Telomeres In A Peak Performance Practice: Practical Questions Answered On Measurement And Dosing Transcript

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Introduction and Guest Background

Welcome to another episode of the Telomere Summit. I'm your host, Doctor Joseph Rafael. Today, I have the pleasure of speaking with an esteemed clinical colleague, Doctor Sanjeev Goel.

Doctor Goel is a medical physician and founder of Peak Human Labs. His mission is to disseminate knowledge of the latest cutting edge medical tools and science to help more people live in a peak mental, physical and spiritual state.

You can learn more about Doctor Goel at Longevity, upcoming TCA. Welcome, Sanjeev. Welcome. Thank you so much for having me. And really, you know, I think I'm quite honored to be, on the summit and, quite a amazing cast of, interviewees you have.

So, thanks again. Well, it's great to talk to you. I know we we were catching up a little bit at, the a forum meeting, about, you know, things that are happening in the field, but what I'm really interested in is sort of hearing about what your journey is, how you got to the the practice that you have at, peak human.

And you know what? Where your passion lies in this field. And and then we'll talk a little bit about your approach to aging and your experience. You apply in clinical, the clinical application of your biology.

Sure, sure. Yeah. So I'm, physician, family physician, trained, in Canada and have been practicing for about 25 years now. You know, I've always been interested in, in, things that are, I guess, nontraditional, in medicine and, you know, have been going for meetings for at least 15 years.

But, about four years ago, I ran, bio, we called a biotech summit in Toronto, was in person, and brought a lot of speakers in to talk about things that are just a little bit on the outside of, of medicine, including hyperbaric and psychedelics and, you know, functional medicines experts and so on, so forth.

And that really led me to, to, to start to peak human, and, and then the journey has now has, has, yes, has really moved into the longevity space because of, really I, you know, I myself am just over 50 now.

So I think you start to do the things that really matter to you and for your own health. And, and then I started doing that and then I realized, oh my God, there's so many people like me who this is a really important thing to deal with, which is aging.

So, and that's have now focus my practice on that's what people really pick humans about. Obviously you don't look anything like 50, so, it's pretty, you know, you're doing something absolutely correctly.

Besides choosing your parents? Well, of course, as I say, but yeah, that is a story that you do hear, that once you start hearing about all this information is out there and you see, you know, you and I both probably had really pretty good health for a long time.

And then I've been in the field like 25 years. But you just start at some point, you start saying, I'm starting to feel a little bit less well than I did, even though I don't have any diseases.

Not sick of any, I mean, chronic medical conditions, you know, what is there that you can do? And there's there's just a whole lot that, that you can do and tell me about the kinds of things you do in your practice, what your assessment is, what your philosophy is around it.

It's all encompassing, certainly almost like the World Health Initiative, World Health Organization. And, you know, true health is a state, not just an absence of disease, but of spiritual, mental, and physical well-being.

Tell me more about that. Yeah. You know, I mean, family medicine has that route. Anyways, we're we're trying to look at a person in a holistic manner, but the problem is that we only get like five minutes, you know, in, in the government, the paid system here in, in, in Canada.

But, so obviously we look at the patient, we spend some time to understand, you know, what their goals are. What what are the current issues, what's happening in their, in their lives, some bio psychosocial model.

But I think what I like about what we're trying to do with Peak Human is that, you know, we're trying to add some we call it like science behind it, some data and that that's everything we try to do, you know, pretty content, comprehensive blood work, you know, beyond the usual, things that, you know, the doctor might order and, and I started ordering the epigenetic age testing from two diagnoses that really began some of the thinking about, okay, what what could we do beyond that?

Holistic Longevity Assessment and Biomarkers

I do, I do like an age on everyone now, and, I've been doing, these days, something called the glycol check, which is like, microvascular score, looking at the tongue, and then, and then we're now doing also some, brain EEG recordings through the wavy.

So it's still early and understanding, you know, how do these things look longitudinally. But that initial assessment, we're trying to make it as comprehensive possible.

Even as someone who looks looking healthy, we're trying to see if we can have something some true biomarkers that we can then follow that are really preclinical.

You know, before any actual disease happens, you know, we can tell people, hey, this is this is what's happening from the effects of the lifestyle, lifestyle interventions or other interventions we're prescribing, because that's always been the limitation I felt with this type of medicine is that we could tell somebody something, but I could never prove to them that I was actually making a difference.

And so that is still like the Holy grail for me to say, how can I how can you prove that, you know, let's say someone's doing, you know, you know, an exosome infusion or, you know, you know, taking rapamycin or whatever.

How is that really impacting their system? So, so that's that's what that's that's right. Now we're doing it human. Yeah. Yeah. I mean, you know, it's kind of funny you use the term preclinical, which we all just kind of rolls off our tongues because it's the natural way of thinking about things. But, you know, it's it really is shows that, you know, medicine is focused on disease and you don't even call it clinical until you actually have a disease, whereas, you know, there but these biomarkers that you're talking about, there are, you know, well documented changes year after year of a loss of 1 to 2%, half a percent.

And so, that is you know, adds up over the decades even before you're diagnosed with anything. And that's also been shown that in most things, and particularly in cardiovascular disease, that looking at and making changes at that point is when you can really do something.

Once the stress test is positive, the horse is out of the barn, you know, you're going to be having an angioplasty or you're going to be having a bypass surgery at some point.

You know, or something else is going to kill you. So I think, that for me has been sort of the Holy Grail has been how do you measure the effect of these things at the quote unquote, pre-clinical level?

