

Why did my brain work so well when I did that, at least for the first six months?

**Benjamin:** [00:38:15] Well, Dave, so you brought up a lot of stuff and I, and I, I don't want to not answer the question you started with, which [00:38:20] is why you're not getting fat. So let me come back to that first part and then to answer [00:38:25] this part right now, yeah, I mean, ketones are the preferred brain [00:38:30] fuel.

One of the notions I attempt that I, I need to disabuse my students of is that they've [00:38:35] been told the brain prefers glucose. And you need to eat this much glucose. That's not true. There's multiple things wrong [00:38:40] with this. Not true. Not true. But just to really establish [00:38:45] the relevance of that, I showed them research from Dr.

Cahill, where you [00:38:50] can take someone with a glucose level of, I'll, I'll use the same kind of units here. Let's say [00:38:55] five millimolar, which would be kind of 80s milligrams per deciliter. [00:39:00] And if their ketones are at one to two millimolar, so less than half, of what [00:39:05] the glucose is, the brain's already getting most of its energy from the ketone.

[00:39:10] So don't tell me the brain prefers glucose. If anything, the brain has a preference for ketones, [00:39:15] which Dr. Richard Veitch and many, many others, my own lab, has reported papers on this. It [00:39:20] enhances mitochondrial performance. For every, for every oxygen, unit of [00:39:25] oxygen that the mitochondria are consuming, when they're fueled with ketones, they're producing more [00:39:30] ATP.

**Music:** Yes.

**Benjamin:** So the, what we described that in my publication is that the ketones were [00:39:35] resulting in a tighter state of mitochondrial coupling.

**Music:** Mm hmm.

**Benjamin:** Which is, how much [00:39:40] energy am I breaking down in order To produce this energetic molecule ATP. And it [00:39:45] was, it was better now in your case of what you're trying to do now.

One of the reasons [00:39:50] I point the finger at carbs is because of what it does to insulin. And so in [00:39:55] your situation, you are either now so exceptionally glucose [00:40:00] tolerant that even though you're eating hundreds of grams of carbs, if we were to act, [00:40:05] you're, you're clearing it so quickly that it's never resulting in substantial insulin spikes [00:40:10] or.

Well, I, I, that's it. You're just that insulin sensitive and glucose tolerant.

**Dave:** [00:40:15] HbA1c went up. In a way that I don't like, so I'm, I'm on low carbs now.

**Benjamin:** Yeah, but do you, [00:40:20] but are you still in ketosis pretty often? Have you been measuring your ketones? Um,

**Dave:** I, not with a [00:40:25] finger stick, but I just use exogenous ketones several times a week.

I, I know there's ketones [00:40:30] present a, a meaningful portion of the time.

**Benjamin:** Well, I would be curious if you, like in a [00:40:35] morning, so one day, like on, on, on a today you ate 500, you, 400 grams [00:40:40] of carb, but tomorrow morning, if you measured your ketones, If you are [00:40:45] in ketosis, then your insulin is not really going up that high.

**Music:** Interesting.

**Benjamin:** Not [00:40:50] only, so the other point I meant to make is that you're either, it's a combination of you're super insulin sensitive, [00:40:55] and You're just maybe so physically active. I have known [00:41:00] Really active individuals who eat hundreds of grams of carbs and they're still in ketosis the next [00:41:05] day Yeah They're just burning so much of the glucose and the magic [00:41:10] of the working muscle is That it can pull in that glucose without the need for an insulin [00:41:15] spike As I mentioned earlier insulin wants to store energy which is [00:41:20] antithetical to exercise which wants to break down down energy.

It wants to break down what's [00:41:25] been stored. And so exercise quickly lowers insulin due to [00:41:30] the sympathetic stimulation. Basically, the body starts moving and then the brain starts to tell the [00:41:35] pancreas, Hey, you got to shut down insulin production. And it does so very quickly. And [00:41:40] so all of this can combine that if a person's already insulin sensitive, which you certainly are [00:41:45] combined with high levels of physical activity, if you aren't getting elevated insulin [00:41:50] for pronounced periods of time, it doesn't matter.

If you're eating those calories, you're not going to store it as fat. [00:41:55]

**Dave:** Even if my blood sugar goes up, but my insulin isn't going up, you're saying?

**Benjamin:** Yeah, [00:42:00] well, it could. That's exactly right. Yeah, so glucose is not a perfect [00:42:05] surrogate for insulin, as much as we wish it were. Yeah, it is important because while we can [00:42:10] measure glucose so readily it is, we are years away from having a continuous insulin [00:42:15] monitor, and they're not the same.

Like, for example, if Dave were to take 100 grams of [00:42:20] carbohydrate and then immediately go exercise, we would see that spike. It, depending on how [00:42:25] intense the exercise is, it may even stay high for a while, but no insulin was [00:42:30] required during that exercise. As it starts to come down, it's not because insulin went up.

[00:42:35] It's because the muscles are pulling it in on its own, because again, when the muscle contracts, [00:42:40] it has an insulin independent way of opening its doors. You'd [00:42:45] mentioned mTOR earlier, and I know you're familiar with mTOR's opposite, which is AMPK. When [00:42:50] you start to contract and relax the muscle, It's flooding the muscle cells with [00:42:55] calcium.

Calcium, as it goes up, will activate an enzyme called calcium calmodulase [00:43:00] kinase, or cam kinase. So a calcium activated enzyme. Cam kinase will [00:43:05] then activate AMPK. AMPK will then activate AS160, which [00:43:10] activates GLUT4. And now we've opened the glucose doors and we've been able to pull in that [00:43:15] hungry muscle, basically says to insulin, Hey, I'm too busy to wait for you to tell me what to eat.

