Podcast Introduction and Guest Background

Regenerative cardiology is a term that I use because the combination of tools that use to help my cardiac patients and all of my patients, it's not a conventional way of approaching cardiologist.

And these are, I think of them as all tools, that optimize our natural regenerative abilities. When I look at what hormones do, peptides, and then more advanced regenerative tools like exosomes, stem cells, you know, they all have a regenerating capacity.

The reason why we are so resilient when we're young is because our hormones are at its optimum and those are nature's regenerated tools. Welcome to the TBD Fit podcast on Dr.

Talks. I'm your host, Daniel Keeley, and I will be guiding you through this wellness journey in terms of optimizing health and longevity, where we unpack the science, the dos, dos and everything in between.

Come join us. Look forward to seeing you inside. Welcome back to TBFIT. podcast, I'm your host, Daniel Keeley, where my goal is simple. I want to find who is doing best in their field and category and bring the conversation back to light so we can all optimize health and longevity.

Together today, today's episode, super special, we're diving into regenerative cardiology, the intersection of cellular medicine and heart health. Joining me today is a true leader in the space, Dr.

Abed Hussein. Dr Husain is triple board certified cardiologist, an integrative medicine expert, faculty member of the Seed Scientific Research and Performance Institute, which I too am a part of and someone who is truly changing the way cardiovascular disease is addressed.

His approach goes beyond sick care, focusing on prevention, cellular medicine, and living a long, healthy, wealthy life. Dr. Jose, it's a true honor and appreciate you taking the time to speak with us today.

It was genuinely a pleasure meeting you in person back at the Longevity Summit in Austin and hearing you lecture. That's why I was keen to get you on the podcast and to just elongate the conversation, especially around cardiology.

I mean, that's kind of the number one killer worldwide. Before we dive into the weeds here, I'd love for each guest to just give us a background into their story.

What was the impetus of you going into your respective field? I'm trained as a conventional cardiologist and I did that for 15 years up until basically COVID.

And in the beginning of my career, it was all end-of-the-spectrum care, seeing people in hospital, inpatient, outpatient work, and emergency care. And I'm an athlete.

I am a person that believes in diet and exercise. We were never taught anything about that in medical school. You know, I thought that when I was going to medical schools, we learned certain things about it, but it never happened.

Fast forward until I started practicing and I didn't have any time to learn it and finally said, okay, this is, you know, still a passion of mine and it's not being utilized.

And so how is it that this can be integrated into at least a practice and to help people and, and prevent what's happening at the end of the line. So I've seen what happening there.

so that, that led to my first steps into functional medicine and hormone replacement therapy, because that's what informed my decisions in the beginning.

What sport? I've done a lot of different sports over the years. I'd been a climber, outdoorsman, always been about a martial artist. And I practiced Kung Fu and a little bit of Taekwondo when I was young.

What Regenerative Cardiology Means

In college, when UFC was just starting out, I did a bit a mixed martial arts and weightlifting. So it's always like a mixture of things. That's impressive.

You might want to tune into one of my good friends who's a patient. He is an avid climber. I would consider him a professional climbers, he's climbed all over the world.

If he sends us testimonials on top of mountains, it's remarkable. Ilija Zlotnik, who is a wealth manager. Um, He has quite an interesting story. Sounds like you have a mixed bag, which is incredible.

My school was soccer. So I went to a play in your school. So I sympathize with many of our athletic journeys and injuries trials and tricks. I will say that, you know, I'm also a fan boy of Arnold Schwarzenegger, Bruce Lee, Muhammad Ali.

So, all those, they just sort of all fit in to my makeup. There's a lot of innate wisdom there, which I can appreciate too. Let's maybe start with regenerative health, right?

In terms of cardiology, talk to our community about how you think in terms regenerate cardiologist. What does that even mean? Yeah. So regenerative cardiology is a term that I use because the combination of tools that are used to help my cardiac patients and all of my patients, it's not a conventional way of approaching cardiologist.

I think of them as all tools, that optimize our natural regenerate abilities. When I look at what hormones do, peptides and then more advanced regenerative tools like exosomes, stem cells, things like that, you know, they all have a regenerated capacity.

Well, the reason why we are so resilient when we're young is because our hormones are at its optimum and those are nature's regenerate tools. There's very few people that look how to use hormones therapeutically from a cardiac standpoint and so that's That's my stance.

