Telomeres, TA-65, and the Science of Aging: A Deep Dive with Dr. Joe Raffaele Transcript

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Introduction to Telomere Testing

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And I think everybody should get at least one telomere length test because definitely want to know if you're in the bottom 10% versus the 50th for the top 1%.

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Welcome to Doctor Talks, the podcast where every episode leads to a healthier you. Join us as we navigate the world of optimal health, uncovering groundbreaking strategies to conquer chronic disease.

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Get ready to unlock the secrets of lifelong health and vitality. This is Doctor Talks, real talk from real doctors on the issues that matter to you most.

Welcome back to another episode of the Recharge Biomedical podcast. I'm your host, Doctor Edward Park, and today we're very fortunate to be joined by one of the pioneers in the field of anti-aging, Doctor Joe Raphael.

Hey, Joe, how are you doing? I'm doing very well. It nice to be with here. With you. Excellent, excellent. So our journey started while mine was, I want to know why people age.

And I thought, oh, it's got to be this telomere thingy. And I wrote a graphic novel. And the next year, the Sciences Corporation marketed to 65. And Doctor Raphael was key in designing the patent protocol named after the founder of the company.

Origins of the Patent Protocol

So I flew out to New York Central Park and we did some tests, and we assessed that I would start taking to 65. I think I was in the maybe the 17th or 19th person to do so, and Doctor Rafael by then had already had amazing ways to measure how old you were, because he'd been in it for 12 years already, trying to figure out, well, I'm giving people hormones and are they getting better or not?

What are the objective measurements? So we'll we'll be going into that with him. But tell us, Joe, how did you get involved with the two scientist people in the first place?

Well, so interestingly to his credit, Noel Patent, the founder of the company, unlike founders of a lot of other supplement companies, really wanted to sort of figure out and go to some of the experts out there to figure out, you

know, whether his product actually had good clinical benefit.

So he came to my office, you sort of knocked on the door and said, you know, I know that you've been doing longevity medicine. We call it an anti-aging medicine.

Back then, he said, what would it take for you to offer this supplement that there's, you know, good animal data on? And it had been started out as a new drug application.

And I said, look, you know, if you're saying it's going to affect your liver length, which is then going to affect the aging process and have clinical benefit, there are lots of biomarkers of aging to look at to, to measure that benefit.

And so that's what I would want to do. So he said okay, let's run a cohort of people through your practice through the biomarker testing that you do molecular studies telomere length but also arterial stiffness, pulmonary function, skin elasticity, a whole range of things from cellular and molecular to whole body, and to see what effect that has, along with the supplement protocol that you've been giving your patients for a long time, which was a multivitamin mineral kind of packet of things, along with diet and exercise counseling.

But this was a cohort, not a randomized controlled trial. But to see how this cohort did taking to 65 along with this supplement pack that we the ultimate ultimately call the protocol the patent protocol.

I tried to get in to call it a Rafael protocol. Well, in regardless, you know, I remember coming out there very hopeful and in the waiting room without violating HIPAA.

There was the wife of a very famous geneticist, and I guess he's still alive in his late 90s. And, you know, over the years, it's been 17 years, 17 years.

I've been taking this every night. And until last year, unlike a lot of your patients, it was the only thing I took. I didn't take any hormones that anything else.

So I remember a gentleman asked at Rad Pass, how do you know what's working if you're taking 70 things? And for me it was 16 years only on 65. So I know that initially I came to I had fatty liver, high blood pressure.

But at three months and we'll get to that study that they did in Spain at or Yukon at three months. I lost a bunch of weight and it was like a visceral fat extinction.

So I'm wondering, in your practice, have you seen other people who lost weight after use up to 65? Well, we certainly have, but unlike your kind of one experiment for many years, most of my patients are taking other supplements.

We do hormone optimization. We do diet and do exercise. So it is a little hard to tease that out. However, you know, as we'll talk about, I'm sure there are multiple molecular pathways for reasons why you would see some improvement there, particularly improvement in mitochondrial health, metabolic rate, things like that.