I'm just going [00:43:20] to eat this because it's here and I'm going to pull it in.

**Dave:** This is why even going for a walk after you eat [00:43:25] carbs or doing, doing some squats. It doesn't really matter. Not even weighted squats, just do, you [00:43:30] know, 30 air squats. And you can watch. I've used a glucose monitor for long [00:43:35] periods of time, and you can see, oh yeah, eight carbs, it's going to go up, and then you do a little bit, and [00:43:40] if you actually do a real workout with weights or something and real resistance, you're [00:43:45] not going to get much of a spike at all.

**Benjamin:** Yeah, so that might be part of your problem. Honestly, Dave, for you to gain [00:43:50] weight, you may have to start changing so many habits. Well, I don't

**Dave:** exercise that much. I do [00:43:55] really good exercise with AI. stressing the system from the upgrade lab stuff. [00:44:00] I do about like 20 minutes a week of real exercise and it's [00:44:05] carefully scripted to, to exhaust and then recover very quickly.

So I [00:44:10] don't think my volume is high, but my, my, my recovery is high and my intensity is high. [00:44:15] So I'm like, I'm, I'm a weird robotic cyborg biohacker guy testing out weird stuff. [00:44:20] And I was like, you would probably know, why is it possible to have my [00:44:25] average glucose go up? And to eat ridiculous amounts of carbs, [00:44:30] sometimes more than I want, and then to not gain one pound.

And it sounds, it sounds like [00:44:35] you you figured it out.

**Benjamin:** Yeah, well, I, I think that if We'd really need to know what [00:44:40] your insulin levels are. It's entirely possible that your fasting insulin levels are still in an [00:44:45] exceptionally good range. And that what you think is a big insulin spike, it could be [00:44:50] much more modest than you realize.

And, and if that's the case, then you're kind of getting into [00:44:55] that, you know, physiological scenario of what diabulimia, [00:45:00] which is that the insulin levels are getting so low that it's just incompatible with storing [00:45:05] fat.

**Dave:** That makes so much sense. I want to touch on [00:45:10] one more thing about calories in, calories out.

Because you and I have both been trolled [00:45:15] by these like angry 25 year olds, you know, cancel out a Snickers bar with a Diet Coke kind of [00:45:20] people. And I, I just laugh. I'm like, well, if calories counted, [00:45:25] there's a million calories in a gram of uranium. And they go, but you have to be able to [00:45:30] absorb it. I'm like, oh, So calories don't count, calorie absorption counts.

And then they [00:45:35] start twitching usually, which helps them burn more calories, which probably is good for their weight loss. [00:45:40] And then I say, well, do you know about Xeronal? And they're like, no. Well, it's a drug derived [00:45:45] from mold toxin that's 10, 000 times more estrogenic than normal [00:45:50] estrogen. And if you give it to a cow, which ranchers do, the cow will get fat on 30 [00:45:55] percent less food.

So, if the drug can exist, and there's tens of millions of dollars being [00:46:00] spent because it works, It's not about the calories. I'm not saying that they are relevant in some way, but [00:46:05] this idea that I'm going to somehow know how many calories I burned without being in a [00:46:10] calorie chamber, that's nonsense because only half your calories are [00:46:15] from moving.

The rest of the stuff is respiration, temperature, humidity, stuff that no one's going to measure. So [00:46:20] you don't know how many you burned and you don't know how many you ate because the dumb little apps like as a [00:46:25] guy who makes You know, a hundred million dollar plus of food things. The number [00:46:30] of times that I've gotten in trouble because when I buy a fricking cashew, [00:46:35] the calories per gram varies based on the time of year.

So you can't even [00:46:40] say a gram of cashew always has the same calories. And so my labels have to be [00:46:45] within a very narrow window. And so it's complete fantasy. [00:46:50] That we can measure what we're taking in or what we're putting out, which is why I'm [00:46:55] like, this is just a distraction, which is what you've been saying as well.

**Benjamin:** Well, yeah, and you can see why I feel [00:47:00] so strongly, as much as I appreciate thermodynamics but The [00:47:05] problem is, we don't eat heat. Let me a calorie is a unit of [00:47:10] heat. We don't eat heat. Like, does, does, if we, if we eat a warm [00:47:15] Snickers bar, is that, does that have more calories than a cold Snickers bar?

You know, cause it, [00:47:20] if it's, if it's just pure calories, and it is, then drinking a hot coffee, you know, that's [00:47:25] not a good example. But, it, it, it's not calories that matter. In an ideal world, [00:47:30] if we really wanted to have A food label that [00:47:35] gave an idea of how fattening this food will be, which is what the calories are trying to [00:47:40] do.

The whole reason we have calories is this idea that, hey, you need to control [00:47:45] your body fat, so look at these calories and count them all. It is such a fool's errand for [00:47:50] all the reasons you mentioned. We, it is impossible. For the average individual to [00:47:55] capture every calorie coming in, let alone every calorie going out.

Now [00:48:00] I, by going in, not only did you mention how even on a label it might have gotten it wrong, but you don't know [00:48:05] how much you're absorbing. You don't know what the thermic effect or the caloric cost of [00:48:10] digesting and absorbing that food is. And again, it's not units of heat that you're [00:48:15] getting. So in the ideal world, if there, if we deemed it necessary to have [00:48:20] some metric on a food label, it would be some combination of how much is [00:48:25] this going to spike your glucose.