To tell patients, you know, who come in, you know, my patients, I'm sure, like your patients are like, what about

this? What about this? Should I try this?

A friend of mine is taking this, I'm like, well, how do you know it's working? And how do you know what's the right dose in you? And and in other things like high blood pressure, which is sort of a pre-clinical thing really, if you think about it, it's asymptomatic.

There's no disease there until it gets really high. You just want to make sure that, that, that what you're treating with is actually working. And that's what these other markers are about, which I think is really kind of interesting.

So, so you're, in your practice, then I guess you're thinking about health more than disease. You're thinking about aging as a prodrome to disease. Tell me a little bit about your thoughts about that.

We were talking about that before we started recording. Yeah. I mean, I just just want to jump in that, you know, the other thing that, you know, in the biohacking space, right, where I really started from is this idea that we should give people as much access and understanding of their own data.

So, you know, I'm really a big proponent of wearables, like things like the aura ring. And I've been wearing an ordering now for three years straight, and I've seen the amount of little variation that the you know, I only I could understand that.

And no, you know, no doctor can really understand that little minor like a four point change in my resting heart rate. I can notice that, okay, there's something that's different about today than there is now, but another day.

Sure. I think that type of stuff, you know, I think it's pretty exciting. And I think it's good for medicine to be involved in that rather than it bypassing us.

Like, that's what probably could happen if we don't, you know, be involved in this, this whole field of, of the data that's coming from, from wearables.

Yeah. So how would you sort of situate biohacking in sort of, space of longevity medicine or age management medicine, the doctor and patient relationship.

I mean, you have yeah. I mean, what you're that term biohacking has been out there for a while, and I think, I guess it means sort of more day to day personalized kind of things that work in a sort of and of one.

You're the one that matters. You see what the results are of things. But when you're you, you work with a lot of biohackers. And how do you do that as a physician prescribing and, you know, certain things that you have the ability to prescribe that they, they don't.

Although some hackers are pretty resourceful. That's true. Yeah. I mean, I take it. Yes, it's an end of one, but, I mean, obviously informed by other ends of one, like, we can we can give, you know, potential, you know, suggestions based on on what other hacker or other hackers are doing.

But I call them like enlightened health citizens, like people who are our biohackers are not really, you know, in effect, we should all probably become biohackers in effect, because I think it's the ideas that we want to put there is everyone's has their own responsibility and, and, and should have their own their own, we call it, autonomy to like undertake to get that data and then make decisions on it because they're probably the best at it comparatively, than someone else, but they're certainly the ones that care the most about it.

Although, you know, we care about our patients. But yeah, you're. Yeah, I mean, I agree with you. I think at some point, having not even at some point having access to that kind of data is important.

I think having and that's part of what I've been doing with, with my software and software, trying to give patients as much access to their longitudinal data over time to see how interventions are affecting them.

We have not yet added wearable data, but we will at some point, because we have sort of longer term tests like, you know, keep at one see, and telomere length and senescence, etc., markers that they wouldn't necessarily get from a wearable.

But, you know, things like blood pressure, we know and certainly central arterial pressure that's going to be most important, to learn on a day to day average over time kind of thing, not just, you know, once every quarter when they come into the office.

And that's why I, I actually one of the technologies I use in my office at four or, you know, it's called the, cardiac, arterial stiffness device is going to be a wearable fairly soon.

So you can see, you know, today what your central arterial pressure is. Maybe you should take it a little easier if it's a little high. Maybe you worked out too hard yesterday akin to looking at your heart rate variability kind of thing.

So what do you what do you think about I saw this at the at the conference about the device, which is looking just a pulse there. Through pulse, they can determine your last the, you know, arterial elasticity.

And is that you think that. Yeah, that's, but devices of that type have been around for a while. It's called digital plus demography, which, is a little light that looks at your capillary pulse and gives, something akin to the wave form, which is a pulse wave analysis.

And it looks like an EKG of the, of the blood pressure cycle. It can give an approximation of that, to the one we get with, with the, the bigger, fancier machine here.

And it's, it's, it's legitimate. It's, it's helpful. You can get an idea about what your sort of pulse tracing looks like relative to, to, someone else your age.

Only problem with those is that, sort of your. The temperature in the room is a little bit more important because the peripheral circulation can be tamped down a little bit, but, they're, they're pretty good devices.

And certainly for biohacking purposes. They're, they're going to be pretty good because you can just put them on whenever you want to. But like I said, the the same technology that's being used in, in my office with the signal core is going to be, available fairly soon.

It's already available as an ambulatory blood pressure instrument. Ambulatory central arterial pressure to a doctor's office, but potentially very soon as a, as a wearable for the biohacking community, community and anybody else that wants to use it.

And we can, you know, suggest to our patients, use it and, and then upload it to something, not necessarily the traditional VR, but but the more data I think you're right.

More data, the better. Yeah. Thanks for thanks for helping with that. I wasn't sure if that's a worthwhile, instrument to add to my. Yeah, I think so. It depends on which one.

But also, and I'm not familiar with the latest ones, and it's also considered less expensive than instrument that I have, which is 6 to $7000 now. I mean, I used to be 20,000 when they were, you know, so primarily pharmaceutical companies.

So they've come down quite a bit in price. Yeah. So, so that's you're an initially you get all that work on your patients and then you do work, in, in looking at their aging process, you but what do you look at. Yeah, yeah.

So generally, so as you know, these these results come back, you know, the, let's say that, you know, I have ordered telomeres, I haven't I forgot to mention that I do I do older telomere testing, but not as much as I used to.