That could could be what it was doing. It could make you feel better. You start to move a little bit more. There's lots of different things in there. But I don't know that there's been any trial looking at that specifically except the Fernandez trial you're talking about.

We're looking at metabolic health and the control for for most things. Yeah. So, you know, you use the term and apply. And I remember as a resident, you know, we were trained at the Socratic method.

So we do rounds, we sign off and the attending would pick us apart and say you half. And then of what it was a very derisive term, you know, and but now I go to these biohacker festivals and they lean into that, and they've reappropriated that term and looked at it with great pride, because, of course, to you, your previous data is super important, and you're going to do biased decision making based on your data stream.

So anyone has now been elevated. What I used to say also as a as a technique for debate, would be, well, unencumbered by any data, you know. So they'd either say you're being too specific on this case or they're saying, well, you know, where's your data?

Quote some data. But I think it reminds me now I'm using a lot of I it reminds you of the type one error, right where it's a false positive. You think that something that you did somehow, you know, stepping on a crack in your mom's back row versus like data mining, which is type two error.

Like if you try and make the model fit the instance, you may miss what's really going on with that individual patient. Right? But now I'm so impressed by I's ability to take data and discern.

And I guess in your humanoid way, you've been trying to do the same thing. When I first came on as a patent protocol, doctor and I, I incorporate your physio age.

We had what age?

Biomarkers of Aging and Multi-Omics

Accouterments for measuring your arterial stiffness, pulmonary function, CNS vital signs for cognition and vision were the big ones. And then we had telomere length and immune senescence.

And those are really solid seven markers. But now as I perused your website, there's you're all comers. Like how can you integrate you and Jerry Fortunato.

You have this ability to kind of comb all this data, all these things that people do measure. Do you feel like it's a working model of aging, or do you still look at it as individual organ system?

The lungs, the brain, whatnot? Well, so I think that, you know, the original seven biomarkers were if you put them together after we did what we did, and that was the genesis of the physical health analytics platform.

We took the results from the original study with the patent protocol and analyze them. And we found that with those biomarkers, we could predict chronological age with R-squared of about 0.8, which is really quite good, quite good.

It was about 114 or so subjects, but that, you know, each one of those looks at the different aspects of the aging process overall, it gives you more information.

But as we you know, we found that over time, adding more and more parameters to it gave us an even better understanding of what was happening in each individual.

You know, just parenthetically, I would say I talk a lot about biomarkers of aging. And when I go over the multi, the multi, the bottle, different markers we use in our practice, what we call multi-omics.

Now patients are often sort of like well I the cardio age of 60 but I'm 50 and I have a pool my age of 40 but I'm 50. You know, what does this mean when there could be 30 or 40 years difference between the biomarkers of aging?

Does it mean that they're all just a bunch of hooey? No, it doesn't mean that at all. It means that they're measuring different aspects of the aging process.

Sure. And each aspect go along at a different rate. And so you get the information for that aspect of it, and you integrate it into the rest of the information.

You hope that whatever you're doing is moving each of those in the right direction, for sure. And that's something that I from the beginning, I never took the bait on the Women's Health Initiative as an option.

I didn't say all people need to stop. You know, you look at a woman who's like a smoker and she's overweight. She

probably doesn't need estrogen. It's probably a risk.

But, you know, to your point, it's like not everything is going to age the same. If I'm a chess master, but I work in a coal mine in West Virginia, maybe my brain's good, but my lungs are shot, you know?

And that's one thing that never sat right with me with the telomere measuring. Because, you know, even Blasko, who's the queen of this field, she'll say, well, neighboring niches in the liver could have different ages depending on their most recent progenitor.

Right? So everything is going to age at different times, just like in your car, like your brakes. You're not going to age the same as your motor or whatever as well.

But let's talk about the elephant in the room, which is measuring telomeres, you know, because people use it. And, you know, I think the devil it really was in the details.