That is the perspective that I come from. I want to optimize patients' clients' hormones, give them the opportunity for their body to regenerate and to replenish and become more resilient.

As much as their bodies can do, along with that are peptides. Peptides are also, you could call them mini-hormones, but they are compounds that our body makes and they diminish as we get older too.

They have even more profound regenerative capacities to stimulate stem cells, natural stem cell, inherent abilities that are body has that we lose as get we older.

And those are abilities we use to bounce back from injuries. So say it's something major like a heart attack or something that's a slow burning sort of issue like hypertension.

You know, we use these tools, these natural tools optimize them to bring back our health and regenerate our resilience, regenerates our cardiovascular ability.

So that's the perspective I use in the content. Love that you pulled peptides mini hormones. They're tremendously powerful, stingingly polypoles. And as you ultimately stated that our hormones are kind of optimized during our youth.

and I believe it's estimated after 20 or mid twenties, growth hormone begins to decline at an average of 15% after each decade to the point of when you're 85, you almost at what, nil?

Yeah, that's right. I do believe that the body has innate wisdom, but with modern technology can replete. We can fill in that gap and we can begin to age gracefully.

Going back to the lens of kind of cardiology, what are some of these regenerative tools? What are these peptides that you're using with a high degree of efficacy in terms of specific illnesses or preventative tool?

If you want to speak in kind through both lenses. When I'm talking about peptides, I am talking the ones that are used from sort of the performance enhancement and bio-optimizing communities, but they're also looking at the ELP1S, you know, hormones and peptide that's from that perspective also.

You know, the GLP ones are, they're peptides. GLPs, terzepatide, ratatratide if that's something that you're using. So there's a lot of different, and these are all FDA approved peptide as well.

The common impression that people have peptidies is often that they are not FDA-approved. They don't have proven data behind them. But there are many peptids out there that had FDA approval and they're used commercially.

And they also, they kind of cross over into performance and things like that. So, to your question, I use GLP-1s, GIP, you know, terzepotide frequently in my practice.

It's almost, you know, one of the mandatory compounds because they're so powerful.

Peptides for Heart Health and Recovery

When you look at the benefits of trazepatide and GLP1, smaglotide, we're seeing reductions in alcoholism mortality, reductions and cardiovascular death.

And this is independent of lowering glucose and weight loss. We're looking at it from an anti-inflammatory standpoint. When we look at experimental data, there's data that they actually have plaque regression, so potentially the chance of lower the risk for heart attacks.

Going to other peptides, looking at Tessamerelin. Tissamerelin is a growth hormone optimizing peptide, a secretagogue. There's data that it will help improve vascular health.

It will reduce the thickness of the intimal media, that lining of of arteries that is an indicator of vascular health When it gets thick, it's unhealthy and associated with plaque, associated atherosclerosis.

As it get thinner, It maintains better health. So when we have patients on testimeralin, we actually see that the intramedial thickness gets thinner and we improve vascular health In addition to that, a new one that's been approved recently, SS31, is a mitochondrial peptide that potentially had really good benefits for not only heart failure and myocardial reduced by reduced ejection fraction, but when we look at endothelial health, it may improve the the mitochondrial function of the endothelium as well.

So, you know, there are a lot of experimental benefits and then I use these on patients and we see reduction of inflammation. We see improvement of of uh the intramedial thickness, reduction So, yeah, there's definite benefits in how peptides can be applied.

It's really just about the context. That's about what specifically I'm going to be treating. So in terms of black regression or black reversal, which you just touched upon, what else do you like?

Do you have specific treatment plans, protocol, if you will, to quickly reverse black that you've found very efficacious in your patient population? Yeah, plant reversal is a tricky thing because it can be done a lot of different

ways.

When you look at what it is that's creating atherosclerosis, it's a multi-layered process. And so you've got to look it from a metabolic standpoint. You've gotta look from an inflammatory standpoint and a thrombotic standpoint Those are the minimum three perspectives you've got to look at, and each has to be addressed.

So if we break that down, the metabolic standpoint is going to the LDLs. It's going be the apobetes. it's gonna be blood sugar. And that's where those GLP1s, GIPs are extremely effective.