I mean, to be completely honest and a bit the concern of that, that was to, I was ordering from a company called life

and in Spain, I was finding that the, there's a huge variability.

It didn't seem like, yet the results kind of match, but, I did do some sequential, some sequential blood tests as well, on people. But, from what I was reading, I was hearing that the precision wasn't so good with this type of testing.

And so I was I've kind of held back from doing that type of testing. Yeah. So I think that, that what what I do hear that. And there are I read that in papers, and I think, that there is still work to be done on it, general consensus.

I mean, there are different technologies that are used for, for like measurement, there's flow fish, there's PCR, there's high throughput quantitative flow fish.

And so, I think what I would say is that if you're using a company, you stick with one company, and follow them longitudinally. The other thing I would say is that, you know, the coefficient of variation on a, telomere length measurement isn't that far off of, both within within the assay, if you use the same, you know, same sample I measure a few times, it ranges from 2 to 3% for flow fish.

Up to 5 to 10% for PCR. That's not the the 2 to 3 is not that far off for cholesterol measurements or any routine chemistry, CBCs, etc.. There is biological variability though, which is pretty important to keep in mind.

And people sometimes I've talked to other clinicians that have lost faith in them because they're like, well, this is one year or six months have passed and there's now a six year difference in their telomere length. And what's this all about?

Well, you know, the biological variability is quite significant. So you have to get a number of telomere length before you get an idea about what the trend is.

You know, particularly so if you have, say, six kilo bases, about average for a 60 year old, but if you have 2 to 3, even 5% variability, you can have a few years of variation.

Just. Yeah, test a test now. And I see that in my patient. I've been measuring telomere length for 14 years. And patients. And you know, I'll see them at 6.56.46.7, 6.3.

And I'll see one that's at 5.8. And that's weird. Is that because they've really lost you in real life? I don't think so. I think it's because the percentage of senescent cells in their circulation on that day was a lot higher.

And senescent cells have much shorter telomeres. So the average is coming down. You have to remember that when you're measuring telomere length in a sample or white blood cell sample, it's a lot of different types of white blood cells, from naive T cells to B cells to natural killer cells, you know, to depending on if you're using like repeat diagnostics, you'll also get gray oxides, which are neutrophils which their T likes don't get shorter once they're in the circulation.

So you have to keep in mind you know what the technology is that you're using to measure it.

Wearables, Biohacking, and Patient Data

All that being said, I would say that the that measuring them over time, you can get the information you need, just have to interpret correctly. There's something called pseudo telomere lengthening and shortening.

And that kind of is based on the cell population, not on actual change in dealer, like the composition of cells that you're getting that sample on that day.

So just keep that in mind when you're measuring them. I encourage you to start measuring again because I think yeah, you're they're important to, to know, so are you saying that by infections like someone Kevin.

Infection that could change. Like what? What causes of biological variability are you saying? Stress? Exactly. I

mean, so there's if you have exhaustive or even, you know, fairly moderate exercise the day of the test, it's one of the benefits of exercise.

It mobilizes senescent cells from the tissues into the vasculature to be removed. And but that's going to change your, your composition so that there's going to be a heavier weight towards shorter telomeres.

And your median telomere length, which is what's getting reported out, is shorter. And even if you're using like life length and they're looking at median telomere length and 20th percentile bottom 20th percentile, well, bottom 20th percentile might go up because there's more cells that have shorter telomeres.

And I mean I'll go down sorry. The length of it goes down. And that's that's not actual change in telomere length. So I if a patient has exercise that I always encourage, not just tell them not to exercise the morning of the test, before they come in to get the blood drawn, or if they have the alcohol intake or really bad night's sleep, or if they're jetlag, or certainly if they have any inflammatory process going on, even a mild cold, or an infection of any kind, post-surgical.

We don't measure those tests under those circumstances. You're not going to get the information that's telling you about the sort of more stable state of the patient normally is in.

So it's important to have all those caveats when you're measuring these things. And that's also true for any time you're looking at senescent cell measurements of the various kinds that are available.

And what's your thoughts on that? I understand that there's an, a methylation test that looks at can basically also measure telomere length. Epigenetic.

Yeah. Methylation test that seems to be apparently more accurate than actual. Well, so it's been hard to find accurate. I mean, that test is used, by the same company, True Diagnostics.

It's a it's a, it's a prediction of telomere length, based on DNA methylation patterns that, are more accurate for certain other outcomes of telomeres.

Not actual telomere length of the actual telomere length is. Right. And I get, you know, two diagnostics tests often in tandem with, and they can be accurate, but they can be I mean, I have to assume that the actual test itself is the gold standard, right?

It's like these, genome wide association studies where, you know, they say that they predict better than the actual marker that you're looking at. I mean, that depends on what you mean by better and more accurately, if you're actually looking at want to see what the actual length of your telomeres are, you measure till I mean, you can get a good approximation.

And it's it's very interesting. And we're working with the company to hopefully improve their ability to do that so that, that, that, you know, they can, really come up with something that perhaps could replace the older lines.

But I don't see that happy. It's not the same as predicting chronological age. So, I think it's interesting information to have. They're looking at it to also to predict senescent cells.

So I think that that there's, there's definitely going to be some progress made in that area. But but for now, I would I would actually, you know, measure the telomere.

It's, it's it's the actual data that you want, if you can use some of the cheaper or another way where you're getting other information, then at the same time and you have limited budget, then sure, that may be a good way to screen for somebody who's extremely short telomerase, although I don't know how accurate it performs under those circumstances.

So you've measured some telomere length. If you ever come across anybody with really long telomeres or really

short telomeres, you know, to be honest, no.