I remember I used to draw my blood to send it to four labs and then come back with numbers like 7009, 11 and 13 year over year intra lab. The variation was small, but the methodology puts you like, if I went to you and you told me I weigh 70 pounds, 90 pounds, 100 pounds or 130 pounds, I'd be like, you don't know what you're talking about.

So you can talk to people who've gone to different labs and been confused. If you mean, first of all, weighing is a much simpler thing than measuring, you tell.

I like your haircut, the molecular level you're trying to put together, all these, these varying telomere lengths because, you know, there are multiple different telomere length within a cell and between cells between cell types.

These chromosome is differing tumors. Maybe there's they're caught. They're correlative to a certain extent. But and then also which cell types you're using.

So certain places use pbmcs other places will break it down into lymphocytes versus granulocytes which you know, in the granule sites are in there. That's going to change things.

So I mean my advice is to stick with a particular lab. The changes that happen if they're a good lab and they have a decent covariant coefficient of variation, will give you information.

But, you know, the other problem we have is that, you know, median or mean telomere length is, you know, I think an important thing to know about a patient but isn't going to tell you the whole story. Yeah.

I mean, really you want to know what's happening in the cell type of the, you know, that you're particularly trying to change. So yeah, I feel a in in senescent cells or in naive T cells.

Well, I can't do that. I think it's true. And, you know, the sad thing about the way that academia works, it's like they just take whatever's accepted and then run with it even though it's invalid, like leukocyte.

Like you said, there's multiple populations. And it depends a lot. I mean, but if you're looking at the liver or the lungs or whatnot, you want to measure other telomeres.

But over the years I appreciate that you used repeat in Canada. The flow for this technique seems valid. The qPCR is never trusted. And the Lifelink. I was sad in the early years when they abandoned the shortest telomere measurement because now they got this test slipping.

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Right. The Tesla tests is is, I think, a very good thing to be confused. The car companies. But it's you know, single to in length analysis which is great.

Look I think you can get good information. And I think everybody should get at least one telomere length test because definitely want to know if you're in the bottom 10% versus the 50th or the top 1%.

Because if you're in the bottom 10%, if you're below the bottom 1%, then you're a significant risk for multiple diseases, increased risk of cancer, cardiovascular disease because you just don't have enough reserve or for sure.

But, you know, people were just they were under thinking the whole fast literature in the last 17 years has been like, oh, we measured your blood telomeres.

And then we inferred something about your esophagus or your stomach. And it's totally ludicrous to think, oh, I check the tire pressure. Now, I know how old the carburetor is.

It was kind of crazy. Well, let's go to your published body of literature, which is significant in this field.

Telomere Measurement Methods and Limitations

So you published a study with Cal Harley, one of the originators at Geron of the product. And this was 2011. And you found that the senescent are the representation in the blood of the senescent or the the non naive T cells went down with J 65.

Can you explain how that could have happened? Well let's see, I don't think we know exactly how it happened. We certainly didn't have the molecular pathway worked out when we saw that.

I believe that what's probably happening is that the mitochondria are becoming more efficient because of the way in which 65 can get into the mitochondria, improve mitochondrial health, perhaps through, sorry, let's say in 65 to 65 increases telomerase.

Telomerase leaves the the nucleus and goes into the mitochondria and starts to make it more efficient, which could potentially then cause the cells to undergo programed cell death and no longer be sent, you know, so your number of senescent cells decreases.

But we certainly did see was a reduction in senescent cells, about 20%. Well, in that study and, you know, it was that was in patients that were CMV positive because CMV positive patients have more senescent cells by about, you know, two fold more or even three fold depending on how long they've had.

It can be being cytomegalovirus, the herpes virus number five. That's relatively benign in immuno competent patient. But we know through I think a lot of studies that and what's not widely appreciated though is that it kind of slowly ages your immune system more I think.

So I think you really you really presented beautifully. I remember listening to that, that it's really probably one of the hidden drivers of stem cell aging.

But let's go to your next study in 2013 with Cal and Wayman, and you guys looked at improved glucose, insulin, cholesterol, blood pressure and bone mineral density in some.

You think that was a pretty robust study design. Well, again, that was the same patient population as the first study. Just a second paper that looked at other markers.