And how much is it then going to spike your insulin, [00:48:30] how many other carbons are coming with it? Because dietary fat [00:48:35] matters if insulin's high at the same time. And because then you're storing those carbons very [00:48:40] readily. But it'd be some kind of algorithm that would give like a fat index, or a [00:48:45] fattening index rather, an obesogenic index that would [00:48:50] say the combination of carbons or calories, if we have to use that term, [00:48:55] and insulin spike means that what you're about to eat is going to be more [00:49:00] obesogenic.

then say something else, that would be more of an ideal scenario [00:49:05] because again, it's not units of heat. Calories don't really work in that sense. But one other comment [00:49:10] on this that I thought while you were explaining the scenario earlier, when insulin comes down, [00:49:15] as I've mentioned now, a couple of times, it is so antithetical to the body storing energy that someone will [00:49:20] say, well, where does that energy go?

You have to account for it. And I agree that you have to account [00:49:25] for the carbons. They need to go somewhere, but the body, it is so [00:49:30] antithetical. to storing carbons when insulin's low, that the body has these release valves, [00:49:35] these pressure valves, in two forms. One is that when insulin [00:49:40] is low, metabolic rate goes up.

We've known this for a hundred years by studying [00:49:45] people with type 1 diabetes, there is a substantial Elevation and just resting [00:49:50] energy expenditure Just the cost of living the body's just running hotter the [00:49:55] engines idling faster when insulin's low My lab published a [00:50:00] report finding that part of it is because of the effect of ketones We found that when a person was [00:50:05] in ketosis and we did human Fat biopsies that their metabolic rate from their [00:50:10] fat tissue was three times higher than when they weren't in ketosis [00:50:15] So there is just this increase of metabolic rate in fat tissue itself.

So when insulin [00:50:20] comes down metabolic rate goes up That's one part of it. The second part of it [00:50:25] is the that's the wasting in the form of heat What I just described, then it's wasting in the form [00:50:30] of ketones. Everyone remember, ketones have a caloric [00:50:35] value that is almost the same as glucose. And when you're in ketosis, [00:50:40] you are breathing out ketones, which is to say you're breathing out calories.

Oh wow. [00:50:45] And you're urinating out calories because you have ketones that are getting excreted in your urine. And [00:50:50] remember, ketones have a caloric value, or in other words, carbons that could be [00:50:55] used. That's amazing. But we're wasting them.

**Dave:** How did I not know that? I never thought of it. [00:51:00] So that's one of the benefits.

of intermittent fasting or being on a ketosis kind of diet, [00:51:05] you'll breathe out calories and you'll pee out calories.

**Benjamin:** Yeah. I mean, the collective [00:51:10] effect of all of this could be as high as five to 600 calories. Holy crap. That's a, [00:51:15] that's a lot of stair stepping. That's a lot of exercise to try to get. I mean, anyone, next [00:51:20] time you're working 600 calories burned [00:51:25] as opposed to just being in it.

No. It's so long. It's so hard. Just [00:51:30] being in a ketogenic state, you're doing it without even thinking about it. This is why you and [00:51:35] I are able to say, don't count calories. If you're hungry, like to the average person who wants to lose [00:51:40] weight, the traditional view is cut your calories. But if you're not addressing your [00:51:45] insulin, That's just going to promote hunger.

Hunger always wins. Before you know it, you'll be right back where you were. [00:51:50] So let your first step on your weight loss journey be, I'm going to lower my insulin. [00:51:55] Calories will take care of themselves. Just control carbs, eat good protein and good fat. And when you're [00:52:00] hungry, eat. If you're not hungry, don't eat.

And with this plan in [00:52:05] mind, insulin will come down. Again, calories will take care of themselves.[00:52:10]

**Dave:** Are people going to lose muscle mass if they go [00:52:15] into ketosis and they lower insulin?

**Benjamin:** Oh, that's a great question. I'm happy to answer it [00:52:20] because of, for, I can rely on some good research. Two thoughts come to mind. One [00:52:25] was the Scottish man, I think who fasted for 384 days and [00:52:30] then worked from my own lab.

So let me start with the Scottish guy. This is a documented case [00:52:35] report where he literally fasted for more than a year. And Dave, something I actually thought about earlier [00:52:40] when you were describing kind of your own feeling and then the cortisol, it's, it's for people to understand [00:52:45] the difference between a fast and starvation.

Yes. The difference [00:52:50] between those two is fat tissue. If you have fat tissue to [00:52:55] burn, That means you're making ketones, which means you're feeding the brain everything it could ever want. [00:53:00] As you start to run out of fat, now you run out of ketones, now the [00:53:05] brain has to rely on glucose. And what's the main source of glucose?

It's going to be amino [00:53:10] acids in that state. Not in the normal state. In the normal state, it's most of the [00:53:15] source of ami of glucose is coming from lactate, by the way. But, when with long [00:53:20] term fasting to the point of, I've run out of fat tissue, now the body starts breaking down muscle [00:53:25] to get those amino acids to convert those into glucose, and cortisol helps it happen.

Now, in [00:53:30] this guy, he had so much fat because he was morbidly obese, he didn't lose muscle. [00:53:35] He maintained his muscle mass throughout this entire fast. Now, in [00:53:40] my lab, we published a report finding, in fact it was right after we did the fat [00:53:45] cell study, where we looked at how fat cells respond to ketones by [00:53:50] increasing the metabolic rate, and there's a lot of wasting of energy.