Not really. No, it's it's been pretty much like, you know, right in the middle there. But, but, you know, I was I was curious about, you know, I have been, I've been using, 1065, I used, you know, probably for eight years at.

Oh, really? Wow. Yeah. I, I stopped for about, four years or so, but, I think before that, the randomized trial came out that showed that there were actually lengthening of telomeres.

Because at that time, you know, before that, I was kind of wondering, you know, I was kind of waiting for the research to kind of be catch up for that.

But but I, I'm using it now and then recommending to patients, but I haven't been able to follow up test to see if there's change yet on my patients. Well, you're right there in Canada with repeat diagnostics just to left or right, depending which way you're facing.

Exactly. Over in Vancouver. Yeah. And they're testing is, I think, really quite accurate. And and useful. I know I've looked at probably this point over 20 liver length tests, with, with them, we have data that we collect and, it's, I'm, I'm always amazed at how well the data that we collect on our patients matches the published data.

In terms of, you know, lengths and average length and spread and that kind of stuff. So that, you know, clinical research, which is fun when you have time for it.

And I've been trying to do a fair amount of it, is, you know, it's really gratifying to see that, so, so yeah. So perhaps think about it again. And if you use this your age, which I'm going to get you on, I think at some point, you get a $100 discount on it, so pretty much pays for the software if you do one telomere length measurement for, per, you know, per month, and you're good.

Because, because, we, we get a pretty good discount from them. So peak human. What what do you how would you define that? Well, what do you what are you looking to achieve with that?

And so you're, you're looking at a holistic because you're, you're going from family practice to what you think family practice ought to be. Just like I went from internal medicine thinking what internal medicine ought to be.

And so what are the kinds of things that, you think our listeners might want to hear about that you do that perhaps other practices don't do. Yeah. Let me, let me let me, think about that.

You know, I, I what what we tried to do is, is, again, get patients understanding of, what their labs are like.

Telomere Testing and Biological Variability

So they actually, you know, take ownership of that, and then, you know, you know, I am I am, you know, recommending now certain patients take, you know, metformin, rapamycin.

We are putting patients, some protocols in their, so really we're trying to keep it evidence have is evidence based, science based. But, you know, these are for patients who are relatively still feeling pretty healthy.

So we want to basically prevent, onset of a disease as much as long as possible. We can we can do that. Again, it's a relatively new practice. You know, it's only been about two years since we've had most of our patients.

Come on. It's kind of early days to see. But, I think just just the fact that patients have an option, I think that's what's the really excited about.

There isn't too many too much here in Canada that really has this type of, option for them. Yeah. It's great that you're offering that, more so they can have a more comprehensive look.

I mean, I think patients are taking, you know, with the advent of PubMed being available and, and all this advertising on social media of, of information in the longevity field, which is just exploding, really just the the amount of information that's available.

Some of it is clinically actionable in terms of taking, you know, molecules, whether it be, you know, 65 or, you know, glutamate or rudicil and, or something like that, you know, patients like, say, I try this, I try this, I try this, but they need to be guided.

Unless they're like, really experienced biohackers like, in which case they're probably on Instagram giving out their information to people. You know, it's, the patients need to be guided as to what is something that actually is working in them.

And metformin is a great example. I mean, I'm all for the team trial. I was talking to, I think one of our other speakers about, you know, I think that metformin, you know, has great data in a certain patient population, which is probably not yours or my patient population.

Most of my patients are exercising at fairly close to their ideal body weight, don't have a lot of insulin resistance, and there isn't any data to show that metformin is going to necessarily have the same reduction in mortality and onset of, chronic diseases that all the data sets that are talked about.

So that's the average person in the in developed Western society. And there is good data showing that metformin can cause a, decrease in your response to aerobic and resistance exercise.

So if your patients are, you know, have a hemoglobin A1 C of 5.0, you know, if you use over there, they use the IFC, IFC or IFC. But, you know, that's really low and really good.

And, you know, they're exercising. I mean, I'm not I'm not on metformin for that reason. Because it's it's I don't think it's necessarily gonna work, but it's possible it could happen.

So off target effects, I'll use something like berberine instead, or maybe even glucose, which is glucose dox, which is a, a Google lipid, MCT that has some effect on, on, Ampk, regulating it.

But that's, I think the end of one, the biohacking part of it. But, you know, I mean, I don't see what do you think about this? We think that this is, you know, it depends on the population and it ultimately depends on the effect.

And that's what you're right. You want to measure it baseline or you want to measure later. Is it actually working in them. That's another reason why I, I talk about it with to 65 or any telomerase activator.

You know, because they're usually not very inexpensive. There may be a dose 100 units in, in our study, in senescent cells, 100 units had an effect. But, but, that may work in one patient, but because of the differences in bioavailability, it might you might need 250.

You might need 500. You might need a thousand units. You don't want to waste your money. If you 100 is working, you don't want to waste your money. If it's not working, you want to go to 250 or 500, whatever you need to go to.

And with delimiters, while people do talk about anecdotal stuff, I know if any of your patients have mentioned that, you know, perhaps they feel a bit more energy.

Perhaps they feel that their vision looks a little bit better. And I've had Tom Dalan, who is an ophthalmologist, and, you know, he published a study on the effects on macular degeneration of 65.

So there's definitely mechanisms for which vision might improve. But you don't know unless you objectively measure it, that that's the issue. Like, I don't want to say.

Yeah, that people are because people are saying, hey, potentially other people more energy and all that. But it's it's

very hard to say that because again, you have a bias.

