DANNY LENNON: Hello, and welcome to Episode 379 of Sigma Nutrition Radio. As always, I am Danny Lennon. And, as usual, I'm here with Alan Flanagan. Alan, how are you today, sir?

ALAN FLANAGAN: I'm good. I'm looking forward to this discussion.

DANNY LENNON: We're delighted this week to be joined by a special guest on this week's episode, Dr. Spencer Nadolsky, and we're going to be talking about a large area with a lot of context and nuance to get through. We're going to be discussing obesity and chronic disease risk and a number of sub topics that relate to that. But first, Dr. Spencer Nadolsky, welcome back to the podcast.

SPENCER NADOLSKY: Thanks, I think it's been five years or something like that, I don't know, it's been a while.

DANNY LENNON: It's been a while, so can you maybe just give some people who might be unfamiliar with your background, just a quick overview of expertise and how that kind of fits in with today's topic.

SPENCER NADOLSKY: So I got in undergrad my exercise science degree, then went to medical school, that's

pretty general for everybody, and then you specialize. I decided to do family medicine, which is like the broadest, so I did family medicine, and then specialized further into obesity medicine, and then added in lipids as well, lipidology. So the difference with me was very strong background in sports nutrition and exercise, and kind of the idea of using lifestyle as medicine as much as possible. But I have a very keen interest in pharmacology too. Basically, there's no such thing as a cardiometabolic medicine specialist, they're talking about making this type of specialists which would be a combination of endocrinology and cardiology, and leaving out in the endocrine part, like, thyroid cancer and other pituitary type of disorders, probably like Cushing's and things like that. And then, in cardiology, you would leave out more of the electrophysiology and more go to cardiovascular disease prevention and treatment without being an interventional type of thing. So you'd combine those two things and come up with cardiometabolic medicine. I kind of carved it out for myself, doing kind of broad medicine with a lot of lifestyle medicine, because obesity is a big component and lipids are a big component, I have a lot of good friends who are cardiologist, very specialized atherosclerosis, researching cardiologist, physician friends, along with endocrinology friends, in fact, my brother is in that realm as well. I try to combine it into one using basically lifestyle and also technology, I do all telemedicine now. I have a team of like 25 coaches, dieticians, I work with at RP, and then I have my own handful of a 200 to 300 patients who I work with personally. And then, of course, my meme, so I specialize in memology.

ALAN FLANAGAN: Yeah, memology.

SPENCER NADOLSKY: Yeah. Interestingly enough, just a tangent, I've come to grips that, like, this job that I have is probably a dream job, because I'm able to help more people – I get messages all the time that, well, you've fixed my cholesterol, and they

weren't even my patients; helped me lose 50 to 100 pounds or whatever it is, and I was like, I don't know, they didn't pay me a dime, some of them did through some of the programs we offer. But interesting stuff, and I get to make funny jokes about it while doing it. So that's pretty much my specialty.

ALAN FLANAGAN: Demonstrable causality of memes working.

SPENCER NADOLSKY: Yeah. Well, the other thing is, I am involved with a big research study, looking at the effects of ketogenic diet induced hypercholesterolemia on progression of atherosclerosis. I think that's going to be pretty cool.

ALAN FLANAGAN: Is that Dave Feldman's?

SPENCER NADOLSKY: Yeah, him across the table party line said, hey, this arguing on Twitter and whatever is just meaningless now. This is a public health issue. I think it's reasonable, we should at least see, from a safety perspective, what the heck is going on in these individuals who get an LDL cholesterol of above 200 milligrams per deciliter. I can never remember the millimoles for you guys. But basically, those in the familial hypercholesterolemia range, secondary to diet not genetic, so I set up this full – it's going to be pretty cool.

ALAN FLANAGAN: I think we've kind of briefly talked about this before, but we spoke about it with Dave when we had the kind of lipid debate, the LDL causality debate, and my sense was just like one year – is one year enough? I mean, if most people in the study do have this, like, Dave Feldman range of LDL, and they're 300 milligrams plus, maybe, but if the mean is kind of around the 200-mark, then is a year enough?

SPENCER NADOLSKY: Right, I didn't think so before, but after speaking with some of the researchers that do subclinical atherosclerosis progression studies, we were adamant that absolutely, if there's an effect, we'll see it, that's how sensitive the –

that's what they said, and I'm trusting them because I'm only foraying into this, and I wouldn't call myself cardiovascular imaging, subclinical atherosclerosis imaging expert; more so, I call myself the cardiometabolic type of disease expert with lifestyle, trying to combine all this and get the experts together to come to a consensus of what a good way to study this. They were adamant that if there's an effect, we shall see it, but there's some other things, and once we get the IRB approved, we can kind of divulge a little bit more, because it's really fascinating.

ALAN FLANAGAN: And the participants are going to, if I understand Dave, are going to be from this kind of low carb community, right? So they're going to be all ready, it's not a dietary intervention. You're taking people who are doing this diet day to day, and then you're seeing whether there was any progression from baseline to 12 months?

SPENCER NADOLSKY: Yeah. IRB wouldn't approve of intervening and causing a 200, I mean, just ridiculous – I mean, they wouldn't allow it. So if we show a safety, if there's like, hey, in a short period of time – it's not going to be like they're going to have heart attacks, well, unless they have cardiovascular disease already, and these are healthy folks who have been following a ketogenic diet with elevations in LDL cholesterol and everything else pretty much normal for at least a few years and watching the progression. There's some interesting stuff about that, because, like, because of how atherosclerosis starts and progresses, you really got to have people that – you have to have some tiny little bit of plaque to see the progression; otherwise it becomes incidence of, once you get the first one, you see kind of a prevalence in this population, and then there's progression, but then there's also incidence; if they don't have any plaque there, what's the possibility or probability that they'll actually develop something a year – it's pretty low. So progression, once they have it, is actually more

easily studied. So this is going to be interesting all around, because, in fact, they've never done this in familial hypercholesterolemia, which is surprising to me, because you would think that that would be something someone would want to see. But anyway, we can do a whole podcast on this. Sorry, we just wasted 10 minutes. Everybody's listening. Why aren't we talking about obesity?

DANNY LENNON: Yes. So to start this, and I don't know if you guys have any differing thoughts, but to get into some of the real nuances, we need to first get to at least some basic premises that people agree on, and there's probably two ends of a spectrum here that we can discuss. So I was thinking maybe that's, if we start somewhere there of looking at, well, why are we having this conversation, what is the typical associational relationship that we see here.