And then when you look at inflammatory components, that's where we check C-reactive protein. We're going to look for any sources of inflammation, and then we're gonna look ways that we can mitigate inflammation.

One of the reasons why statins are used so widely is because they impact not only LDLs, but inflammation. So if you use a statin at a low dose, you can probably get the anti-inflammatory benefits without a lot of the side effects.

And now when we look at other options in treatment of atherosclerosis, it's the inflammation component that we focus on. Colchicine, which is gaining some attention as a cardiovascular agent, reduces, inhibits the innate immune reaction to inflammation.

It reduces NF-kappa-beta, and that's a driver for the immune system and the driver force things like IL-6, which are heavily implicated in creating atherosclerosis.

And then a lot of part of it, you got to look at what influences fibrinogen. What's going to create the playlist adhesion to the endothelial wall and then maybe inflation and drive other processes that promote after most grosses.

So, so, you know, when we look at the peptides that might be able to influence that. You're looking at the GLP-1s, you're at terzepatide, and there's crossover.

Those GLPs go to other places. SS-31, as I mentioned, also has an anti-inflammatory component to it. Reduces lipid oxidation and then the secretagogues, the testosterone, epineuralin.

When they improve the way your body is utilizing fat, it will reduce the inflammation that may be being caused by fat. In terms, in terms of dosing, you sideline patients on and off, is there a kind of upper limit to how long you can have someone on there for kind, how are you looking at in in, terms trying to get someone to optimize potentially their lifestyle, or are, are just utilizing something like trance hepatitis in a microdosing perpetuity?

So a lot of it depends on context. Where are they coming to me from? If it's somebody that's already optimized and they have evidence of a little plaque on a scan, like a clearly, or you have some elevated lipids, then yeah, microdose of the GLP-1, a combination secretagon, I like to test the merolin, hypermerolin.

And I would cycle that. That's really the only one that you need to cycle. And then the, you know, a microdose of a GLP-1 GIP can be done in perpetuity as long as they want to.

And, then potentially add on things, add-on agents like BPC or TB4, not because they have, because, they help optimize and promote resilience. They'll keep inflammation a little more regulated and then help to reduce fibrosis.

And then there's this fringe benefit of helping recovery in the rest of the system, helping muscular recovery, keeping them active and reducing overall inflammation.

When I'm looking at somebody that's come to me after an event and they're not well regulated, they have metabolic dysfunction and glucose dysfunction, and their inflamed.

That's a different patient, and I'm probably going to be using medications more aggressively. Similar medications, but they're going be at a higher dose.

The GLPs, VIPs are not going micro-dose. They're gonna be therapeutic. I'll shoot for weight loss, inflammation reduction. Probably going add colchicin for inflammation and reduction, The, the, yes, to create a gods are definitely going to be added on.

We're going look at different mechanisms for weight loss. And then depending on what, what type damage was done to the myocardium, you know, then I'll scale up the dose.

You can talk about an infusion of exosomes. It's somebody that's within the first three months of the miocartial infarction, something like that. has the potential to bring back a lot of the myocardium that might be on the edge.

So now we're underscoring trigger generation. Is it just the exosomes or is it stem cells with exasomes? Does that matter? You know, using stem cells right now as a infusion doesn't seem to have the best efficacy.

So it's hard for the stem cell to land and stay retained in the myocardium. The beauty of exosomes is that they're like homing missiles. And they have growth compounds that actually go to areas of damage and higher inflammation in need.

So they tend to have a better chance of going to that area that's infarcted or inflamed.

Assessing and Treating Plaque Risk

I don't necessarily use stem cells, but if given the opportunity, I will use exosomes. And you mentioned BPC has more net effect. Yeah, BPC has that effect and so does thymus and beta-4.

So they both improve angiogenesis, but they also will control fibrotic tissue deposition so that they can lay down collagen and connect to tissue and it's still a a parallel form, so it reduces scar tissue and that can reduce the incidence of CHF in the long run.

And then TB4 has some data that shows experimental data and it's rodent data, but it shows that it can stimulate progenitor cells with myocardium. It's got to be really high dose, if you can get even a chance of recruiting a little more than they would otherwise.

Yeah, I would say that's a useful tool. And how does the administration differ, whether it's injectable or oral in terms of a cheating? Yeah. I will prefer injectible because the only agent that we really know survives the gut environment is BPC out of the ones that I've listed.