And yeah, sure, placebos are put in effect. So you know, it's that's why I think we need we need probably more more long term data. And that's why I think the clinical I mean the, the biomarkers are so important.

If we can see changes on that and then I'm more excited about, those results. So using rapamycin or using you do exercise prescription and diet and exercise.

Yeah. That's what yeah a lot of, a lot of, you know, again, I even mentioned is that I really come from, you know, interest in the impact of diet on health.

It's that quite a bit of reading in the, in the whole space on fasting, which I do recommend to patients. I recommend the fasting mimicking diet as well, the prolonged and then the four cycles myself, in the past year.

So, but yeah, I, I'm a big believer and that that's important for the body that one needs to probably take a break from food and whether that could be also keto potentially, I guess.

And so I try to, to also have a lifestyle kind of component to this as well. And for exercise. Yeah. You know, weight training I think is is critically important.

Some, high intensity interval training, the things I ask my patients to do. But that takes them. That's a much more. But, you know, I think everything is in gradual like when people come in, I kind of have to focus on where they're at.

And for them, the first person that gets just maybe just cutting out soft drinks, that might be just. Yeah, that might be just quitting smoking for. Yes.

You know, that's like, I'm here for change management, but I'm still smoking. Yeah. That's problematic. Yeah. So that's a really low hanging fruit that's actually it's on the ground.

So yeah. Yes. People don't come in saying, I want to like, I want to like, you know, I'm here because they're all super already, knowledgeable. Most people are just like, okay, I want to just feel better and and are there, you know, like people like I said, I'm 50 and they're looking to, like, noticing that something's changing.

And, you know, maybe they need hormones. I mean, that's really how this practice started is that, you know, people want to come in for hormone replacement men, primarily, growth hormone, you know, replacement and so on, so forth.

And, and it's kind of now moving them along from that to what I think is true, you know, you know, interventions to kind of, prolong the aging process itself.

Yeah. I mean, that's actually the, the, Genesis or not the Genesis. I guess the genesis of my practice was really, you know, my starting and, sorry, my parents on growth hormone and, and other sex hormones.

And then, you know, back opening the practice up once the Women's Health initiative hit, it was ideal and have done and continue to do a lot of hormone optimization.

But in the last 5 to 10 years, there's been so much on more direct aging process stuff, and molecules involved in that. But I've been really heartened to see that all of this stuff that we were doing with hormones prior to the Women's Health Initiative, calling it an anti-aging medication, is being borne out with these newer molecular diagnostics.

For instance, I had Gordon Nelson, who is the founder of the glycan age, creator of it, and, you know, menopause worsens your glycan. It makes your, your, immunoglobulins, IgG more inflammatory, makes them, you know, raises your like an agent.

I see in my practice I put someone on HRT and they're like need to come back down ten years or 15 years. So those are important. Molecules are still really important.

Part of the the arsenal that we have, you know premenopausal you don't necessarily need it. But and testosterone does the same thing in men. And then same with DNA methylation and epigenetic age.

You see that menopause accelerates that HRT slows that down. So, I think it's just been really, really interesting to see that these sort of more gero science markers have validated this stuff.

And even growth hormone, which a lot of, you know, vulture Longo, you talk about, you know, with prolonged and intermittent fasting or sorry, fasting, mimicking diet and fasting, you know, IGF one is is the bad guy.

It's the weight. That's true, you know, that we don't have and that the Ecuadorian dwarfs don't have, or, and, and, you know, you're trying to lower IGF one with the trim trial showed that there was an epigenetic aging reversal with metformin growth hormone and DHEA.

So how do you how do you, how do you make sense of that? That's there's still more work to be done as far as I'm concerned. Yeah. So you don't have a clear opinion on this?

I would love to hear your opinions. You on the growth hormone piece. You may have much more experience. Like what do you think on that and longevity, you know, is it, is it just a fine balance?

Well, yeah. I mean, I, I think, look, you know, I do have certainly many years of experience, treating patients, 25, 24 years or something like that. And I've been on a growth hormone a long time.

I think that that the problem is, is that lower animal models, are not good models for looking at the effects of growth hormone. And they've also looked at them in the wrong way.

They looked at them in transgenic models. We know that acromegaly is not good for you. We know that. Right? You got IGF 1 to 5000. That's not a good thing.

You know, in some bodybuilders on stage probably do have an idea of what, a 5000. But that's not good for their longevity. Their organs are increasing in size or heart's increasing in size.

But we also know that IGF one, you know, in observational studies, is associated with higher cognitive performance in older individuals. So I think the story is, is much more complex.

The effect of IGF one on immune system functioning. You know, there have been studies done looking at giving middle aged mice a rats growth hormone, and they actually do find that you actually do a little bit better.

That's what we're trying to do in humans. We're not trying to make transgene in humans. They just make too much growth. And we know what that is. That's gigantism or acromegaly.

So I just think that that's not the right way to think about it. And that, you know, I know as a clinician that if someone's IGF one is low below 100 and I bring it up to 150 to 200, which is still in the physiological range, they feel a lot better.

Their cognitive they feel better. They their mood is better. Your energy's better. Their body composition certainly improves, their hair grows faster, your nails grow faster.

So performance wise and quality of life wise and health span wise, I think that you see benefits. And that's why, you know, one of the reasons growth hormone is still FDA approved for lifelong therapy in growth hormone deficient adults, who are not any different in circulating growth hormone levels from somebody with severe IGF one deficiency, from aging or from obesity, or from stress or from lack of sleep.

So, I think this idea, it's akin to the whole idea of just wiping out estrogen and, you know, women because that's going to cause breast cancer, you know?