We just proven that to the immune and telomere length results in the first paper. And we wanted to present the second paper just because it was the first paper out.

And already but also it wasn't a randomized control trial. They were doing other things. But we did see this signal of an improvement in bone density and in lipid profile, etc..

The things you mentioned, glucose that we were a little bit surprised about, but now with subsequent studies, we're

less surprised because we know the way in which to 65 through activating telomerase transcription, that allows it to make cells more efficient metabolically and improve these things.

So it's the subsequent studies that sort of kind of have borne that out. The death is probably real, even though it wasn't a randomized controlled trial right now.

In 2013, Maghera et al did a study and they deactivated the mAPK pathway and kinase pathway, which is a master regulator of stem cell differentiation, immune and inflammation.

And they said this is how T 65 works. And it may be. But let's look at, you know, the nitty gritty of how the origin story came about. Like according to Gretta, who I was just with this week, and she's like, well, I introduced Noel to these people at the Jockey Club in Hong Kong and and yada, yada, yada.

They wanted to use a Chinese herb astrologists and test some molecules. And they went to the Geron corp when Cal Harley was there and they tested some candidates, they may have tweaked some of it, but essentially for those that don't know if we're talking and we don't know because the the USPTO or the patent Trade Office, they try and obfuscate the identity of the molecule.

So assuming it is something like something similar to a strangler site for or cyclist charge, now it has that four ring steroid moiety, right. That structure.

So comments on that like are we just given like a relative of the steroid molecules. We age because our telomeres shorten and our stem cells to play. But what if we could support both?

I've been taking tier 65 for 17 years. It's the only supplement I trust to support better mood, better sleep and exercise recovery at age 57, I don't have any gray hair and I don't need reading glasses to 65 is available now.

Go to Recharge Biomedical E-commerce to iPhone 65 and enter promo code Recharge ten to save 10% off. I mean, structurally it looks quite similar. There are many other molecules in the SAP and in family that look like that.

You know, it's a small molecule, so it can get into a lot of cells is lipophilic. That's why you can give steroids to the skin and hormone replacement therapy or testosterone replacement therapy.

I have, you know, a molecule of that size with that structure. I'm never I'm never going to stop being amazed by how many things steroid hormones do. Yeah.

Okay. 65, however, is, you know, I wouldn't say it's relatively inert. I guess that's if it really works to turn on telomerase and, you know, through that mAPK map kinase pathway, but it's the right size to sort of get into the cell easily.

Do what it needs to do, sits on certain receptors. I don't think we know exactly which one at this point sits on. We believe it sits on the you know, De represses the promoter of telomerase, but that hasn't been perfectly worked out.

And so, you know, I think, you know, it's another one of those molecules in that size class and in that structure that, you know, just has amazing, amazing tropic effects.

Now when you look at the original patent trade office submission and you look at the dose response, you see that it can be effective over three orders of magnitude or concentration.

And I just wanted to talk about your 2016 study where you guys took CMV positive people and you gave them multiple, multiple, multiple doses and the regularly available 250 unit or 25mg.

Now, it actually shows statistical significance in increasing telomere length, whereas four of those bad boys didn't. Do you have any guess as to why was the study design?

I'm you know, the assay that was used for that telomere length was the life length assay, which is I think, because Maria Blasko, who is one of the premier tumor biologists in the world, you know, it's based on her technology.

Clinical Studies on T65

So I'm not saying that's not a good answer that way. But the the, the ranges for that are about two fold longer than in most other assays. So like a 60 year old would have about six kilo bases of telomere length or maybe a little bit less in that assay.

That's going to be ten field basis right now I know that. Yeah. You can maybe looking at some telomere regions where the telomere is just below the telomere, what effect it's having on those regions.

I don't know for sure what effect here 65 is having on those. I think there's a little bit more assay variability in that one. So it could be that that was just the assay potentially.

It's not the one that I'm used to using a lot, which is the flow fish lymphocyte lymphocytes and granulocytes from repeat diagnostics. But it could also be concentration differences.