SPENCER NADOLSKY: Yeah, why do we even care about obesity, right? What's the definition of obesity? How do you define it? How is it clinically – how is it diagnosed? So I think those are important things. We could probably start there, but I think on just the very basics, people will just say, obesity, which we'd probably say is excess amount of adipose tissue, obesity increases your risk of XYZ disease and dying. That's what the statement would be. Of course, we are going to have to get into the nuance, because what does it even mean, obesity? Are there different types of obesity? Does it matter how severe the obesity is in terms of how much fat or does it depend on the severity or where you're storing the fat and consequences of, so I think that's where the conversation starts. So obesity defined anthropometrically, I think everybody knows this, but like via BMI, and, of course, there's a lot of arguments against the use of BMI, but basically body mass index using your weight and your height, kilograms per meter squared, it's found that that's a decent, not a perfect, but a decent marker of adiposity, general body adiposity, your body fat – decent, when I say decent, I'm technically

overweight, but I have a six pack, so according to BMI, so again, it works okay in a population level. So then what are the levels of BMI? So basically 25 to 29.9 or so or under 30 would be considered overweight. So then normal weight, you could kind of argue somewhere around 18 to 25 or 24.9 is considered normal weight. I put that in quotation because we could get into what is normal anyway.

So then overweight is at 25 to just under 30. Then we used to be just, you have obesity, if you're over 30 BMI. And then over 40, BMI was considered morbid obesity. Now, we've gotten into making it more specific, where you get into classes of obesity, which would be a 32, just under 35, would be class 1 obesity 35 to just under 40 would be class 2, and 40 and above would be class 3. And then they can even go further with different classes, but that's generally how we do it now. So that's basically it, so then, what people say is obesity defined as 30 and above, you're at risk for all these things and dying early. That's kind of how people say, but, of course, we're going to get into the nuance.

ALAN FLANAGAN: Yeah, I think in terms of the definitions, I think that's really helpful to lay out, I think that one of the problems with the current discourse is that it likely stems from considering or characterizing obesity, one word, as a single phenotype, and it's not right, and we will get into this. There are different phenotype presentations that relate to adipose tissue distribution, the metabolic activity of that adipose tissue that can relate to distribution, and a number of complex interplay of kind of biological, social, environmental factors that can all coalesce to create risk in an individual. And within each of these classifications, or within each of these respective phenotypes, there are kind of aggravating and mitigating factors as they relate to other health behaviors. So just a quick example, an aggravating factor would be sedentary behavior and a higher BMI, whereas an attenuating factor would be high

levels of cardiorespiratory fitness. And at certain levels, within particularly the kind of, we could call it this gray area of BMI, when we define adiposity or obesity by BMI, we have this gray area, certainly, in epidemiology, maybe between say, 27 and say, kind of, depending on the fitness level, under 35. Right? So in that range, BMI can sometimes be an insensitive metric in relation to overall risk, because it may not account for fitness levels, it may not account for adipose tissue distribution, particularly in women.

So as those are laid out, as Spencer laid out, these classifications are just rough guides. Part of the problem with the conversation is people are treating them as if they've always been viewed as some sort of kind of static, sacrosanct way of considering these things, and I wouldn't say that anyone in research or clinical practice has. And so, within each of these are these various kind of nuances that we can consider, and I think that perhaps the most important of those are the kind of predictive relationships in epidemiology between various of these kind of factors that relate to weight and BMI and risk, and then, from our understanding of kind of interventions and experimental research and clinical research, what we can say that helps us actually explain some of these nuances a bit further.

SPENCER NADOLSKY: Yeah, so you see two people at the same BMI, but one person has stored most of their adipose tissue on their hips and legs, versus one person that's stored it around their abdomen, and you say, well, they both have obesity according to BMI, they're both going to be at risk for XYZ. And we find that's not at all the case, as you mentioned, so body fat distribution, things like that. So how do we kind of refine that diagnosis a little bit? Well, because of these differences in where we store the fat, we can also do clinically, what's called a waist circumference. And really, this is just kind of a surrogate way of measuring visceral or abdominal adiposity or fat, and we can probably get into the path of

physiological differences of why the fat is different where you store it. So yeah, we can then go and go, hey, wait a second, why don't we see where they're storing the fat because that actually plays a role in how you stratify their risk. And yeah, it turns out that, waist circumference does change things quite a bit, until you get to a certain point over 35 as you mentioned. Once you get over 35, it still tracks along the way, but once you're over 35, you do have a lot more adiposity than...

ALAN FLANAGAN: Those nuances do in the epidemiology seem to fall away, once we are over 35, where it's a metric that is providing, independent of distribution at that point of adiposity, the risk is higher, and that may just be simply the volume of adipose tissue with spillover and stuff like that means inevitability, you have an increase in some of these more kind of metabolically harmful depos.

SPENCER NADOLSKY: Clinically though, what you see, the common thing is someone goes in, the doctor's not even looking up from their computer, they're typing away, the MA nurse or whoever did their weight and their height, calculated the EMR, calculated the BMI, they put obesity on the chart, maybe they said something about it, maybe they didn't. The patient then sees they have obesity diagnosed, and that's it. And then they say, you got to lose weight, and that's it, and they don't necessarily stratify further. What should be done clinically is BMI really should be used as purely a screener, and people may say, well, why don't we use a DEXA scan, why don't we use these better measurements of adiposity. With a DEXA, you can see where the fat's stored, you can see how much. It doesn't discriminate, like, if you have a normal BMI, let's say, a BMI of even 23, and you actually are under muscled, and you have more body fat percentage despite BMI being normal, that's also a big hurdle. So why don't we just DEXA everybody? It really comes down to cost and feasibility and efficiency. Ideally, people would walk into the clinic and we'd have some sort of,

whether the tricorder or whatever gizmo thing, you basically use your phone and you'd click and you'd see everything with somebody walking through the door. Yeah, ideally, we'd have that, but we don't, so BMI is a decent marker to begin with, but it should only be used as a screening method, and then further the doctor, physician, whoever's looking at you, should assess everything else, muscularity, and then consequences of or signs of end organ damage, and all sorts of different things from the potential obesity.

DANNY LENNON: I think this is probably going to be a recurring theme throughout some of what we're going to discuss of different tools being used appropriately, either in practice or how people interpret things that are meant to be used at a, say, a public health level versus at an individual level. So, for example, as you say, they're spent so that BMI, being ideally used to screen people, and then with that, that would then going to cross reference that with other measures to give a clearer picture for this individual what's their actual risk. So at this point, we've at least alluded to that with increasing adiposity, and certainly as BMI gets very high, or we get into those classes, class 2 or class 3 of obesity, we're getting to a point where there's this very clear association with increased chronic disease risk. However, maybe we should get into some of that pathophysiology that you mentioned of, well, why do we see this risk, and there's probably two separate questions here that one is essentially the metabolic state that is obesity, being more than just simply just more fat mass around; and then the second part of that risk would be where that distribution is. So maybe on that first question, Spencer, can you just talk a bit about what we understand around obesity, and why that as a state is something that confers some of this risk?