We did a comparable [00:53:55] study, not quite as strong, we didn't use humans, but we did a comparable study with [00:54:00] muscle tissue and ketones, and we found that ketones, Enhance [00:54:05] mitochondrial coupling of the muscle. So the muscles getting more efficient with its use of energy [00:54:10] and it, they, the muscle cells were more robust as we kind of [00:54:15] insulted the muscle cells with some chemical stimuli to kind of knock on them and hurt them a little bit.[00:54:20]

When muscle cells were fueled with ketones. They were more rigorous, they were [00:54:25] tougher, they were more viable and more resistant to injury. Suggesting that at the end of it [00:54:30] all, people have long said that ketones defend muscle. Now [00:54:35] what they meant by that was that if ketones are up, then there's going to be less [00:54:40] breakdown of muscle because we don't need those amino acids for gluconeogenesis, which is true.

[00:54:45] But, my lab added evidence to this, which is to say that ketones really are defending the [00:54:50] muscle, because when muscles are fueled with ketones, they're literally tougher. They're harder to kill.

**Dave:** These are really good [00:54:55] arguments for cycling in and out of ketosis. I've found that when I stayed in ketosis for long [00:55:00] periods of time, that I would develop insulin resistance.

This seems like [00:55:05] a common occurrence, so that's one of the reasons I recommend cycling. [00:55:10] So talk, talk about insulin resistance and people in ketosis for a long period of time. Oh, I'm so

**Benjamin:** glad I have a chance to [00:55:15] bring this up. Yeah, so Dave, you'll you'll, you'll not be [00:55:20] upset for me to say that's not insulin resistance.

Oh, cool. What people are describing [00:55:25] in that case of a ketogenic diet causing insulin resistance, it's not insulin [00:55:30] resistance. Insulin is working exceptionally well. What it is, is an acute [00:55:35] glucose intolerance. Now, let me, let me sort of provide some interesting [00:55:40] context here. People have heard of the concept, concept of metabolic flexibility, which is this [00:55:45] idea that when you eat a mixed macronutrient meal with carbs.

You go to [00:55:50] glucose burning mode and you can detect this. And then when you enter a fasted state about [00:55:55] five to six hours later, because insulin's come down, you go to a fat burning state. [00:56:00] So the body shifts between sugar burning and fat burning. Some scientists at the [00:56:05] university of Pittsburgh years ago, 25 years ago now, I think [00:56:10] documented this concept of metabolic inflexibility, which is where there, there are [00:56:15] people.

Who have insulin resistance, because that's the cause of this. That when they eat, they're in [00:56:20] glucose, sugar burning mode. When they're fasting They're still in glucose and sugar [00:56:25] burning mode because their insulin is still elevated, which is insulin resistance. So they're [00:56:30] stuck in sugar burning mode. Now, in the long term adherence to a [00:56:35] ketogenic diet, I like to say you kind of have an inverse metabolic [00:56:40] flexibility scenario, where it's almost like the body is stuck in fat burning.[00:56:45]

Now, it's not. What has actually happened is if you've been [00:56:50] in a long ketogenic diet, actually, it doesn't even take long. It's, it's, it's, even if a person [00:56:55] fasts for 24 hours, what I'm about to describe happens to them as well. So, [00:57:00] if someone goes and takes an oral glucose tolerance test, they go drink a bunch of glucose, you [00:57:05] measure a glucose curve and you see it come up and down, and say about two hours, it's back down to [00:57:10] about 100 milligrams per deciliter.

That's a good response. Now, if this person were to then [00:57:15] adopt a Well, even fast for 24 hours, let alone be in a ketogenic diet. If they take [00:57:20] that same oral glucose tolerance test, now you would say, wow, my glucose went higher and [00:57:25] it took even longer to come down. It didn't come down until three hours or three and a half hours.[00:57:30]

The temptation. is to say I'm insulin resistant now. [00:57:35] Because if you were looking at a person with type 2 diabetes, that is what's happened. But that's not what's [00:57:40] happened in this person who maybe just fasted a little too long or is on a ketogenic diet. [00:57:45] In that case, it's because of a lack of pre formed [00:57:50] insulin in their beta cells.

So basically, when we eat carbs [00:57:55] The insulin the beta cells want to be ready, and they literally [00:58:00] have a bunch of pre packaged insulin in the beta cells stored, like, on the [00:58:05] shelves behind me, ready to go. Here I am in my beta cell office, insulin, glucose comes up, I get a [00:58:10] knock on the door, I know, okay, I gotta address the glucose, I'm gonna start shipping out all of this [00:58:15] insulin I have made right now, which is called the first phase insulin release.[00:58:20]

I am constantly monitoring the glucose. I know this isn't going to be enough. [00:58:25] And so at the same time I've been shipping out my preformed insulin, I've started making more [00:58:30] insulin. That's the second phase insulin release. When a person has [00:58:35] fasted too long, Well, 24 hours or so, that's not too long, but prior to a normal [00:58:40] glucose tolerance test, it is, or they're on a ketogenic diet, the beta cells [00:58:45] are so efficient, and I'm sympathetic to this because I hate clutter, that the beta cells [00:58:50] basically start to say, hey, I got all the stuff on my shelves here and I'm not using it.

Mm hmm. I'm just [00:58:55] going to get rid of it. And so it literally breaks the insulin down to its base amino acids and then just [00:59:00] recycles them and does anything it wants with them. But it starts to think, I don't need all this [00:59:05] insulin. And then all of a sudden, hold on, red alert. We need, glucose has just flooded [00:59:10] the system, aw, crud, the beta cell says.

Well, I don't have all the insulin made, so there [00:59:15] goes that first phase insulin response that I'd normally have, but I still have the ability to make [00:59:20] a bunch of insulin. It's just gonna take me a little longer, so, body, pardon [00:59:25] me, beta cell, for being too efficient, it's gonna take us a little longer to clear that glucose.