There are some oral fragmented options available for TB4. I'm not convinced that those actually make it past the liver. And then that also is an issue.

Even though BPC survives the gut, how much of it orally makes it pass the first pass metabolism? And so that's why I prefer using BTC and TB for all of these as injectables.

Then scaling up the dose. Most places will start with 500 micrograms. There's no real significant documented toxic effects of going up on some of these peptides like BPC1TB4.

So you could use upwards of one gram, you know, twice a day and then not going to cause, shouldn't cause it. The only side effect you can get with BTC is if it's oral and you're going about those like diarrhea.

So we have, I haven't seen any sort of negative consequences or adverse reactions within our patients. And especially going back to our common thread here of being a former athlete.

We have athletes coming in with stress fractures, any form of fracture, soft tissue injuries, tears. pretty severe and we'll use that exact combination BPC and TB4 or TB500 and it resolves each circumstance almost in half the time, literally like percent faster improvement with frequent injections of those two compounds alone, which has been an absolute game changer pun intended, not only for our practice, but for these athletes in terms of return to sport.

And so what it's doing in terms of cardiology is nothing short of astounding as well, it sounds like. And, and I would add for, so when I, for my post coronary artery bypass patients, they, when they show up quickly after the, after hospitalization, the faster they come to me, The more quickly I put them on my, my peptide protocols and the fastest they get recovery.

I remember 20 years ago, starting out, patients would take six months to a year and a half to have to recover from bypass surgery. The recovery time right now is three to six month.

Unbelievable. It's an absolute game changer. And yes, it can be expensive and it takes higher doses and you've got to be willing to do the injections.

But the recovery times can dramatically improved. Going back to the use of statins, patients kind of know where we stand here. We'll use red yeast extract in higher doses to kind achieve the same level of efficacy.

But in terms of a cholesterol panel, because I mean, this has probably been. the greatest misnomer in medical medicine, which I think has been, hopefully now has, been uncovered and dispelled.

But in terms of a cholesterol panel and how you're looking at that, and you mentioned APO-B and LPA, how much does a Cholesterol panel actually provide you in term of actionable insight versus some of these other markers?

Where do you want to see APo-b, Lpa? where do want see particle size, oxidative markers. I guess, how does everything look in terms of today versus perhaps 10 years ago, or maybe even, you know, one year ago?

I think that we have the tools to make a really nuanced understanding of inflammatory atherosclerotic risk. It's just not being utilized. The community, the longevity community and the functional medicine community.

And arguably the community that looks at statins a little critically understands how to look at it, look at a lipid panel better now than in the past.

And, you know, when I began training, it was just, let's look at the LDLs and the HDL. We barely even paid notice to triglycerides unless they were significantly elevated.

So, now we have better understanding and, the tools are there. Look at the APO-B. APo-b is the true assessment of atherogenic risk. It looks at all the particles.

So utilize that, understand what levels you want to get to. Understand that the combination of looking at an LDL and an APob, because if you look at ratio of LDl to APOb, that can give you an understanding of how big those LDls are.

If the LDLs are big and fluffy, then they're less likely to cause a problem. And that's probably someone you don't need to be as aggressively lowering their cholesterol with.

They're lowering your LDLS. That could be somebody that is probably not going to need aggressive treatment and would be one of those people that. Is at risk for a side effect or, you know, as an aside, I think one the reasons why we don' have as good data on statins is because we've been just treating everybody inappropriately.

And if there was just a little bit more understanding of who to focus on, we'd probably have better outcomes. So that's a person that you don't need to treat and goes to the negative side of the study, not contributing to positive outcomes So, look at that ratio.

1.2 is the cutoff, LDL to April B. If it's below 1.2, they're making cannonballs. They're not making beach balls that bounce around in the arteries. they are making Cannonballs that are going to damage the endothelium and cause atherosclerosis and inflammation.

So that's somebody that would benefit from more aggressive treatment. Then you take that patient and you look

their inflammatory risk. You look a C-reactor protein.

you Look at oxidized LDl. Look it maybe myeloperoxidase. You know, these are all indicators of how inflamed they are, if the immune system is being activated, If they have oxidized LDL in its presence in the serum, then we definitely know that they also have Oxidized LVL and inflammation in their vascular wall.