SPENCER NADOLSKY: Yeah, it used to be thought that adipose tissue is just inert, it just was an energy depot, basically, for us to use or store literally what we

eat. Then we come to find out that actually it has its own metabolic organ, which is very interesting. So actually, the fat tissue, adipose cells can actually secrete adipokines, different types of hormones that have metabolic effects, where we store the fat, it has different effects of where there's like pollicis, and where it can go in different organs. It's a really interesting thing. So like, yeah, why would some people with obesity start developing type 2 diabetes or insulin resistance versus someone else with a BMI of XYZ? That's the same BMI. Why would somebody do it versus the other person? And it's interesting, I mean, a lot of this stuff isn't really well-known other than at some point, that fat tissue becomes dysfunctional, it's unable to expand any more. You sent me an email kind of discussing the differences between the hypertrophy versus hyperplasia. For some reason, these people that have metabolic, you know, we could get into the metabolically healthy versus unhealthy obesity, but for some reason those who have all those unhealthy things related to metabolically anyway not fat mass related. So just real quick, metabolically, would be things like blood pressure, lipids, blood sugar, those types of things, liver, versus like the disease of having too much mass, which would be things like osteoarthritis, sleep apnea, reflux and stress incontinence, those types of things.

So for those with metabolically things, why is it that some of these people have these metabolic issues, and really, it comes down to, for some reason, these people, their fat cells aren't likely genetically related, but for some reason, their fat cells, instead of multiplying and having numerous small fat cells, their fat cells eventually get bigger and bigger. You can get what's called like hypoxia, low levels of oxygen in the area, and eventually they start spilling over and then start going into different organs. And it causes dysfunction, and also, there's these adipokines that are secreted, that can also potentially wreak havoc on the body. So it's an interesting thing, because it's like, okay,

so this is why some people with a BMI of like, say, like you said, in the low 30s, maybe they're doing okay, because they're able to expand their fat mass healthfully, like, how you're supposed to do it, whereas somebody else is not able to, and then has all sorts of metabolic disruptive cascades going on. Interesting stuff.

ALAN FLANAGAN: I think the hypertrophic adipocytes is quite interesting, because intuitively, you would expect that because they're large fat cells, they're large adipocytes, in your head, you think, well, that should mean there's a lot of room for disposal. But the kind of balloon analogy I've always thought is kind of helpful, it's like, there's only so much you can fill a balloon before it starts to spill over, and in hyperplastic obesity, with these smaller fat cells, they may be smaller in size, but they seem to preserve GLUT4 translocation. So people with this phenotype that are able to preserve glucose tolerance, and remove glucose from circulation, they're also associated, it seems, with an increased adiponectin secretion, particularly observed in women, that's insulin sensitizing, because they're really good at clearing free fatty acids from circulation, you don't have elevated levels of blood lipids, and you have decreased pro-inflammatory cytokine immune cell recruitment, and just an overall better state of insulin sensitivity.

What I think is really interesting with this is the fact that it does seem to be mediated by sex hormones, and that's why we have this sex dimorphism of adipose tissue distribution, where, with this gluteal femoral region, like hips and thighs that you mentioned, we can see this preferential distribution more prevalent in women and in men, and that's believed to be under the influence of estrogens, may create the kind of metabolic environment for these kinds of adipose tissue related functions. And that may be one reason why in the postmenopausal period, we see a big shift in, for example, cardiovascular disease risk in women. And then in men under the influence

of androgens, we seem to have this preferential distribution within visceral depots. I've seen one kind of theory on that, that relates to kind of portal vein theory i.e., if, from an evolutionary perspective, energy was needed to be mobilized in a much quicker capacity, then that kind of visceral depot would have provided that access point for really quick pollicis and mobilization of energy. Whatever the evolutionary underpinnings may be, the reality is we do know that this depot is associated with spillover into the liver into the pancreas, visceral organs, and is the opposite of nearly everything we just described for small kind of gluteal femoral subcutaneous adipose tissue. So we see high levels of inflammatory signaling, high levels of circulating free fatty acids, impaired liver, insulin and peripheral insulin sensitivity, and these kind of metabolic complications, and because – I don't want to make it seem like it's completely dichotomized along sex lines, because in women, for example, with androgen dominant condition, like polycystic ovarian syndrome, you can see this central adiposity phenotype emerge in some of the PCOS phenotypes as well. So it seems to be perhaps mediated by sex steroid hormones, but yeah, interestingly, we get this kind of dichotomy then in distribution and the related metabolic effects of those adipose tissue depots.

SPENCER NADOLSKY: You're absolutely correct in how the sex hormones can play a role. Genetics play a role. There's a big Jiva study looking at like, hey, these SNPs are related to obesity, but decreased cardiometabolic disease, and I think, so okay, I'll just explain clinically, if someone comes in and they have obesity, according to BMI, and their waist is actually pretty small, and they're holding it mostly in their butt and their legs, and they – and we can get into definitions of metabolically healthy versus unhealthy obesity, but let's just say their stark normal health. I mean, and I look very deep looking, not only glycemia, but like lipids and liver and everything else, and, let's say, I could

do a liver ultrasound just for funsies on them, they have no liver fat, clinically, I may not want to be as aggressive with that person versus somebody that comes with abdominal obesity, who has some dysglycemia, their blood sugars are just slightly elevated in the prediabetes range, and their blood pressure's a little bit elevated, so clinically, we probably want to be more aggressive. And you're the epidemiology expert here, but from a risk standpoint, those folks that are metabolically unhealthy and where they have this fat distribution, there are at much higher risk of pretty much everything, including death, compared to those who don't have those metabolic abnormalities. And the good news is, is that we can just help them lose 5 to 10% weight, because as you said, that visceral adiposity tends to be relatively easily lost within that first 5 to 10% of weight, and we can bring them to that metabolically healthy obesity type of status.

So that's clinically how we would look at it. You should. But many doctors right now are just looking at BMI and they really just don't go deep enough, and maybe it's lack of training, like, lack of system resources, lack of time, whatever. But that's, I mean, what you just explained is pretty much why we should stage their obesity, look at it, look at them clinically, look at the whole picture and then some may say, well, then why do you even need a BMI in the first place, and again, it's just part of the whole picture. And yeah, if somebody has a 25 BMI versus a 45, the person with a 45, it doesn't matter, I mean, they're at a point where they just are at much higher risk regardless. And, of course, we still look at those other things, but it still matters.

ALAN FLANAGAN: It still matters, yeah. I think there's just a lot of this kind of unnecessary zero sum thinking has crept into the conversation. And the reality is that’s a problem of the kind of activist component in this conversation, which maybe we can just kind of talk about later, because it is relevant, but it can also sidetrack from the

kind of evidential discussion. But it is part of the landscape, because in some of that activism, like, the situation you just described, is a lot of really meritorious factors that we need to talk about, the idea that someone might experience a lack of due diligence in their risk assessments within the healthcare system, or they might experience direct stigma within – these are things we need to talk about, but we are talking about them, and the conversation is improving now. There will always be a discord between the time at which a lot of people start to have a bit of a critical mass discussion on something versus it trickling down into just your routine everyday healthcare practice. And that's where the activists will never just have an appreciation for that, because it's just like I wanted to change now, and if it doesn't, everyone is in search, whatever, derogatory comments that would be thrown at someone. And it's just like, it takes a while to turn a big ship, and these conversations are happening. It's not to deny that people haven't had unfortunate experiences within the healthcare system, but we need to be conscious of that, but we not need to without throwing the baby out with the bathwater. And I think BMI is particularly one of those where we see ludicrous arguments made about its origins, the reason for its use, the utility of its use, and the reality is it's not a useless metric, it can be very helpful at the population level, like you said, because it translates across most populations.