But, [00:59:30] says the beta cell, to finish off this weird story, If you do this [00:59:35] again within a 20 hour period or so, I'm going to keep this insulin here just in case [00:59:40] we do it again. So My long winded way of saying it's, it's, [00:59:45] it's that there is become an acute glucose intolerance because of a very [00:59:50] temporary reduction in insulin on hand Preformed and so if a [00:59:55] person is on a ketogenic diet or they're going in for an oral glucose tolerance test And [01:00:00] they think that they're gonna crush it by fasting for 24 hours You've actually set the [01:00:05] stage to get a false positive or you're gonna fail that glucose tolerance test or get a [01:00:10] worse score Because, you haven't eaten carbs recently, so [01:00:15] eat some carbs eight hours or so before you go take the test.

And, and it doesn't have to be [01:00:20] sugar, just eat some starches. And then the beta cells will say, ah, okay, maybe we're going to start [01:00:25] getting into carb consumption again. I'm going to hold on to some insulin so that when we do this test [01:00:30] in 8 or 12 hours, I'm going to be ready for it. And then you'll pass it with flying colors.

[01:00:35] Because a ketogenic diet will improve insulin sensitivity [01:00:40] exceptionally well. Remember, Dave, that everyone remember, the way I described insulin [01:00:45] resistance earlier applies. Every time we invoke the term insulin [01:00:50] resistance, which is insulin will be high. If insulin is low, it is not, it [01:00:55] is impossible for the body to have insulin resistance.

So, this scenario isn't [01:01:00] reflective of insulin resistance, but just rather a temporary state of glucose [01:01:05] intolerance, because the body's kind of been saying, well I'm not sugar burning, I'm fat burning. [01:01:10] Oh, hold on, wait, you want me to go back to sugar burning? Alright, well I wasn't quite prepared, so pardon me [01:01:15] while I just sort of make some adjustments.

And I'll be better prepared next time.

**Dave:** So, you just taught people [01:01:20] how to pre load insulin if you're in ketosis and you're gonna do a life insurance test. [01:01:25] That would be important.

**Benjamin:** 100%. And I want people to know that. [01:01:30] Like, if I imagine, you know, a pregnant gal who's been in a ketogenic diet to help with her PCOS and get [01:01:35] pregnant, Now she has to go in for her oral glucose tolerance test.

You, you want to eat [01:01:40] some carbs in, in at least some period of time before you go in, 8 to 12 hours, just [01:01:45] to have that insulin on hand and you'll be just fine.

**Dave:** Oh, that's such a gift. I, I thank you for [01:01:50] sharing that. There's some other things that affect insulin and insulin resistance, like [01:01:55] circadian rhythm.

We know melatonin going up will make you insulin resistant. We know that [01:02:00] at night you're insulin resistant. Does it matter when we measure our insulin and when [01:02:05] we measure our blood sugar and when we eat?

**Benjamin:** Yeah, yeah, it does. In fact, tragically, [01:02:10] measuring insulin in the morning is probably one of the worst times.

And that's why I [01:02:15] kind of described that there's that bit of that gray area that kind of Orange light range or [01:02:20] yellow light range where insulin might be a problem or it might not [01:02:25] be. And I actually kind of bake that into the formula because of the morning volatility [01:02:30] that in, when we're waking up in the morning, we will have higher cortisol levels [01:02:35] naturally, which starts to just mobilize the glucose to get ready for the brain to come back [01:02:40] online.

If you will, you know, as the body starts to wake back up and get alert. But also, [01:02:45] insulin can start to climb in response to that glucose climb. And so morning is one of the [01:02:50] worst times, but it's the most convenient because we're fasting overnight, we need a fasted [01:02:55] state. If, in an ideal world, we wouldn't measure fasted blood [01:03:00] levels until, you know, late morning or early afternoon to let that pass.

Can, can [01:03:05]

**Dave:** you adjust for that? Like, let, let's say you, you do your insulin test in the morning and it's, [01:03:10] Should you just subtract a point off of the morning score to get a more accurate score? I don't know.

**Benjamin:** I don't know. [01:03:15] That's a really, really good question that's never been explored. This is, this is Yeah, you'd wanna, I mean, what [01:03:20] you'd wanna do is take that same person, measure their morning, and then measure their afternoon consistently [01:03:25] across a group of people, and then find out that, okay, here was morning, giving us a little bit of [01:03:30] that false positive range, or a false concern.

Here was afternoon, so if we make this kind [01:03:35] of algorithmic adjustment It allows us to get the morning, which is the easiest time to do it. [01:03:40] Everyone wants to do their fasted tests in the morning so they can end their fast. I get it. But yeah, it would be [01:03:45] nice to be able to kind of have an adjustment on hand to say, okay, well, actually [01:03:50] it came in at 15 microunits per mil, but based on what we see across the population, [01:03:55] if we were to measure this.

Four hours later it would have come back down to seven. Okay, [01:04:00] so you're actually doing great. Okay. Yeah, that's never been done.

**Dave:** Wow Well, this seems like [01:04:05] something your lab would be uniquely suited to do. Yeah, I vote for that We've talked about doing [01:04:10] squats after a meal or going for a walk to lower blood sugar Which ought to [01:04:15] also lower blood insulin we hope Are there other non dietary things we can [01:04:20] do, the biohacks, like red light therapy, pulsed electromagnetics cold plunges?

[01:04:25] What do those do?