There's, you know there's a lot of tools to look at and stack risks to understand who's going to be at risk. So we're getting to a place right now where this is becoming more understood.

Tools are there and they've been looked at in silos, but I think that it's everyone still is in this mentality of what's the one marker that we're going to look at.

There is no one market. You've got to be a bit of a detective and you've gotta put together. to understand global risk. And that's how, you know, we're trying to take patients and fit them into a singular process.

We've got to process and, and then we can really impact how positive therapeutic change.

Advanced Imaging and Preventive Screening

Forgive me for saying this, but I think that's where conventional medicine falls short is that we're not just one pathway and usually you see, you know, marker off and here's the pharmaceutical to treat that.

That's not the epitome of practicing holistic health or in a full organ system medicine. We're not just one organ or one pathway. So I appreciate you saying that.

In terms of the ratio, you like to clarify 1.2 for the LDL to APOB ratio. Is that correct? That's right. Above that is indicating a predominance of fluffy, less atherogenic plaque.

Below that, more dense, pro-atherogenic plaques. So is it less concerning those individual markers, say APO-B, just in general? Are you looking at that as a specific number or are you just always comparing that to LDL?

You want it to be 1.2 and then we're in the Goldilocks zone. APo-b is a little bit different. Usually when the APob numbers go up, it's going to more consistent with And so the more particles you have, you're going to have more chances for atherosclerotic events.

And even if they're big, so it will be, it can be used in isolation, but you've got to look at everything in perspective, in combination. What do you often find, now going back to let's call it root cause medicine, driving APOV?

Cause that's tremendously arthrogenic route. So what do find in like inflammation on the three pillars previously, but what are you find that usually driving it?

In our world, it's caloric overload. It's blood sugar, dysregulation, cardio metabolic issues. That's definitely one of them. So, you know, if they're insulin resistant, If they are diabetic, that's going to change the hepatic biogenesis of the LDLs and it's gonna create more small dense apobene particles.

The other thing that contributes to that is inflammation in general. And so that can come from any source. You know the 70% of our immune system is concentrated in our gut.

To not look at the gut is negligent in my mind. You've got to look at the main interface we have with the environment. And that's going to be not only what's growing in our gut, our microbes, but what we're putting into it.

So we got make sure that we assess what it is they're eating and then how that is influencing the microbiome. Then if they have an infection that they acquired from travel or eating something that was infected.

In addition to that, you've gotta look other inflammatory sources. The other ones are going to be nasal passages.

And then, you know, cause these are interfaces that are highly metabolically active.

and then the final one I also look at is environmental toxins. So those are the sort of like a low hanging fruit, and a lot of places just don't take the time to look it.

It's also expensive. I mean, immediately it's not something that gets covered by insurance. In someone that's got consistent regular inflammation and recurring problems, we got to work at these things.

That was an absolute grand slam. You stole all the pillar in my mouth. Because as I off the call, when I went down to the Chicago cardiologist, they've done the road for me.

And when i was expressing those concerns, hey, how can we reverse this? Are you looking at your microbiome, right? Which confers most of your immunity, which then regulates your inflammation?

are you Looking at environmental toxic load? Or are looking an infection? And it was no, no no. We haven't seen evidence that this will impact or reversal.

Here's your stat and here's you're blood pressure reducing medication. To me, this is now medical malpractice and I'll take it one even step further is what about your genes?

Are you doing any sort of genetic report? I only say that because within the genes of myself or my patient, we have a propensity for higher cholesterol, blood sugar dysregulation or higher blood-pressure.

And while genes are not our destiny, Certainly a little elevation can be seen within the patient population that confers some of these genetic sniffs.

I don't do a full genome test and when appropriate, I will do the specific cardiac, cardiac specific, uh, gening panel, but we look at empty HFR in people that are, that have, them suspicious may need to get their APOE genotype looked at.

I look at that. If they have early family history, I'll look even at factor five and factor seven, you know, to see if there's an early thrombotic risk.

So, yeah, we check genotypes and we look gene SNPs regularly here, and it's part of our assessment. When your cardiologist and your primary practitioners say, we don't look at that because there's no, nothing that says it really helps.

That's bullshit. There are meta analysis that are being published in Journal of American of Cardiology. This is the standard and looking at things like particulate air matter and its effect on the cardiovascular system.