Now, there are nuances within that. For example, with people of South Asian ethnicity, we know that there is a difference in – their risk is related to a lower BMI, but we also understand that, we understand that largely that's to do with the fact that genetically, people from South Asian ethnicities seem to have lower subcutaneous adiposity, lower capacity for subcutaneous adipose tissue to store. And going back to what we were talking about earlier, this means that you kind of end up with this spillover, and they end up with

quite a lot of visceral and ectopic fat deposition in the central abdominal region, which is the reason why they may have a BMI that, for a Caucasian person, would be defined as the normal health range of say 19 to 24.9, but actually, their risk within that because of adipose tissue distribution is significantly greater for cardiometabolic disease.

SPENCER NADOLSKY: Like yeah, that's the thing that just doesn't make sense. It's like, "oh, because of muscular folks, we should just throw it out," that's the fitness thing. They're like, yeah, I have a page full of bodybuilders who go "BMI is worthless". Guess, how many when I was in the clinic, you know I'm all online now, but when I was in the clinic, out of 100 people, I would have to say, a 1000 people that I would see, it was just a few, it was just really a few where the BMI didn't work, and you could see that they were muscular. There are a lot of people who are like, oh the BMI is worthless. I'm like, well, no, actually, no, you have obesity, you have sleep apnea, you do have some good bit of muscle, but your waist circumference is 42, you have good muscle under there, I do agree. But yeah, the BMI was not worthless. There's one guy, and guess what, this happened, I mean, like, once or twice, and most of the times they were on anabolic steroids. So I'm just kind of like, okay, now I'm talking about obesity, if you're looking in the overweight range, it did happen more often, but in that obesity range of 30 and above, most of the times, they were taking some sort of anabolic steroids. In fact, when I was in college, my BMI at one point was like 31-32, but, pretty sure I had sleep apnea, my blood pressures were slightly higher, despite being a very active wrestler. So I had probably some of those preclinical components of the disease of obesity ironically.

ALAN FLANAGAN: But I think this idea that BMI serves no part, like, if we're going to get granular with this idea of where BMI has these gray areas, if you were to listen to some of the kind of louder activist

voices, they'll obviously say that BMI is completely useless, it serves no purpose.

SPENCER NADOLSKY: It has no bearing on your health.

ALAN FLANAGAN: No bearing on your health, whatsoever. The reality is, if we dig into the prospective studies, this gray area is actually a fairly narrow range, like I said kind of earlier, it tends to be between 27 and 35. That's the range in which there's a gray area where someone in that range with a high level of cardiorespiratory fitness is going to be at a lower risk of cardiovascular disease, diabetes or whatever, compared to someone who's in a normal BMI range, but is unfit, or unsedentary and/or sedentary. And so, these nuances exist in a narrow enough range of BMI that even, for example, some of these studies that have compared BMI, different levels of BMI, to risk relative to cardiorespiratory fitness, you still – the lack of difference between risk will tend to be up to that 35 category. So the overweight category for BMI of 25 to say 29.9, that's a real – that really is an area that doesn't tell us much, if anything, at the population level about health status. But once we're going above the 35-mark, then the picture becomes more clear. And even if you were to take someone with cardiorespiratory fitness at that level of BMI, they'd still have a higher, we would still expect to see and do see in cohort studies, a higher level of risk compared to an overweight category or a class one, or a normal weight category with cardiorespiratory fitness or high levels of fitness.

So this idea that BMI, in and of itself, is entirely redundant across all categorizations is just simply incorrect. We do have probably the overweight category of 25 to 29.9, that's not very – doesn't really communicate anything with regard to an individual's kind of risk as it relates to adiposity, because we can't tell from that metric, what their level of adiposity even is. They might not even have any excess adiposity.

SPENCER NADOLSKY: That's where I would lay – I'm right there around that 27-28.

ALAN FLANAGAN: Yeah. And then, once we get above that, then there's a further gray area that's modified by health promoting behaviors. And then, once we get over 35, it's really difficult to find studies where you don't see, compared to these lower categories, a higher risk. And then once we get over 40, like you said, particularly that recent study that looked at the polygenic risk, where we're getting into fairly clear delineations in the risk, and it's significantly elevated in those classes.

DANNY LENNON: And I think at this point, it should hopefully be clear to people that we're talking about evidence here of just acknowledging where risk lies, we haven't even talked about what interventions look like, for example.

ALAN FLANAGAN: Just around that often BMI, like, the underlying metabolic risk is an interesting place where we can really see that is with diabetes in prospective studies, right? So you can see people who are in the normal weight, or just the overweight category, have greater prevalence; but if they've done fasting measures of glucose at baseline or any metabolic risk, you can see that actually already at that time, independent of their BMI, it was the underlying metabolic abnormalities that were greater in that group. So it doesn't always tell the picture of metabolic health, but it doesn't mean, and it's a redundant metric.

SPENCER NADOLSKY: But no, there's some interesting things about Epi in terms of these observational studies with the BMI. There's one thing that they try to do to correct for the kind of this obesity paradox, and maybe you can talk about that in a moment, but basically, the idea is that, wait, why don't we see this increased risk all the time in certain diseases like heart attacks, heart failure, and other things when somebody has above that 25 to around 30 BMI. And one of

the things that they've done to look at this is they've actually looked at what is their highest BMI they ever achieved, and then they use that to stratify. Because smoking, for example, smoking – people that could try to correct for things like smoking, but smoking reduces your weight, but that probably has an overall increased risk of all-cause death, obviously, heart disease and cancer, so their BMI may be a little bit lower than what it would be, but they're smoking, and they try to correct for these things, of course. And then there's all sorts of things – if you have heart failure, all sorts of disease, you lose weight unintentionally. And that's also where you see some of the issues with weight cycling, like, people that lose weight, you got to really know if it's intentional versus unintentional. Unintentional weight loss is, I don't even know when it's a good thing, I don't even know clinically, I'm trying to think of when is unintentional weight loss good. It's never good. That's why when people say weight shouldn't even be a vital sign, people say that, by the way, weight shouldn't be a vital sign. Guess what, I've caught cancer in multiple patients, because we weighed them, and it was like, well, you lost 30 pounds in the past six months, what are you doing. I don't know, nothing. I'm like, oh shit, like, what's going on here. So there's where a lot of the confounding is in some of these studies. But yeah, and then also, if somebody is muscular, that's going to be a big confounder, BMI doesn't account for muscle, unfortunately. That's where, really, they need to be doing waste circumferences to, at least, within that narrow range, like you said, between that 25 to 35 BMI, should really be doing the waist circumference to further assess where adiposity is.