65 is very highly bio. Bio has very high bioavailability. So the company did a study and one dose could give a good signal in one person. Serum levels in one person, the next the next person almost nothing.

It could be up to a tenfold difference in bioavailability. So it was a small enough study that you could see those kinds of, I think, just changes by chance.

Maybe somebody didn't get high in the flow. In vitro, though, there is a little bit of a signal where there's a sort of a sweet spot at about 0.1 micromolar.

Okay. And if you go higher, it's not as strong. And I don't think it's getting turned off, but it's not a strong signal. So that's a possibility. I think more work needs to be done there.

I've been using 60 in my practice with hundreds if not thousands of patients at this point. Some do well and need 1000. I use it to 65 and I can see telomere length increase or at least maintenance.

Some will do great on to 50 or even 100. Yeah. So I you sleep as if you're sick. What do you use to titrate the dosage? I use two things. One is over time I looked at telomere length trend, and I can see that I measure at least annually in my patients.

And I patients have been measuring it for 20 years, close to 20 years. And you can see trends up, up, up. But it's really kind of a moving average thing that you have.

You don't see the trend until after 3 or 4 measurements over 3 or 4 years. A more, more quick response of a marker is the senescent cells that you can measure at that UCLA immuno phenotyping lab, where they look at the absence of Cd28 marker on the cell surface, and that I see a signal in the first three months.

If the dose is good enough, you're getting enough of a tissue level, then I can see that come down 10%, 15%. So I don't see that. Then I'll raise the dose.

Yeah, it's really great. And that's really how I do it. And at the same time you can see the ink an improvement in naive T cells as measured by absence.

So you really are embracing the data as an endpoint okay. Now let's talk about this last study is 2018. And the current pharmacological design Maria Louis Fernandez and others gave a relatively low 16mg dose for 12 weeks.

And they claimed to show increased HDL decreased BMI and waist circumference, lower CRP, or for inflammation and liver functions, and lower TNF alpha.

Do you see that clinically? Because that's what happened to me at three months. I do see that again, clouded or, you know, basically confounded in a good way, I suppose, by all the other things we do.

So I can't say 100%. But what I will say is that that recapitulation in the randomized control trial of what we saw in the cohort study, almost to a t, happened exactly the same way with the senescent cells in the original cohort, we saw about a 20% reduction in the C and D positive subjects in the randomized controlled trial data that we did, looking at the effect of to 65 on T cell subsets, senescent cells, we saw a 20% reduction statistically significant in, you know, in that randomized control trial in the CD8 and the senescence suppressor cells.

So I was always been it's been incredibly exciting and remarkable that they have recapitulated themselves not only the signal, but also the magnitude of the signal in both the cohort real world thing and a randomized control trial.

So I think that is that that's evidence that these are really actual things that are taking place. And and because of the way in which printing on telomerase can improve not only to liver life, which changes gene expression, but also it allows for further cellular division, but also, you know, a control function, which is the not the basis of metabolic health.

Yeah, absolutely. Well stated. So you know where this is small space and that we've been giving lectures. You giving a lot more lectures at these over the years.

You know, there have been people who have come up with alternative telomerase activators. And so I want you to comment, you know, obviously, if I was first to market, in my opinion, the safety profile was great.

I never had anyone have a real bad effect on it. I think the baseline cancer rate incidence is lower. And so I think it's a very gentle kind of adaptogenic drug.

But, you know, if you look at the soft sciences out there and all these people are like, you know, I remember the Nobel Prize when it was like, I'm not taking that crap.

I'll give it to my dog. Maybe I will, but people are really funny. So I wrote a book called The Telomere America, and I just cited some of these people studies about meditation, yoga, diet, and how it affects telomerase activity.

But it's kind of a funny part, occupational hazard. I mean, I was listening to Peter Attia, who wrote this book, right? So incredibly prominent, big platform.

But his number one flex is that you can't slow aging or live a moment longer. And yet he writes an entire book and dedicates his career just to the opposite.