They're looking at pollutants and telling us that that increase in risk for all cause more calories. So that data is out there and it's in journals that are widely read and easily available.

I don't understand why it is not being adopted more readily. I think it's because most practitioners are reluctant to continue their education or stay current, unfortunately.

And in our modern area, we're just bombarded with stimulus and our go, go go mentality where everything is just stress inducing. It's just like to learn or to have to, everything's, just one more thing.

But that's why I appreciate the breadth of what you're bringing today's conversation. I've done all these panels that you suggested. In fact, if I look at my genes, I lost the genetic lottery.

Very high risk for cardiovascular incidence. My great grandfather died in his sleep in Sicily at 104. Same genetic lineage, grandparents moved to the urban industry of Rome, both passed from cardiovascular incidents at 84 and 86 respectively.

the environment certainly plays a huge role. This notion of epigenetics is really the driver behind what I attribute to be most cardiovascular incidences.

I look at my CAT score, it's at a zero. So we can take steps to practice preventative medicine, which some of these peptides that you mentioned, not only do they reverse, but it can help prevent incidences from occurring.

What is your thoughts on, so we'll suggest patients to do a life screening, if you're familiar with lifelinescreening.com. So you mentioned the CIMT as an example.

That's right. Okay. I know what you are talking about. Yeah. Like any sort of kind of arterial screening. Are you suggesting that with patients, how valid is that?

Are there certain other screening tools that you really like that can help with practicing again, this notion of preventative medicine? Yeah, the lifeline screening is it's easy access, but I think we can do better now.

You know, the idea of lifeline screening is that it looks at the carotid arteries, then it does a screening of the abdominal aorta. Maybe I think it doesn't need KG or something like that.

But you know the, you need to look at that because we need see if it's dilated and that's an easily preventable cause of death. All right. And it indicates that you have some connective tissue issues in the way your body is recycling sort of connectives tissue.

The carotid arteries, that was used as a surrogate marker for your coronary arteries.

Reversing Atherosclerosis and Hormone Support

That was because we didn't have a good way to look at the coronal arteries at that time. So we said, let's find a cheap way that could be reproducible to get a secondary idea of what might be going on here.

So it goes way back to before CTA, CT angiograms are even really used for coronary arteries. So let's look at how thick this is and that can tell us, okay, the blood flow over here is very similar to here and what's happening here should be happening.

There is some correlation, but this very operator dependent, meaning the sonographer has to be really good and it varies from the Sonographer to Sonographers.

That may change with automation. We may get more automated measurements that will be more universal, but it's still, you know, not looking at the coronary arteries directly.

And we now have the ability to do that. You can do a CCTA, which is a coronal CTTA angiogram of the corner arteries. Yes, it involves some contrast and there is little bit more radiation.

In the right hands, It can be done. It could be the equivalent of just two mammograms. So you're not getting blasted by radiation if you do it at a good center.

With that, you can look at the coronary arteries, the wall, and if there is a sclerosis, degree of it. And it tells us composition of what's inside plaque that might be there.

That's the ideal test to go with. It's not designed necessarily as a screening test, but it is worth doing because if your suspect that there's something wrong, it's a test that's going to tell us what is wrong.

A calcium score is just looking at calcium, and it's missing all of the cholesterol part of a plaque. So if you have a high calcium scores, it tells us that you've got a lot of plaque and you really need to look at that seriously.

And you need do the CCTA so that we can see exactly how much soft plaque or cholesterol plaque is there. If you

had a calcium scoring and its low, It doesn't eliminate that there's not soft plaques.

All right. So, in a person like that's got a family history or risk factors, and they're in their 40s, they do a calcium score and it's low or zero, that person might be fooling themselves.

They might get a low score because there's no calcium, but they might have a lot of fatty plaque. And they may have one that is in particularly dangerous spot and could get missed.

That's why I think that CTA is the optimum test. Now, if you had an old calcium score and you do another one, that's reasonable because you can get trajectory and concede.

If you've developed a lot of calcium, then know that you have some stuff that wasn't detected before and probably need to get further testing. If it's about the same, then maybe that's an accurate test from the past, and you don't have a lot of plaque.

But there's a line of unknowns here. And when you're working with cardiovascular health, unknowns are not what we want. We want to know definitive risk, we wanna get as much information as we can, because the last thing you wanna do is, for the sake of a little bit of money, risk dying.