DANNY LENNON: One of the things that we've mentioned a couple of times, but maybe I think is worth digging into is this concept of metabolically healthy obesity. And so, we're obviously talking about a case where someone is diagnosed with obesity, but from a metabolic standpoint, this

person is healthy from a metabolic standpoint. However, I think probably the question where there's most of the debate here, and I'd be really interested to see your answer, and this, Spencer, is, when it comes down to the lag time of progression of some of these diseases, it can be several decades, let's say. So in a case of someone who has metabolically healthy obesity, one might say, oh, they're at no increased risk to someone else, because of these markers; and then the other side of the argument is, well, may be right now it looks that way, but there hasn't been enough time for enough disease progression to show up. Where should we start thinking more accurately about this kind of idea?

SPENCER NADOLSKY: What you're asking is what we call the durability of the metabolically healthy obesity phenotype. So just to kind of go over some definitions, this is actually a big issue in the literature, because how do you find metabolically healthy obesity. Some of these studies, and you really got, you got to know this clinically, because otherwise you wouldn't pick it up, but some of these studies use metabolic syndrome – or not metabolic syndromes. So the metabolic syndrome, depending on the organization, they define it slightly different. But let's just say, it's prediabetes, slightly elevated blood sugar without having type 2 diabetes, that's one component, slightly elevated blood pressure, it's usually 135 over 85, and there's some slight differences, you could have 130 over 85, it doesn't really matter, slightly elevated blood pressure. And then another one is slightly elevated triglycerides. Most people use 150 milligrams per deciliter. I can never remember the millimoles for you guys. And then one of them is waist circumference, but we wouldn't use that in this case, since we're talking about metabolically as opposed to an anthropometric measure. And then the other one's a lower HDL, or for women, it's 50, for men, it's under 40. By the way, metabolic syndrome is having three of those components. You have to have three of

those components. So what these studies do is they go, well, if you don't have metabolic syndrome, meaning you can have two of those components, they define you as metabolically healthy obesity. That's insane. If you have one of those components, you're not healthy. You don't have – you're not metabolically healthy. You likely have consequences of the obesity.

So what do you do? Well, you got to really refine that stratification down a little bit, you should have zero components of metabolic syndrome. And so, they've done this, and then they, instead of having like, oh, looks like 30 to maybe 50% of people have metabolically healthy obesity, all of a sudden, when you really start stratifying down, when you start looking at the components, if somebody has zero components, really the prevalence is down to like 5 to 10%, give or take; and then if you went even further, because really, having that level of triglycerides is probably too high of a cutoff, you really should be going down to more of 100 milligrams per deciliter, even down to the mid-90s. And that's what some of these people have done, it should be a lower cutoff, because once you start getting up to 100 or above of triglycerides to that mid-range, mid-normal range, you have insulin resistance, and you're not metabolically healthy.

Okay, so the definition really, of metabolically healthy obesity, you should have zero components of metabolic syndrome, zero insulin resistance; of course, they could give you glycemic clamps; you're not going to do that, unless it's a research grade. Basically, they infuse you with glucose and insulin and see how well you dispose of the glucose. And then also, one other thing you could do in a research grade, you could see if they have any liver fat, hepatic fat content, and then that probably takes the prevalence down even further. So it's actually, you could go from oh, it's not that rare, it's 30% to it's pretty rare, it's just like 5% maybe at the most. And I think that's where I would start with making sure we understand

the definitions of what it means, because there's studies out there looking at the durability then of this metabolically healthy obesity, and if they start off with somebody that's unhealthy metabolically, their durability is not going to be there. They're going to transition into that metabolically unhealthy obesity. They're going to be at a higher risk of disease. So when you get really strict with it, those people that are metabolically healthy and have obesity anthropometrically, they don't do that much worse than somebody who is lean and metabolically healthy.

Well, they do – they still have a slightly increased risk, mind you, when you look at these studies, but they do a lot better than somebody who's metabolically unhealthy. So that's why clinically it's important to really stratify this out and look at it correctly. And the durability then, some people say, like, depending on the study, it might be 50% over the course of time, but some people say, no matter what, over the course of however many years, since we don't have enough data to really look at it, over the course of 10-20 years, most people will probably move into that metabolically unhealthy obesity, unless, I would say, if they're doing those non-intentional weight loss behaviors – cardiorespiratory fitness, as you both said, if you're just staying as fit as possible, working out as much as possible, trying to eat a healthful dietary composition, dietary pattern, and getting enough sleep and whatever. Those things again, it's hard to do, but that's what I'd say the conversation – and so it's not very durable, that's why we still, even if somebody has metabolically healthy obesity, we still want to, at least, advise working on some of those other things, even if it's hard to lose weight maybe, they don't lose weight, but at least work on fitness.

ALAN FLANAGAN: I think that definition is really helpful, because the biggest, the crux of the issue with that literature is the fact that the lack of the use of

zero of the metabolic syndrome criteria, which should be a no brainer, but, as you say, it's not, and a lot of the studies have one or two, that metabolically healthy is defined as people with risk factors for disease; and not risk factors that are kind of moderate in the relationship with disease, I mean, elevated LDL cholesterol, triglycerides, impaired glucose tolerance, hypertension, like, these are major, major risk factors for cardiometabolic outcomes. So I think that that operational definition issue is a problem. And yet, when we do dig into this, even if it's defined by BMI, this metabolically healthy phenotype that may not be seen to have the same level of progression is typically in say, again, this kind of overweight, or perhaps even just class 1 phenotype, relative to a normal BMI phenotype. Again, that erodes, once we go beyond that level. So there is a point at which even metabolically healthy, say, class 2 obesity or something like that, that baseline is not going to be associated with any similar outcomes, typically, we would still see that higher risk, it may be – the magnitude of that effect may not be as great as someone who was unhealthy across the board.

But the effect is still different, and there's still a greater prevalence of progression. So I think that is important, because I think sometimes the metabolically healthy phenotype can be overstated to the point where it may infer that one or two risk factors don't need to be addressed. And I think that's a problem, but ultimately, it does come back to this idea that if we're talking about this phenotype, you still, like you mentioned, Spencer, we still want to encourage these kind of broad health promoting behaviors. Right? And some of the diabetes prevention program, particularly, the Indian Diabetes Prevention Programme, or the PREDIMED trial, they were interventions that didn't deliberately target weight loss, necessarily; physical activity and dietary interventions were generally the – and a combination of physical activity and diet, generally, the interventions. And you saw kind

of improvements in the risk for progression to type 2 diabetes, that were independent of BMI from engaging in those behaviors, and again, we're talking about BMI in this kind of range of 25 to 30.