**Benjamin:** Yeah, so the only one I can speak to with some authority is cold plunge. Okay. Although I think [01:04:30] everything you've just mentioned does help, including just sauna. It's the interesting quirk of just temperature [01:04:35] extremes. With cold immersion Yeah, I mean, the results are really [01:04:40] remarkable across the board, and I am an unapologetic advocate of cold immersion, but [01:04:45] there is something to be said for, there are two mechanisms that cold therapy will engage, one which is [01:04:50] shivering induced thermogenesis, and then it's mitochondrial uncoupling, [01:04:55] um, and, and the the brown fat being the most famous example of that, but with the [01:05:00] shivering, of course, the, the, the whole body contraction of the muscles, That's, [01:05:05] that's really, I mean, you are exercising.

You are contracting and relaxing muscles in order to [01:05:10] generate heat. Well, that heat has to come from burning something, and a lot of that something's going to be glucose. [01:05:15] Now, at the same time you're shivering, You're also sending a signal to mitochondria [01:05:20] particularly in fat tissue that hey mitochondria and fat tissue I need you [01:05:25] to become less efficient Which which is to say I need you to [01:05:30] burn energy to create heat because heat is a waste product You know and but when the [01:05:35] body gets cold it actually gets a little well wasteful It starts to want [01:05:40] to burn energy just to create more heat.

And so you have this uncoupling where the [01:05:45] mitochondria, it's basically like You're, you're, you're revving the engine in your, your car, [01:05:50] but you're keeping your foot on the clutch at the same time. And so you're getting a high RPMs, [01:05:55] but you're not getting any speed. You're not moving. So you've uncoupled the burning of the [01:06:00] fuel in the engine with the movement of the car.

You know, which is not the same that I [01:06:05] mentioned earlier. Earlier, I mentioned that Ketones help the muscle be more coupled in that case. Now you're [01:06:10] revving the engine and you're seeing movement with the speedometer, but with [01:06:15] cold and with ketones, generally you're uncoupling it where you're revving the engine, but [01:06:20] you're not moving anywhere.

You've uncoupled those two actions. And so cold [01:06:25] therapy is an incredible way, but there also are kind of other hacks. Like [01:06:30] apple cider vinegar is surprisingly effective, but you were kind of saying non [01:06:35] dietary. So yeah, of all of those, I personally leverage [01:06:40] whenever I can take advantage of cold immersion and sauna.

They're exceptional in [01:06:45] this regard.

**Dave:** I do them a lot. The other thing that I notice has a big effect on my blood [01:06:50] glucose stability, which is a proxy for insulin, is [01:06:55] darkness at night. So I'll wear the TrueDark glasses that trick the brain into thinking that it's [01:07:00] nighttime. And if I wear these for an hour or so before bed, especially if [01:07:05] I'm traveling, I have much more stable blood glucose the next day.

And I think just darkness at night [01:07:10] to reinforce the circadian rhythm works.

**Benjamin:** Yeah, it does work. Just to put a really [01:07:15] clear statement on that, one of the reasons East Asians have [01:07:20] such unexpectedly high levels of glucose is because that these are cultures that have the highest [01:07:25] bright blue light exposure later into the evening.

That is one of the reasons there's some [01:07:30] other quirks of physiology across ethnicities. But it is an absolute consistent [01:07:35] feature of East Asian culture. Lots of bright blue light into [01:07:40] all hours of the night, which absolutely will disrupt circadian rhythm to such a point that you'll [01:07:45] have higher cortisol levels the next morning, and in turn, higher glucose volatility.

**Dave:** [01:07:50] Environmental toxins and insulin resistance. Is there a connection?

**Benjamin:** Oh, for sure. [01:07:55] Yeah. In fact, in my, my first book, you mentioned my how not to get sick, which [01:08:00] is this one over my shoulder. One step over outside of the camera is my why we get sick [01:08:05] book. I actually devoted a small section to, as I talked about the origins of insulin resistance [01:08:10] to describe some of the evidence.

Looking at certain molecules [01:08:15] from like plasticizer agents and detergent agents and more and [01:08:20] more even certain pesticides and herbicides. so these are all chemicals [01:08:25] that have varying direct or indirect effects at promoting insulin resistance [01:08:30] where the direct effect is such like lectins. have a direct [01:08:35] effect as a, as a kind of food anti nutrient toxin.

Yeah. Lectins have a [01:08:40] direct effect of causing insulin resistance, whereas some of the [01:08:45] detergent molecules like diethylstilbestrol It's not going to have as [01:08:50] much of a direct effect, but rather an indirect effect by promoting the growth of the fat [01:08:55] cell. And when fat that comes back to the kind of slow insulin resistance that I alluded to earlier, where when the [01:09:00] fat cell gets too big, it starts to generate its own form of insulin [01:09:05] resistance.

Which just as a interesting aside, it actually starts to promote insulin resistance to try to [01:09:10] prevent further growth. But in the process, Starts to kind of spread that insulin [01:09:15] resistance with a couple different signals throughout the body. So yeah, I mean, what we eat and drink and [01:09:20] even, even breathe.

We've published papers working with my colleague, Dr. Paul [01:09:25] Reynolds. We just published a paper, um, late last year, actually, finding, this was an [01:09:30] animal study, where when these animals were exposed to diesel exhaust [01:09:35] particles, even when they ate the exact same amount of food as their litter mates just [01:09:40] exposed to normal room air, Their fat cells got significantly bigger.

So once again, our little rant [01:09:45] earlier, don't tell me it's all about calories. There are these signals, even [01:09:50] some things we breathe, that are going to influence the overall dynamic energy [01:09:55] storage, the balance of metabolism. So yeah, there are Unfortunately, [01:10:00] we live in a bit of a dirty world. And, and where people are able [01:10:05] to, to mitigate some of these risks, now I, I know that in some, some people feel a little [01:10:10] overwhelmed.