So I think people trying to inoculate themselves against being snake oil salesmen, and that's a really tough thing to negotiate. I mean, you actually, I think are the best at it because you have data and you have models and, you know, you're open to different modalities.

But isn't that like a soft underbelly of the anti-aging or longevity science that people just come out and say, I don't believe in this, but then they go and do everything to show that they do well, you know, look, it's a I think you have to separate the wonderful advances in gerontology where you can double model animal models, lifespan with various general protectors and interventions, be the gene therapy or even small molecules from what's achievable in the human and what's provable in a human because of our long lifespan.

So I do think that what we're doing probably does lengthen lifespan or certainly healthspan in patients. And, you know, we right now let's take for instance the start two inhibitors.

You know, they started using them for like Jardins or for Siga. You started using them quite effectively for lowering glucose. But they found in these trials just like they used to fight with metformin.

But you need even stronger signal that there was a reduction in all cause mortality and cardiovascular disease and

heart failure and progression to chronic kidney disease.

And we know now that those have an effect on the same pathways as rapamycin. So I think that these things are going on. It's just harder in a long live species like a human to prove it.

Dosing, Wearables, and Longevity Debate

So people are trying not to get labeled that way by saying, you know, lengthen your life or slowing your aging process down or reversing aging. But ultimately, that's our goal, and I think that we're moving incrementally towards it.

I think that's a well stated. You know, it's like I have I just ordered another one of these wearable rings. I had tried the Galaxy. The oral is, of course, the most popular, but it's pretty remarkable.

They they do a great job with sleep, architecture and quality, with so-called heart rate variability, a readiness score. And now they have SpO2 and temperature and blood pressure and even an inferred glycemic rate.

But there's a lot of, you see 20 healthy 28 year old people wearing patch. Yeah I'm like, wow. But it's like people are obsessed. You know, maybe it's a form of the culture of narcissism, but we're obsessed with knowing where we're at.

I mean, you pretty much know if you're tired or sleepy or hungry, but nevertheless, they have so much data which is being fed into their models and clouds.

Is there some onramp onto your model for that? Because like, you're kind of saying in the subtext, when you have abnormal shifts in glucose, you get inflammation, you know, and that leads to inflammation as they like to say, right.

So it's all part of, you know, you got your glycan age, which you do. You got your epigenetic age, you got your driver's license age, you got your telomere age, you got all your organ systems.

I mean, it's all happening at once. I mean, we're fooling ourselves if we're thinking we're not getting older, we are. But can we slow it? I would say yes.

But how do you deal with the people that come at you? Like, oh, I want to believe, but I don't want to be tricked by snake oil. Like what? What? How do you how have you dealt with that?

Well, I mean, I give them I give them their own data. So we measure it. We know there's not a lot of controversy that more elastic arteries are better than stiffer. Right? Right.

I don't want controversy that lower blood sugar is better than higher blood sugar. Right. So or, you know, more delta or deep way of sleep. Slow wave sleep is good for you.

So if you can show in those individual markers that there's progression and improvement, that's how you can you know you can. That's the end of one experiment, the most important one.

You know, Michael Schneider at Stanford has sort of pioneered the whole concept of looking at multiple biomarkers parameters, you know, hundreds if not thousands of them in an individual.

And one of those individuals was himself over time. And then you see perturbations from steady longitudinal trends. That's an early marker for bad things starting to happen.

And I think we should be doing that with ourselves, just like we do with our cars. When mean check engine light comes on and you want to do that. So you're a fan of wearables that I'm a fan of, wearables I'm a fan of.

I mean, look, I don't think a 20 year old needs to have a CGM on them if they're 15% body fat and their diet is pretty

good, I mean, that might be interesting to them, but if their hemoglobin A1, C is 5.2, they're doing a pretty good job.

There is a tendency you know, the Brian Johnson tendency towards over. Yeah they've really gamified it to try and get their number. For those that don't know, Brian Johnson is a Johnny come lately biohacker with a lot of money and he measures everything and he does interventions.