So the emphasis on those behaviors at that BMI in people that maybe have one elevated risk factor, can be associated with really significant reductions in subsequent risk. So the idea that we would just assume that this metabolically healthy state at a point in time, perhaps with one or even two elevated risk factors would be just left because well, they're defined as metabolically healthy for that, you know, one, there's the operational definition problem of saying that it's healthy when someone has high cholesterol or high impaired glucose tolerance. But then there's the assumption that the stage is transient, and neither of those hold true in terms of the evidence that we have in this phenotype.

SPENCER NADOLSKY: When you start talking about the risk, there's ways to do it with a good bedside manner, and what people will say is that you are fat phobic or whatever. No, no, no, you do it in a way that's just talking about facts without using emotion. It sucks because it's hard to lose weight and keep it off. It's tough. We can talk about the weight stigma a little bit, but yeah, it's like, it's relatively rare to have metabolically healthy obesity. And yeah, that's why, if somebody has a 40 BMI, so the higher your BMI, the more adiposity you get, even if you're metabolically healthy, increases your risk of transitioning to a metabolically unhealthy status, your age matters.

So just a quick little anecdote, I wanted to get into some epidemiology research, so I could basically be as cool as Alan, and I was looking at a couple of cohorts which were really interesting that studies have already been done on, and I contacted a researcher who did some of the these metabolically healthy obesity studies, and I was like, hey, you know what, I

don't think your definition was strict enough. I was trying to be as nice as possible. And what it comes down to is that there were not enough people, and this is crazy there were not enough people in this huge cohort – the reason they had to increase the number of components of metabolic syndrome that they were able to have to be metabolically healthy, the reason they had to do those, because it would have been way underpowered. There were 15 people, you couldn't do a study, you wouldn't be able to do it. So they had to increase the components, just to get more people in it. And you have to look at it in this context. Those people weren't metabolically healthy.

So in the clinic, when my patients – no, not in the clinic, but now I monitor them, telemedicine. The people that are, what I would consider, metabolically healthy obesity, they are into fitness, they are into eating well, and they continue those behaviors. And I've been monitoring them for five-six years now, and they seem to – it's durable for now, but they're also a little bit younger, so it's hard for me to say, I don't know what's going to happen to them. What if they get injured and they can't work out anymore? I don't know. So anyway, it's tough, it's tough, to deal with.

DANNY LENNON: But as we've kind of acknowledged already, the fact that there is some extreme claims made on an activism side is not that they're invented out of thin air, as you've noticed that some of these things have some real basis that we could say, yeah, actually, patients aren't taken care of properly in a lot of settings, or, at least, the best way they could, that to avoid the stigma that comes along with this or that they're screened appropriately, and so on. But that's not to say those things don't exist.

SPENCER NADOLSKY: Correct.

DANNY LENNON: And so, if we come down to the position now, and let's say, we were to have everyone listening, at least, agrees with the premise that

at these increasing levels of adiposity, there tends to be an increased risk, and therefore, now we're in a position of, okay, well, what do we do with this. Right? In terms of interventions, what do we do at this position now, what is the best place to start that conversation do you think?

SPENCER NADOLSKY: Here's what you'll see this dichotomy, and you'll hear these little stories, you'll see them on TikTok and Reels and whatever, people telling their story about they just went to the doctor, and, let's say, they're having back pain or pelvic discomfort, if they're a woman, they're having period cramps, or whatever, maybe vaginal bleeding, which is, you know, that needs to be worked up, like, no matter what, I don't care if you have a 60 BMI, if somebody comes in with vaginal bleeding, that's not normal, that shit needs to be worked out. You miss all sorts of things, endometrial cancer, whatever, there's all sorts of things. But the classic things you see are the doctor, in a sense, basically, says you're fat, and you just need to lose weight. And that doesn't even make sense in some of these clinical scenarios that I read, these doctors get sued, and they just keep saying, you just need – and patients will keep coming in, you just need to lose weight and then all of a sudden, oh, they had a metastatic cancer. These aren't the norm. They don't happen every single time, and maybe the weight is contributing to whatever it is, but that's the issue that you see with some of these doctors, or, they just don't even need to – they don't even ask appropriately.

So the way you're supposed to do it, if somebody comes in and, again, if I'm in an obesity clinic, they're coming in for obesity, so it's implied that they want to talk about their weight. But if you're in a general clinic, and somebody is coming in, and you're doing just a general wellness physical, they weigh in, you ask them, again, most people with obesity know they have obesity, you don't need to necessarily remind them, but you just ask

them, hey, would it be all right if we discussed your weight, would it be all right if we discussed nutrition and lifestyle to improve your health. You literally ask, because what, you know, most people have had such a poor experience with physicians in the past that they're anxious about it, they don't even want to get weighed in because they've been – they've had the finger wagged at them before, and they already know they need to lose weight. So first you ask, and I think that's a good way to do it, you don't just say, you know you're fat, you're at risk of dying early, you need to lose weight. You can ask and, if they say no, and you could – you could potentially say, okay, yeah, I know obesity, your weight, I'm sure people have discussed, it can increase your risk of disease, but maybe in the future, we can talk about it more. But usually people say, if you ask nicely, they will say sure, I'd like to talk about it, and you go, what do you know about it, and they may understand that it increases their risk and stuff, right.

So first you ask, right? You get permission. And now you've at least helped them be at ease that you're not going to shame them and wag your finger at them. Then yeah, so then you figure out what have they done in the past, what have you done. Most of them have done multiple different types of diets, and not intensive behavioral change types of programs. It's usually the South Beach diet. I always say the cabbage soup, it's stupid, it's just stupid diet because they're ashamed, they want to lose weight, they're looking for some way to fix their pain. So figure out what they've done, and then you decide the next course of action. So yeah, what do you do about it? Losing weight is hard. This is why we consider it a disease is because it's kind of this relapsing remitting chronic issue that when you try to lose weight, unfortunately, our bodies try to fight us to keep that weight. And when you lose the weight, our bodies will keep fighting to get back at a certain higher weight. So that's kind of why we call it a chronic disease. So what do you do? And we

can have that discussion now, but first it's ask, first it's make people feel safe about it, and then decide the next course of action.

ALAN FLANAGAN: I think all of that is really important context, because the assumptions that we are, particularly in the social media space, that some activism has come from somewhere, is, there's no doubt. It hasn't been fabricated out of thin air. People have had bad experiences. And possibly, I mean, and this is something me and Danny talked about before on an episode we did on the evidence around maintenance of weight loss over the long term, like, I think there has been a failing of the research community to really kind of develop more of this nuance and actually get us into the research agenda, you know, blanket weight loss is still a primary outcome simpliciter for loads of interventions, with no other considerations of kind of other factors, in particular, some of the research looking at predictors of relapse and regain, has identified a strong psychological component with people having high levels of disinhibited eating, restrained eating scores prior to an intervention. Right? Studies still don't screen for this, like, that's a basic failing of a really simple task that can minimize the harm that people are potentially put in, if they are struggling, and they're not appropriately screened. And so, I think there is, as much as we obviously want to have this conversation, discussed through the lens of science and through the lens of kind of like an objective kind of rational, critical appraisal of things, if we're going to do that, it's also incumbent on us to kind of own where science, the research agenda, and the trickle down into practice has less people down, and be really upfront about that.