I still firmly believe macros matter most. If you can just [01:10:15] focus on one thing, focus on getting your macronutrients. Which is,

**Dave:** give me the right ratios for macros.

**Benjamin:** Yeah, yeah, [01:10:20] well, for me it's very simply control carbs, so don't get your carbs from bags and boxes with [01:10:25] barcodes as much as you can, whole fruits and vegetables, depending on your overall scenario.

And then it's [01:10:30] prioritize protein and don't fear the fat that comes with that protein, and just don't fear fat in general. [01:10:35] But as a reminder, in nature, All protein comes with fat, there's [01:10:40] no exception. So every, as much as we love protein, people talk about [01:10:45] protein, there's always a little part of me that says, and don't forget about the fat, that is supposed to come with that [01:10:50] protein.

The body digests it better, the combination of the two is more anabolic, [01:10:55] than just the protein alone. So macros matter most, and then all these other kind of more micro [01:11:00] influences, that's not to say they're irrelevant, they matter, but thankfully, [01:11:05] if a person is Eating. Fewer carbohydrates. [01:11:10] That is the most offensive macronutrient source.

Now you can have [01:11:15] like proteins depending on the source. They can also be somewhat influenced in a little [01:11:20] polluted, although animal proteins have much lower levels than plant proteins do, [01:11:25] but even still, it's just, as a person controls carbs, you end up. [01:11:30] inadvertently getting rid of things like the anti nutrients, you're going to get far fewer levels of [01:11:35] pesticides and herbicides, just as you're eating less carbs, because those are the main vehicles for [01:11:40] those kinds of things.

And whereas fats and proteins have generally gone through the animal and the animals [01:11:45] cleaned out some of those things on their own, or they're stored in other tissues that we don't eat as much [01:11:50] of certainly the muscle is going to be one of the cleaner tissues in the body. So, Cool. That's, [01:11:55] that's why a person, I would say, don't expend all your energy depending on where you are at and [01:12:00] what your financial capabilities are and your bandwidth to focus on this.

If you are just [01:12:05] managing your macros, you are, I believe, Addressing the variable that matters most, [01:12:10] but these other things do matter, but don't let that overwhelm you, would be my advice.

**Dave:** I like to [01:12:15] outsource my liver and kidneys to cows. Yeah,

**Benjamin:** that's right, yeah, well said. [01:12:20]

**Dave:** They pre filter all of the plants for me so then I can eat them.

And the [01:12:25] results are profound when you do that. Or it can be sheep, if you're not into cows or whatever. [01:12:30] Yeah, no,

**Benjamin:** no, but you, you and I, yeah, ruminant meat. So if it's a multi, a polygastric [01:12:35] animal that to me is some of the best meat you can get.

**Dave:** So we're, we're aligned there. [01:12:40] One of the potential causes of insulin resistance is excessive damaged omega [01:12:45] 6 fats.

Because they go into the cell membrane and they mess with things. How much of this is [01:12:50] caused by carbs versus just eating all this canola, corn, soybean, and all those things?

**Benjamin:** Yeah, yeah. [01:12:55] So, Dave, you are, you are putting me in a position where [01:13:00] I am going to have So, I don't mean for this to sound disparaging.

In all sincerity, [01:13:05] I don't. You can disparage me.

**Dave:** Like, I'm curious. Well, no, no, not you

**Benjamin:** even. Not you. But, like, I don't [01:13:10] want, like, When I say seed oil crowd Like, I don't mean to like, put everyone in a little [01:13:15] tribe here. But there, of course, there are people who, they will say, Ben focuses on [01:13:20] carbs. Well, that's not the real issue, it's seed oils.

I just want to remind people that my main [01:13:25] focus is insulin resistance. It is not other problems. So, [01:13:30] so, within the, the direct realm of insulin resistance, I will claim that [01:13:35] hyperinsulinemia is more of a problem than Then seed oils. Now, [01:13:40] at the same time, I want someone to know that if I'm actually [01:13:45] talking about like liver fibrosis.

Ah, seed oils are actually probably more [01:13:50] problematic. Cancer and damaged mitochondria that feeds cancer cells, seed oils are probably more [01:13:55] problematic. So, as much as it seems like I'm disrespecting the [01:14:00] seed oil view, I actually am not. I'm just trying to kind of stay in my own Lane where [01:14:05] I really am kind of more of an expert.

**Dave:** I respect that greatly and so it's [01:14:10] possible that eating excessive seed oils Contributes to insulin resistance because of cell membrane changes, [01:14:15] but that's not what you're studying You're studying the effects of insulin directly on this.

**Music:** [01:14:20] Yeah,

**Dave:** and so acknowledging what you're an expert in And not going into areas [01:14:25] that you haven't studied, I can only respect that.

Good, good, yeah. Yeah, in my case, I [01:14:30] think it's advisable to minimize those. I haven't eaten seed oils in a long time. Totally agree,

**Benjamin:** [01:14:35] totally agree. But let me, let me just kind of answer the way you framed the question, because I do have [01:14:40] thoughts on it. So the connection between seed oils and insulin resistance, I do not believe that it's a [01:14:45] direct effect.

**Music:** Wow, okay.

**Benjamin:** For example, you can incubate cells with [01:14:50] linoleic acid and they will not become insulin resistant.

**Dave:** That's a pretty good piece of evidence. [01:14:55]

**Benjamin:** Yeah, it is. And I want someone to, to hear, because I've literally done that. I've [01:15:00] incubated cells with all kinds of stimuli. And then you just, it's a simple set of experiments.