Yeah, I couldn't agree any, you said it best. Definitely. This is the notion of practicing preventative medicine and hit death is certainly prudent. I did a CCTA as well because I had some concern, obviously genetically.

And so they had suggested I developed an arrhythmia, which I don't know if that was from sport or from infection, both I think possible, but needless to say is that leaky heart, quote unquote.

Had some regurgitation. Okay. one of the valves, I guess, was had collapsed or fused. And so upon further examination, things look normal, but certainly things that, that I just need to monitor.

and I think it would be prudent for everyone to get imaging done. So the two imaging, obviously the CAC is the calcium score that you mentioned. It's important to cut people one step further to assess for soft black, which is kind of a more dangerous thing rupture.

Because plaque is contained, it's relatively safe. The body has kind of sequestered that. And you said there's contrast in radiation there, but anything else to consider?

I mean, I do think that this is useful. If you, if you do see plaque there then that can influence what kind therapies we do and they influence brain health too.

Because it not just, as much as I love to say, is all about the heart. There are other parts of the body, you know, so the brain is important. So let's see what, what flow looks like to the brain.

So this gives us a little bit of an idea of that. You know, echocardiograms are not invasive. It doesn't have to be done on everybody. But I think that if there is a murmur or if it's a useful test, if you're looking at longevity and you are looking performance, then a VO2 max performance test is useful tool because then you can look at lactate thresholds and then also look peak performance.

That's exactly where I was hoping you were going. So that's another one that we were doing with our patients and that is critical to assess your engine lung capacity.

I think breath is often overlooked. We breathe in 11,000 liters of oxygen per day and the question is how clean is that but also your capacity? Because I thing the mitochondria can only live up to 5 seconds without oxygen and then they die off.

So breath, breath work, lung capacity, just the healthier lungs is also critical. And for the patients who are not tuning in video, I believe you were pointing to a CIMT.

So that's another one that is readily available that we will send out for our patients. But all great, great options to get a very robust picture of what you can do to, to optimize your cardiometabolic profile.

I'm partially biased in terms of my focus the last 10 years has been in term of our microbes because obviously we're more bacteria and we are just scratching the surface on that kind of interplay between our microbiome and all biological systems of the body in each organ.

In terms of reversing now, atherosclerosis, I know you already spoke to that. We've already seen that in our patients. How much is actually possible? You know, can you bring it down to zero?

Can you make this factor down, especially now in lieu with some of these magical peptides, which to me, there's nothing magical there. They're messenger molecules and they're very potent, very targeted and very good at what they do.

But how much reversal have you been able to achieve or seen in your patients? There's multiple ways to do it. So if you look at it from a traditional medicine standpoint, it's documented that if your drive an LDL, the A port B down really low, down to the 20s, you can get plaque regression.

And we've documented this in previous practice that I worked in where we would use high dose statins and high-dose PCSK9 inhibitors, Rupatha is one of them.

We could get black regression, but soft plaque mostly. You would get reversal of inflamed plaque, and so the reduction of inflammation would reduce the size.

And then you would also get reverse cholesterol transport. You'd affect some of the natural processes so that we pull something out. So that's been documented with standard therapies.

The use of peptides in reversing plaque is still pretty new. This includes GLP-1. You know, that data is experimental and it's probably two years old, three years, old where it really started.

We started looking at it. So, you know we do get plaque regression and the question is, do we have to do it with aggressive LDL reduction? My belief is that if you really want to impact it quickly, then you do both.

two years of aggressively lowering APROV plus adding things that improves reverse cholesterol transport. That's a combination that is optimal for being able to pull out LVLs.

And so we know that stock plaque can be reversed. It's the matter of what they're willing to take and what doses we can use.

Foundational Supplements and Endothelial Health

The question mark, and I think still where it's difficult, is the calcified plaque. Calcified plaque, once it gets deposited, really hard to remove. What you said there, this kind of, again, the medical misnomer of trying to get cholesterol down to zero.

Let's go maybe into hormones. How does that impact our sex therapy hormones? I know there's definitely studies indicating higher LDL with higher IQ, increased mortality or morbidity.

So there is. How does hormones now play into this? I know you mentioned at the beginning, bioidentical hormone replacement, TRT, testosterone perhaps is the most cardio protective model that we create.