Yes, I gf one is a mitogen signal. It does stimulate cells to divide. But now they have large databases following things. If there's an increased risk that's slight in colon cancer perhaps.

But that's, you know, very high levels. Of growth hormone and acromegaly. I think it's a balance.

Peak Human Philosophy and Preventive Interventions

You know, you want to walk a balance and you see how the patient responds to it. And there was also another study that was interesting. We looked at if you gave growth hormone, there was an increase, risk of cancers in these rodents.

But if you gave them a great supplement pack that reduced their oxidative stress, that was reduced. And so I always give my patients, you know, full supplement regimen to help, you know, reduce their oxidative stress.

So, I mean, I think that, that we have to look a little bit more in a nuanced way at these things before we decide that, you know, one molecule is really good or one molecule is really bad, even telomeres.

I mean, you know, there's talk about really long kilometers being problematic, and running in families that increased risk of gliomas and some other cancers. Non non squamous cell or adenocarcinoma of the lung.

But those are very unusual circumstances. And for most people I think keeping your own telomere length where it is when you're young or keep you from getting shorter is is much, much better for you.

So it's it's all about the balance, keeping the balance. Did you think the I mean, I'm normally ask, you know, my patients to do it for three months, take a 1 or 2 months off.

Like, do you think this may have some benefit because, you know, naturally, not everything is always growing. Not everything is always dying. I mean, the fact that maybe the body has to go through some type of cycles, it's normal.

You know, that the whole cyclical nature of things is really interesting. And I've looked at that. I've done that myself. It's gone off a bit for periods of time.

It's just hard to know because, you know, just as in, you know, the growth hormone deficient adults, there's not a period of time and they're, you know, sort of normal, so that those studies need to be done.

May be interesting if they were done. I mean, you know, molecules have different effects with different dosing intervals. That's clearly true of rapamycin, which, you know, if you gave it and the, you know, cancer or immunosuppressant therapy, doses every day, you know, a couple of milligrams, five milligrams or whatever, is much different than five milligrams once a week for eight weeks.

So so it's possible, I don't know the answer to that, and I don't think anybody does, but I think that we shouldn't dismiss these molecules that are in pretty reasonable abundance at our peak of health and, and say that, you know, we're better off having very low levels of them. The age old.

And I don't even think that if you, you know, you look at a centenarian, say, this is the hormone omega that you want to have in order to live a longer life, that of a centenarian, that doesn't necessarily make sense to me either, because it's centenarians while they're still alive, and they may be disease free or not.

You know, performing the way of 25 to 30 5 or 45 year old is performing. And and that's ultimately the goal is to maintain humans at a level of performance that, you know, they are at their peak performance for as long as possible.

But that's that's my take on it at this point. I think that, you know, practices like yours and like mine, that's why I created visual aids is to, to help have data on patients that are getting different regimens and look at it, and see, you know, how how people are doing it and using our vast computing power that we have these days to to really analyze what is happening and don't 50, 100, 500 is page management longevity practices around the country and around the globe.

And then we start to see patterns. You know, maybe in certain individuals you don't want to use one therapy, but in other individuals that same therapy has a beneficial effect.

It's one thing that I've learned, and, you know, I don't if you had this feeling maybe for a little while, but I had it for a little while here after the Women's Health Initiative came out and all the patients were coming to my practice, and we were making them feel better with biochemical hormone replacement therapy.

And I said, like, well, I know what I'm doing with this now. And I guess, you know, I'm, I'm, I'm an expert at it. I don't really what else is there to learn?

Well, you know, now I'm just like, I can't catch up. I can't keep up with all the information that's coming out in aging. You know, biology itself. But certainly the biology of aging is super complex.

We know some things. I think we're doing some things right. You know, some we don't know some things. A lot of things. And I think people are often a little bit too dogmatic about how they think about these things.

I'd love to ask you a question which, you know, maybe, hopefully you haven't talked to with anybody else, in the, in the summit. So what's your feeling about CMV infection as you think about how much contribution does it cause to aging?

Sure. Yes. Well, that's a that's a good question. We have talked about that somewhat. But, you know, I think there's pretty good data on, on it. You know, that it was it depends.

So there's been a couple of great studies that looked at CMV in older adults, the Cotto trial and the Nonna trial in Sweden. And in healthy ish, you know, like not carrying multiple morbidities and free living 80 year olds, the CMV positive ones, if it's they've had it long enough and has had enough of an effect on them to cause an inversion in their CD4 to CD8 ratio, which is normally around 1.5 to 2.5.

The helper cells to the suppressor cells if it's under one, so that there's predominance of suppressor cells, that predominance is there because of the, accumulation of senescent cells that are focused on CMV and also not dividing anymore.

There's senescent, and they're secreting a lot of inflammatory markers. They have a much higher two, four and six year mortality rate, like 50% higher than those who are CMP negative and have a or not in what's called the immune risk phenotype.

But that's at the later ends of life. Right. They then went on to look at 60 year olds. The Hexa trial, and they found that while there are some people who are in that immune phenotype, immune risk phenotype with the inverted ratio, they don't have the same mortality risk.

But there are many other cardiovascular studies, for a colleague and, that's worked on to 65, I hope Kim Spears applies that in, in the UK, is just finishing up a trial looking at 65 in, survivors of, acute coronary syndrome, either in MI or, you know, sort of, angina requiring angioplasty.

They, they it was actually an acute MI that you had to get into it. And an event was considered one of those things, either secondary MI or, angina, they're looking at to 65 to see whether or not it reduces inflammation.