And so if you're at one you're aging one year per year biologically. But he says he's gotten down to the top of the leaderboard at 0.4 something. So for every year that I'm aging, he's aging 0.4, which is, you know, great.

Yeah. So I mean, that's that's you know, I think that's a reasonable goal to sort of work on that sort of thing. I use the pace of aging in my practice without the so one, because that's looking at one aspect, gene expression.

Epigenetics. There's other things to look at, like how how much air you can blow out in the first second for pulmonary function. Yeah. All these are very well established long term biomarkers that predict mortality.

But I think the idea of paying attention, like dentists do, to maintaining the health of the oral cavity should be applied to the rest of the body. In the multiple organ systems we have, we have a lot of very good biomarkers to do that. Yeah.

How deeply and how comprehensively you decide you want to invest your time and your money is up to you. But, you know, do it the smart way. Do it with somebody who knows what they're doing.

But I think that's better than waiting and being reactive. Well, I think it's amazing that people can have this real time feedback about their stress levels or sleep quality.

I think it is a net positive, you know, but it's interesting that I can see things in the data and, and abstract patterns that even we can't. So I think it will be a good health partner going forward.

But when when you hang around with these people long enough, these venture capital people and you know, these singularity people, they they've been promising that in 15 years we'll hit a longitudinal escape velocity.

And that was the over 15 years of past. So that's kind of a red flag for me. Whenever I hear someone say, in 15 years, there'll be no aging. What's your take on that?

You've been in the field the longest 27 years, probably, yeah. I always use the example of what happened in the 60s when there was two major moonshots taking place.

One was the getting to the moon and the other one was the curing cancer. And everything was like curing cancer. That shouldn't be too bright to but to be a problem.

But getting off the moon. Forget about what happened. Got to the moon, you know, in 1969, right? So I don't think we can predict with great certainty.

But I think the advances in systems biology, AI and the ability to analyze biological problems in a very comprehensive way will accelerate it. And I wouldn't be surprised if we reached Levy in the next ten years.

I'm not going to guarantee it, but I wouldn't rule it out. I tell you what, you know, I've had lots of people get help by the year 65. I'm sort of a one trick pony.

I don't do testosterone. I wish I would be a little scared about it. I'm not a fan of growth hormone that much, because I see these guys at the conferences and they look like blockheads.

I don't let that scares me, but I'm hopeful that interventions and people, the consumers actually are sometimes way ahead of the doctors and nutritionists.

The natural past. Like they're really hungry for this information. I think biohacking is a real thing, but if they want to have a one stop management.

I know you've licensed the physio aids platform and you have doctors all over the country. I have the equipment in my closet, but it's just the setup and the, you know, upkeep.

And the thing is, I'm in and of one practitioner, it's kind of prohibitive. But people can sign up to objectively measure on a mature, thoughtful platform.

So how do they get plugged in to a doctor who can measure these things? Yeah, I just go to physio age.com coag.com and you can look at the software platform what it measures.

And then you can always email us in to find a practitioner that's utilizing it. I'm working right now to take that same software direct to consumer with a treatment program, sort of like a lot of ones that are coming out now, function health and that sort of thing. Yeah.

And you look out for that maybe in the next 6 to 12 months. Oh I think that's great. Yeah. I just talked to a guy who has he's a data scientist and he has a company called Heads Up.

But yeah, there's so many competitors in the space be like heads up sort of. Yeah. And that's the journal. They take your different things from your wearable and they present something.

They may give you advice, but everyone is trying to monetize the space. But you have like a pretty mature actual model. And so if you could push that consumer that would be amazing.

But stay tuned. Yeah. All right. And if they want to see you personally as a patient they've got a Raphael Mediacom Raphael Medical. Oh all FDLE medical.

Are you still Central Park South or I'm still here. Yep I see five years. Cape, I think. Well, thank you for helping me with my health journey. I feel good. No pain? No.

Yeah. Appreciate that. Good to talk to you. Yes, sir. Thank you for tuning in to Doctor Talks. We hope today's episode has enlightened and inspired you on your path to optimal health.

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