Now I'm [01:15:05] going to incubate with some insulin for 10 minutes. I'm going to harvest those cells. And now I'm going to look [01:15:10] at the signaling. The, the, the protein phosphorylation states across a handful of proteins that [01:15:15] will tell me, okay, how well did insulin work? So with a, a, a high degree of authority, I can tell [01:15:20] you, linoleic acid does not cause direct insulin resistance.

It only

**Dave:** gets [01:15:25] fried for two weeks, and then you do it.

**Benjamin:** Okay. So now we're not even putting on linoleic [01:15:30] acid. So this is a really good point. If you start to just put on like, the peroxides [01:15:35] of, of this, that starts to change thing where it is, but just so people appreciate how [01:15:40] difficult it is to work with these molecules.

They are so reactive that they just become toxic [01:15:45] cytotoxic where the cells just start to die. And so you can't really even you to [01:15:50] try to find the right dose is actually. Problematic.

**Dave:** So the highest [01:15:55] expression of insulin resistance is death.

**Benjamin:** Yeah. Well, yeah, that's right. Yeah. I mean, what's so, so what if you're [01:16:00] insulin resistant if you're killing all your cells?

So that's a really, really good point. Okay. That as much as [01:16:05] I've just been talking about linoleic acid there's conversation really ought to be looking at the [01:16:10] peroxides. Right? So, so that's a really, really important consideration that I need to keep in [01:16:15] mind. Linoleic acid alone doesn't appear to cause insulin resistance, the degree to [01:16:20] which its peroxide metabolites do.

I don't know, but I bet it, I bet it [01:16:25] does. I bet there's an effect there. But even still, the way I describe the relevance of [01:16:30] linoleic acid is through what it does in the fat cell. So it takes me back [01:16:35] acid's unique that you can track it. If you eat more of it, [01:16:40] you store more of it. And, and, and this has been shown in fat tissue from humans.[01:16:45]

When linoleic acid gets converted to these more reactive molecules like [01:16:50] 4 HNE in a fat cell, it forces the fat cell to [01:16:55] grow through hypertrophy rather than proliferating through [01:17:00] hyperplasia. And the difference there is that if you have more fat cells but they're small, then you're [01:17:05] insulin sensitive, from the level of the fat tissue at least.

Because small fat [01:17:10] cells are insulin sensitive and happy fat cells. They're very anti inflammatory as well. So, [01:17:15] linoleic acid converted to 4 HNE, one of the more common reactive [01:17:20] metabolites, tells the fat cell, hey, there's no proliferation happening here. You're just growing through [01:17:25] hypertrophy. The hypertrophic fat cell is an insulin resistant and pro [01:17:30] inflammatory fat cell.

So when I talk about linoleic acid or seed oils, [01:17:35] with some caution, I don't like to say that they're a direct cause of [01:17:40] insulin resistance. Like other ones are, insulin is a direct cause, inflammatory [01:17:45] proteins are a direct cause, like in that same cell culture, I can put on cytokines and they're insulin [01:17:50] resistant in minutes.

Stress hormones. I can incubate those cells with cortisol or epinephrine. [01:17:55] They'll become insulin resistant. But if I do it with linoleic acid, like I said, it's not quite the [01:18:00] same effect. But with the, with all of the myriad metabolites that can be [01:18:05] generated, it is very possible that some of those could have a direct effect.

[01:18:10] When I talk about it, I try to be a little cautious. And just say, well, it might, but I don't [01:18:15] know if it's direct, but I certainly believe it's an indirect because of its effect on the fat cell. [01:18:20]

**Dave:** That is so academically honest and controversial. [01:18:25] And it sounds like you don't eat a lot of linoleic acid on purpose either, because it's probably not good for you, but.[01:18:30]

Yeah. Well, and again,

**Benjamin:** when someone, when someone's obeying that first rule, [01:18:35] the control carbs, I believe the vast majority of the linoleic [01:18:40] acid seed oils that people are getting are coming from bags and boxes with barcodes. [01:18:45] So if you're just controlling carbs and eating less of that, the good news is. Your [01:18:50] linoleic, your seed oil's essentially gone to zero.

**Dave:** Cool. Ben, I know that we're running up against the end of [01:18:55] the show because you've got an appointment coming up here and I just want to thank you for [01:19:00] your, your willingness to go against the mainstream in [01:19:05] academia. And to do it with data and science and to not be pissed off all the time about it. Like it's easy to [01:19:10] get angry.

Yeah. You could tell you have this like playful curiosity, like, but this is how it works. And I [01:19:15] love your vibe. I love your knowledge and just keep doing what you're doing. Thank you. You're at Ben [01:19:20] Beekman. com. Your book is how to not get sick or how to not to get sick. What is your [01:19:25] YouTube channel?

**Benjamin:** Yeah. So you mentioned the first source, please guys go to my website.

It's just Ben and B [01:19:30] I K M A N. com. But yeah, my YouTube is Ben Beekman. And then all my social media [01:19:35] is just Ben Bickman PhD. Yeah, I mean, all of this is just my own effort to, as much as I'm an [01:19:40] academic and I love it, I also hate the constraints placed on it where [01:19:45] no one's going to read my peer reviewed journal articles.

And, and why would they, you know? So I wanted to have a [01:19:50] way to just share the, the fundamental research with people in a kind of digestible way. And that's [01:19:55] always been my hope.

**Dave:** Well, you're doing a great job, but keep it up. And you're always welcome to come back on the show. [01:20:00] Thanks,

**Benjamin:** Dave. It's been a pleasure.

**Dave:** See you next time on the human upgrade [01:20:05] podcast.