And potentially, second events. So, you know, I think CMB is a very important factor in people. It's, not a benign virus like we're taught to believe.

It just has a very asymptomatic, a very long asymptomatic prodrome before it starts to cause problems. And that, you know, in some people is in their 60s with a cardiovascular disease or potentially, you know, other diseases that haven't been found out yet because of the increase in inflammation or it's in their 70s or it's in their 80s where they just succumb from overwhelming infection, and die, because of their inability to fight it off, because of their senescent cells.

So I think that, that it's incredibly important and there are whole organizations that look at CMB and hold conferences on them. And, I think if we could have a vaccination for CMV, we'd probably save more lives.

And if we have a Covid system, then having a Covid vaccination, potentially, more people mean many more people are CMB positive. Well, at this point, right now, it's probably going to be everybody to see, Covid positive soon.

But so yeah, I mean, I think that I check in all my patients. I want to know because, you know, I think that if they're in the immune risk phenotype, you need to talk to them about, you know, I think you want to look at you're telemetry length and you want to think about things that can reduce the senescent cells to reduce that denominator, to improve their, their chances of, of having a longer, healthier life.

So if you're even if you don't do theater length testing, I recommend at least screening with the routinely available CD4 to CD8 ratio. And if it's inverted, then do further testing.

You could do it that way, right? I mean, I am putting people on tier 65 to have a see if they have seen the infection in the past, but if they're a bit of a, you know, older, older gentleman.

Yeah. I mean, I've only caught one person in all my years doing this actually converting at a pre. And they were seeing me negative. I got their senescent cells.

I had everything on them. They're asymptomatic. The next time, six months later, they came in and they were seen to be positive. And their daily lives were shorter because senescent cells have gone way up.

It's all been described in the literature that it causes a remodeling of your immune system. It's can be, you know, up to 10 to 20 years of of aging, of the immune system.

And, you know, we you we caught it, right? It was a snapshot and then longitudinally follow them. I haven't, you left the practice. I haven't seen them.

And I'd be curious to know what what's, What? It was a young guy who was in his late 30s. Oh. So, pretty, pretty interesting to to see that that change over in what it does and what viruses can do in general to, to you.

And and that's where, again, I think dealers are important because it's the reserve that you have to fight off chronic viral infections, reactivation of them.

And the herpes viruses are really in that family of viruses, CMV being the worst. But all the herpes viruses are the gift that keeps on giving. And I say that, you know, facetiously because it's not a gift.

What what can we look forward to, as in, in, other, compounds that, linked and dealing. There's do we have any anything else that right now, you know, the size and hyperbaric study that came out, was anything else too, right?

Yeah. I mean, the the hyperbaric oxygen therapy study was very interesting. Quite a significant deal here. Like the increase in decrease in senescent cells was a little worried about the measurement technique.

And I want to see that study, reproduced. But I did talk to, another, Jason Saunders who runs, another oxygen, hyperbaric oxygen therapy, company. And they're looking to kind of do that study again.

And, and I'll be looking at hyperbaric oxygen therapy for myself. So, so there are sort of home units that you can have, too. I think. So it's kind of interesting that, you know, you know, that the, the, the mobile ones.

But in terms of, in terms of actual molecules, I mean, there are some molecules that Bill Andrews, has talked about that are polymerase activators, but there are studies in humans to show that they're absorbed, they're in vitro select on those activators that they're absorbed, get into your blood and then turn on telomerase and make changes in your TLR length or your senescent cells.

So right now it's still so that, you know, people talk about the Italian, being potentially a telomerase activator. Right. And that's that's possible.

Hormones, Growth Hormone, and Aging Debate

But you have to you have to do the study. So I, I, you know, all the, all the lifestyle diet stuff, I think is important and the kind of stuff that you're doing in your practice.

So I think is important, for, for that, I'll doing some reading that the newborn telomere length is by far the biggest predictor. What your lifetime, you know, until.

Yeah. No question about that. I mean, the, tumor length is a 70% heritable. Yeah. Which, again, goes back to the whole joke. You choose your parents wisely.

Because, you know, and that difference between eight kilo bases and 12 kilo bases, at birth and it's, it's pretty much spread across that, is, is a little more than you're expected to lose over your whole lifespan.

So if you starting down at eight, you better not lose it at quite the same rate. And if you're at 12 now you do lose to older like faster the longer it is.

That's well well documented as well. So there's a steeper loss. But, you want to it is more you want to be up there in the higher, higher range. That that is true.

But apart from your parents, telomere length, is there anything else? And, intrauterine, that could affect telomere length? We just a, Yeah. I mean, I think that there's, there's stress that can do that.

I can't tell you right off the top of my head. And listen to Apple and Elizabeth Blackburn, Elissa Apple and Liz Blackburn, we talked about that in their book.

But there are I'm sure there are so many factors, in uterine that affect telomere length and metabolic health and so many other things, that, I'm sure that there's important, important stuff there.

Maybe I'll try this experiment with you for the first time. I haven't done any visuals here. I'm going to share my screen and put up this, okay. Put up this interesting plot that we have, for people to look at.

So let me, do this and come over here. Put up. There it is. I see it, you know, here's my screen. That's your questions? Yes. My question is, what do you want to see?

There we go. Yeah. Sigmoid fits. So this is. Oh, wow. Look at that. The data from, repeat diagnostics. The paper done by, Peter Lamb stars and Geraldine Zubair, and we took their data set, which was available, you know, publicly.