And I think that that is a big component about this wider conversation, that a lot of what you hear, like you said, it's not the norm necessarily that someone's weight loss, rapid weight loss was applauded, as opposed to someone thinking, hold on a minute here, that this is

potentially a risk for something else, and appropriately referring them. So there are those unfortunate stories. They're not necessarily the status quo, thankfully. But there's so much from the non-diet, nutrition literature, there's a massive body of evidence now in relation to weight bias and weight stigma to the point where the WHO released a report on it last year, you know, we need to acknowledge and embrace those elements that are grounded in the research and bring it into practice. And I think a big part of that for nutrition research generally, and these weight loss interventions is a real absence of qualitative data, and the human voice is really not present in what is ultimately a highly behavioral and psychosocial kind of behavior and intervention. So I think that there's a lot that the research community in this area can do to improve kind of best practice as things stand, and to have that trickle down into actual clinical practice for both medical and dietetics and nutrition.

DANNY LENNON: One of the things that we've mentioned on that podcast that Alan brings up, and I think it might be useful to explore here, was that on those kind of extremes of, on one side, thinking that the solution here always is weight loss, and as Alan mentioned, there's a body of literature that can show that weight loss interventions are not benign, that they carry real risks that we can acknowledge, but then on the other extreme positions where people may claim that weight loss is never appropriate, intentional weight loss should never be a target. And so, when it comes to actual interventions, one might decide to go with some weight loss. But, as we've discussed, there may be some other aspects of lifestyle that can be changed without targeting weight loss. But in specific relation to obesity and what you may do in clinical practice, there's also interventions that include medications or drugs or surgery options and so on. I guess, one of the aspects that I thought was worth revisiting is that concept of, oh well, all diets are bound to fail; weight loss

interventions, therefore, just are going to cause a net harm in all cases; so we need to move away from this and instead just focus on behaviors, but we shouldn't really target weight loss.

SPENCER NADOLSKY: Yeah, I posted the other day, I can't stand these people that parrot that 95% of diets fail, that doesn't even...

ALAN FLANAGAN: It's not statistic.

SPENCER NADOLSKY: It just doesn't even make sense. I looked into the history of where this statistic even came from, it was a small study from 1959, and it really comes down to what do you consider a diet, what do you consider success, what do you consider failure, like, what are we even targeting. And these people that parrot, they can never get into the nuance, and it pisses me off, because it's just like, you guys don't know what you're talking about. But then you have other people that say, of course, diets work. And it's like, well, you don't know what you're talking about either, because we obviously see this chronic relapsing and remitting chronic disease of obesity. It is hard to lose weight and keep it off. Okay, so let's kind of define it, I would say, let's define it as intentional weight loss through purposeful, meaningful changes in what you eat, and maybe how you move or something like that. That would be kind of a good definition. And then, what's the intensity of the intervention though? Is it a self-read book on how to do it? Or, do you have weekly meetings with a qualified dietician, psychologist, and physician over the course of how long? And then how long have we actually followed these people? So the study that they looked at, it was like some meaningless statistic of, like, they either looked at 20 to 40 pounds of weight loss. I don't know what the actual relative amount was, for how much they started, that 20 to 40 pounds is meaningless to me, I don't even know what that means. I don't know what they're starting, like, what was the percent of weight that they lost.

The reason that new studies are looking at more of like, can they lose 5% and keep it off, can they lose 10%, 15%, at those certain cutoffs is where we see a massive clinical improvement. So at around the 5% of weight loss, you start seeing improvement in blood sugars and blood pressure, maybe a little bit in the triglycerides; around the 10%, you see more improvements in those, and then you start getting into improvements of sleep apnea, and all those other different types of things. So we really want to – my definition of successful weight loss is like losing 5 to 10% of your weight and keeping it off for a long period of time. I mean, again, I would say, forever, but we don't have that type of data. So we have to look at studies that have lasted that long, and really not that too many studies have done that, and of course, the people will cherry pick observational studies, which are very hard to, again, with the confounding variables of what was intentional, what was not, what was the intervention that they use, I don't know, did they pick up the cabbage soup diet and that was their definition of trying to lose weight versus did they do comprehensive multidisciplinary weight loss program in some awesome clinic or something like that. So these things all matter. I'm sure you guys talked about the Look AHEAD trial in your podcast, but that's the one I bring up. Of course, I bring that up, and then these people that cherry pick that 95% will be like, that's not a good study to look at him, and, like, why. They never know why. They never know why. They just can't – these people piss me off, but I am sorry.

ALAN FLANAGAN: No, you're right. And one of the things we talked about on the podcast was exactly that, the whole concept of how have these statistics, you could make up any statistic, it could be between 30% or 95, whichever, depends on what you define as success in terms of weight loss, and what you define as follow-up and long-term success.

SPENCER NADOLSKY: You could say 99%, 99.9% of people fail, if it's losing X amount of weight and keeping it off for the rest of your life, most people will not do that regardless of what they do, unless it's bariatric surgery. And even then, there's nothing, literally nothing well. So like, yeah, it depends on your definitions.

ALAN FLANAGAN: Right. And so, you see those statistics largely coming from three to six months studies, some going to a year, and an average weight loss of between three and six kilos over that period, which, as you know, is an unbelievably poor return on investment. And again, I think this is another part where the research community probably also has to put its hand up, and when you look at some of the dietary interventions, no wonder they fail, they're horrible. They're awful, awful interventions, both in terms of what ends up on...

SPENCER NADOLSKY: Yeah, if they're not keto, they're not good, right?

ALAN FLANAGAN: Right, yeah. If they're not drinking butter, why do they expect to have success. So their interventions are often, you know, I think the biggest thing that I took from Look AHEAD was that of the factors most predictive of the maintenance of 10% or more of initial body weight loss at the eight-year follow-up, number of practitioner contacts. With Look AHEAD, they made a huge amount of availability of the practitioners, I think they had, was it weekly or biweekly for the first few months...

SPENCER NADOLSKY: Yeah, starts weekly, and then it goes like every other week or...

ALAN FLANAGAN: And it goes to every month, and then from year four onwards, it was like every couple of months with an extra phone call optional. And the people who engaged with the most available contacts had the most contact with the practitioners in the study, which was MDT multidisciplinary kind of team, were the ones who were successful. So there is something, of

course, that we can and should be taking it out of that, both in the clinical context and also in the research context. And so many of these interventions that we have, it's some sort of kind of low something diet with no real additional kind of support, not necessarily any prescription for physical activity or exercise, and people are essentially just left to their own devices to adhere to whatever the prescription is, over the course of six months. And then we get this underwhelming result, and it's like, what's failing here, is it the actual intervention in terms of the prescription, the actual nitty-gritty of the diet, or is it just the fact that none of these wider variables were included in the intervention and the intervention itself was designed almost or destined, sorry, to kind of result in a fairly poor return on investment.