So looking at through the lens of hormones, how are you kind of assessing everything? Testosterone is part of the treatment. I mean, it influences reverse cholesterol transport, influences vascular health.

It is one of those hormones that I think should be used therapeutically instead of figured because of some

inaccurate studies. My view is that I want to maintain testosterone.

And then if I'm using high dose lipid lowering therapies, I am definitely going to keep testosterone and then I will try and withdraw those lipids therapies once we get to a safe point.

So it really depends on what… timeframe that they want things to be done in. Remember, this took decades to get to, and it's not going to reverse in two years unless we get really aggressive.

So if they are okay with a less aggressive approach, then we can stabilize, not be as aggressive, For lipotherapy in terms of natural nutrients, how many patients do you have on a foundational program that would encompass, let's say fish oil or pro resolving mediators, niacin, CoQ10, which is predominantly reduced in the use of statins or preservation of muscle.

What are some like foundational considerations that you had with your patients? Yeah, definitely Omega-3 fatty acid, O and D, CoQ10. What else? I don't use niacin as much.

If I use it, it's in low doses because that can influence a little bit of that lipid profile, but we don' want to use in high doses cause that could actually promote inflammation.

So yeah, those are some of the ones that are foundational that I work with. I've used a lot of bergamot and I used tangerine and nobelatin, not as much of the red yeast rice.

Cause I think that that's lost a lot of active ingredient. And so, you know, it may not be as potent as it once was. Are you doing anything for endothelial health, like the production of glycocalyx, any products or any kind of ingredients that you like there?

There's a compound called rahaminin, which I Think is a really good, really Good compound. I like that for glyco calyx. That's a whole other podcast going into glycocalyx.

We can do that another time if you want. Yeah, I know. I, know we're approaching time here. So I genuinely appreciate how you've been very open with your time today and everything that you been able to share and bestow.

It's, it's refreshing to hear that cardiology isn't stuck in the dark ages anymore. And there's there is often for regeneration, which you're on the cutting edge.

Well, appreciate kind of. How your practices evolve and the tools now that you're integrating with your patients to be able to achieve a very high level of reversal and success to kind of wrap everything up.

Any specific takeaways, any big bullet points that want to instill for our community. Patients are not going to fit into one process. Think about multiple processes.

Key Takeaways and Closing Remarks

Look at the lab tests and correlate those labs with the processes because they're there, they are easy, easily accessible, and that can give you some understanding of how you can nuance your care.

The last question that we end every episode with is if you could have one superpower, what would it be? Oh, you know, different times of my life, that's probably would have said different things.

Right now, sometimes I just, I like the idea of being able to fly. You know, that's the one that I say and how many guests have also said that, but what was beautiful there is you said at different points in your life, you would have said different things.

And I think I recall very quickly, a quick anecdote where I had gifted a book to one of my employees and it didn't

really, it wasn't for him. But he read it five years later.

He's like, Daniel, I needed it. Five years. Later. Not at that time. Our bodies are fluid. We're constantly evolving. Were constantly changing. And I think that's a huge takeaway given what you said to connect the dots is understanding that medicine can be personalized with the tools that we have and not everything is for the right person at the time and sometimes we need something differently at a different time, and it's what always worked in the past doesn't mean it always works in And I think one of the biggest components is make sure that people are staying relevant with screening, right?

With tools that we were just, simple, you know, CMP, CDC, full lipid panel, obviously different tools, that allow us to stay ahead of our risk factors rather than practicing reactive medicine as a consequence.

So I appreciate everything that you've shared today. Where can our listeners find you and learn more? Oh, you can find me at Boulder Longevity Institute.

That's where I see patients. And then I'm usually out on the lecture circuit. You can see me, what is it, New Bio Age. They have two lectures a year. I'll be at Vibrant again.

Yeah. This was absolutely wonderful. Again, I appreciate your time. I look forward to seeing you again in the flesh upcoming here. It was a pleasure to meet you in person and I looked forward.

To continue the conversation for everybody. Tune in. Thank you. Hope you guys had some very big takeaways. The message certainly is also hope, but we can undo the damage that has been done.

So keep tuning in until next time, stay healthy. Thank you for tuning in to the TBD Fit Podcast on Dr. Talks, where we unpack all things health and longevity.

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