And I had a data scientist analyze it, to look at what was the best fit. You know, you talk about telomere length as being linear decline and say it's not such a great biomarker that way, but it's not actually a linear decline.

And the company, when they report out their type of assay, the flow fish assay does show that there is a a steep loss from birth to young adulthood, which makes sense, right?

Because you're growing from a fetus. So the newborn baby to a much larger a lot of cell division has to take place. Telomerase is not active, nearly as much because it's turned down significantly at birth.

And there's a spread you're talking about, about eight kilo bases to up to 12 kilo bases here down to. Yeah, seven, 7.88 to, now, you know, if you have a telomere ofthe where you have 50% telomerase activity that you've inherited from your parent, you're going to be down here in the 4 or 5 range.

And that's those are known they're called the telomere biology disorders. But but this, my data scientist was able to do is this red line is the instantaneous rate of change, of the curve.

And this is a specific curve that we fit to this, cubic curve. And so you can put in the value, what's average for a 44 year old, say a 52 year old and, you know, come up there.

I'm sorry. You have to go to linear like a kilometer length here at eight. The average is 6.1 at 52. And you see this spread. And so, you know, if you start out up here and you're going like this, you're going to be much better off if you start out here and even have a flatter, you know, because you have a lot of good lifestyle stuff.

So, and that's why, you know, this spread is why telomere length is a single biomarker. They say, isn't such a great biomarker. But following somebody longitudinally is, I think, very valuable.

And we're going to have this same plot available on in the software fairly soon so people can look at it. And, you know, we give you a telomerase already, which is, you know, what your age is.

But to have people know what, and so you can see the rate of loss here is around, the slope is quite significant. You know, it's, at age, say 14, you're losing a 10th of a kilo base.

Right. But at age 20, 20 something. Let's say age 30, they're all losing half half that.053. And even at oh, way over here you lose. Sorry. Let we come over here to this age.

You know, your slope is different there. So, slope is 0.035 less than 0.05. So it does change over time. But yeah, I'm a pretty good idea about what expected loss for that time.

So if you have a 60 year old, you're going to say, well, let's say whatever this is right here at 62, you're expecting 0.035, whereas at 25 you're expecting closer to, you know, 0.5.05.06.

But that begs the question that do you think that these telomerase activities might be more effective when you're younger, when the well is happening?

I don't know if they're necessarily more effective. I think that they are. You know, that begs the question of when do you start taking a telomerase activator?

You know, the official company position is sort of age 40. But, you know, theoretically, you can start at 25, to maintain your telomere length. You know, I'm not doing that yet unless they have unless they have a 25 year old who has a telomere length of a 50 year old, which you do see, but, you know, I think you want to keep your telomere length, pretty much the same as it is around age 25 or, you know, potentially even younger, if you can.

That's that's the way I look at it. And there's another reason for that, because of the whole concept of, telomere position effect, telomeres are not just about allowing cells to divide.

You know, besides their role in mitochondrial biogenesis and having healthy telomeres allows you to have more mitochondria, mitochondrial biogenesis and healthier mitochondria.

Telomeres also regulate the genome by suppressing expression of sub telomere genes. That's a that's called sort of near the telomere, some telomere, epigenetics.

But they also have telomeres over long distance. Gene, regulation up to ten, ten megabase pairs away because of the way in which the telomeres fold around on themselves.

CMV, Senescence, and Future Longevity Tools

So, and when they get a little shorter, just, you know, between, let's say, 25 and 35, there's a change in gene expression that maybe some of the gene expression changes.

We're picking up with a test like true age. The two diagnostics test. And so if you keep it the same length and you don't get that gene, potentially you don't get the gene expression change.

So these are all open questions still, of course. But if you look at it logically, I think, not having any children of length loss is the best. It's the best thing to do.

Have you what is your like the longest that you've been tracking. To 65 using patient. And have you seen, lengthening of their telomeres. I have seen lengthening of telomeres, in a patient.

And the loss of traction is in the original patients, the original cohort, and that, for 14 years now, and myself, 14 years. And I haven't lost the other leg for years now.

Have I gotten older? Yes. See, pictures of me and I'm younger. I'm definitely older. Says other aspects of the aging process, but I feel like 65 is certainly done, that I have many patients that have had no loss of telomere length, and I have had patients who've had significant increase of feeling like up to a killer base over five years.

And then lots of patients pulled out of the immune risk phenotype because of 65 losing my lights. Now. So, it's sort of like when the theater dims, you know, the lights come on in the theater and they say, the movie's over.

Okay. Know, we've been talking for quite a while now, so thank you. Thank you. Do you have, any final closing thoughts or any. No. No other questions we can pick up later?

Huge enjoyment. I hope the viewers, got something from this. And and, you know, I've taken back that to go and go get my, telomere testing again, back to for my patients.

So I'm going to start doing that and I'll report back to you at the next summit or some other time we meet. Yeah, that would be fantastic. Let me just, stop the share so I can see if.

Oh, there we go. And, Yeah. So, yeah, I encourage you to do that. And it's great to have other practitioners that have the same view of what we should be doing in medicine.

And, I look forward to seeing you again at one of the meetings or maybe popping up to, you know, why don't you tell the viewers, exactly what your, your, social media and your URL stuff are so they can know what it is.

Yeah, they can, again, my, my website longevity speak human. Okay. If they want to email me, they can, reach out to my email, which is Doctor Gabel at peak human.

Okay. So DRG oh, well, at Peak Maker, and I think I'm on Insta. Just look up my name and you'll you'll find me there. Very good. Well, all right, thank you very much, Sanjay.

It's been a pleasure. And, we'll talk soon.