SPENCER NADOLSKY: The reason I like to look at it – well, the reason most researchers like to look at it, it was a huge RCT, and followed up for longer than any other, and did a very intensive intervention compared to just kind of standard of care. Now, the primary outcome was not weight loss, it was looking at cardiovascular disease events, but we can still gather a lot of good information in terms of other risk factors and differences. And also the other thing is people like see, but it didn't work. Well, now, the post hoc analyses of it show that those who actually lost 10% and kept it off, they seemed to have that mortality or cardiovascular event decreased improvement. Again, yeah, people don't want to look at these things with a fine enough lens, I guess.

ALAN FLANAGAN: It comes back to this problem of creating a false dichotomy, so when you see even papers, published papers that kind of criticize weight loss as an intervention or as an approach, or even in kind of the more popular space, you'll see a lot of these things, of these issues that we're talking about, frames as a false dichotomy. Okay, so BMI isn't predictive all the time, it's redundant, get rid of it. Well, no, that's an incorrect zero sum. And we see the

same, I think, with weight loss, where people will say, I want to challenge the assumption that weight loss results in improved health outcomes all the time. And it's like, well, no one said it did all the time, and if they did, well, then, they're not embracing or engaging with the nuance in the literature.

SPENCER NADOLSKY: Right, like, everybody taking a step, not everybody taking a step is going to get benefit, but not all those people taking a step were going to have a heart attack.

ALAN FLANAGAN: There's a phrase I kind of use before, in a conversation set, but evidence and activism don't mix, and they don't mix for multiple reasons, they don't mix because the activist part will always come first, and because the activism will tend to tie to a very, very core worldview, an ideology that someone has about an issue, that is going to come, that is the cart that will come before the horse of evidence and kind of rational appraisal. And there's no way of really getting around that, and you can make the argument, oh, well, we need a degree of activism, because that brings about change. I'm like, I think that's true, but I think that the idea, again, this zero sum, like, what I see when people veer to the activist side is that it's almost a willful abandoning of evidence, you know, oh well, all science is biased; oh there's no such thing as value neutral, so science is, I can just throw that whole science thing out the window. And it's just like, no, that's another ludicrous dichotomy to create. Again, science isn't value free, but that doesn't mean that it's not the most useful method that we have to try and figure out the world around us in a way that's somewhat accounting for some of our biases.

So I think that idea that unfortunately the activist element of this conversation is compromising the legitimacy of the actual conversation itself, I think what you're starting to see now is a lot of people who could be listening, have just shut their ears because they assume – and this is within the healthcare

space now, like, doctors, dieticians, nutritionists – people are just shutting it out, because they're now assuming that it's all just the kind of crazy nonsense that you encounter on social media. And that's unfair to the actual merits of the argument within the published literature to do with a lot of these issues. And so, that's another kind of reality of activism that activists don't tend to really want to acknowledge is how their rhetoric and approach to the scenario can do more harm than good for the movement that they purport to speak for.

SPENCER NADOLSKY: Right. They basically turn themselves like, all right, now nobody's going to listen...

ALAN FLANAGAN: Now nobody is listening, yeah.

SPENCER NADOLSKY: You just said something so stupid, and then you give fuel to the fire to these other folks who are stigmatizing. And so, it's interesting, because, yeah, you get the same on the other side, where it's like, if you have obesity, you're going to die, you don't have willpower, you just need to eat less. Right? And then the other side would say, well, diets don't work at all, and obesity isn't even related to health anyway, and it's all weight stigma that causes the health issues. It's like, what the hell are you guys even talking about, it's just frustrating, it's really frustrating.

ALAN FLANAGAN: I think, yeah, I think both extremes are fairly ludicrous, and they don't seem to, particularly, I don't think they realize how much they reinforce each other's bad thinking on the other subjects at hand. And look, we see that play out in a lot of kind of issues in society, in terms of two polarizing extremes don't tend to ever really come into the middle, they just end up kind of in this circular argument back and forth that reinforces the ideology of the other. But I think with the whole, yeah, on the other extreme, this idea, you know, weight loss is the solution to all circumstances, it's mandated or warranted in all circumstances and will

improve health in all situations, is as equally problematic an assumption, set of assumptions, incorrect assumptions, as some of the extreme positions on the other end of the spectrum.

And unfortunately, I'm seeing that again, more as a result of some of the kind of weight activists elements have become more vocal and more kind of unhinged in some of the arguments that are being made. I'm seeing this happen. I'm seeing people in dietetics and nutrition just start to go the other way. And that's an example of how this goes against the very kind of cause people in the activist side purport to speak for, and I don't think that's helpful at all, and you're seeing this kind of doubling down on, well, any excess weight's a problem, everyone needs to lose weight, and it will benefit everyone in all circumstances, like, look, that's just not true.

DANNY LENNON: Well said. I'm conscious of time, because we've tried to squeeze a lot in here into one discussion. So maybe before we wrap up completely, I'll leave it to you, Spencer, of maybe, out of all the things that we've touched on here today, if you were to leave people with what are the kind of big take home points you think are most important for them to leave this conversation with, what are some of the first things that might come to mind?

SPENCER NADOLSKY: Yeah, I would say, excess adiposity does increase your risk of XYZ disease – where you store it matters just as much, if not more. You can have a healthy obesity and metabolically healthy obesity, however, it's somewhat rare. I would say, weight stigma can increase risks of all sorts of different things, including misses from the doctor, but also obesity is also an issue too. One's not more important necessarily than the other, They're both important. Diets in weight loss interventions do work, depending on how you define success. You don't always have to lose weight, especially, if it caused more psychological harm from doing so, for whatever

reason. Working on behaviors, focusing on behaviors over the outcomes may be more beneficial anyway, and we should probably do that regardless, because the behavior, we can't directly control the scale or adiposity; we can, indirectly, through our behaviors, and we should probably as practitioners focus on behaviors, no matter what. However, using those behaviors to then intentionally lose weight can be a good idea. BMI is useful, it's not the best, and it's not the most precise way of assessing adiposity. After a certain point, it just, it really doesn't matter in terms of BMI. Low BMI is also an issue underweight. Using waist circumference is a good way to stratify further. Yeah, clinically, the doctor shouldn't just look at a BMI, and then talk to you, they should look at everything, look at the whole person in front of them.

DANNY LENNON: Perfect. And so, for people who are looking to find more information from you and more of your work and where they can find you online and on social media, where should they go?

SPENCER NADOLSKY: @drnadolsky on Instagram, on Twitter. I'm starting to do the TikTok, oh my God, that place is global, but I'm also a part of the Renaissance Periodization crew, I'm their chief physician.

DANNY LENNON: Thank you so much for giving up your time and coming to talk to us about this.

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