Introduction and Guest Background

Hello. This is Doctor Joseph Raphael, your host for the Telomere Summit. I'm very pleased to have today, Doctor Joseph Cleaver, on the show to talk to him about what he does.

Welcome, Joe. Thank you for having me, Joe. How are you today? I'm well looking forward to this conversation. Let's, tell our listeners about, your extensive background.

Doctor Cleaver is a co-founder of the Paradigm Wellness medical group in Napa Valley and Dallas, and as medical director at Boutique Wellness LLC in Winston North Carolina, and has been practicing medicine since 1990, just about the same as me.

He is a rheumatologist and is board certified, an internal medicine fellow of the American Board of Anti-Aging, Regenerative Medicine. Doctor Cleaver is a board certified, is board certified also in anti-aging and regenerative medicine, has achieved certification by the ABA and Advanced Metabolic Endocrinology, specializing in bioidentical hormone therapy as a faculty member at a forum and the Metabolic Medicine Institute.

He lectures nationally and internationally and holds the position of Adjunct Clinical Professor at George Washington School of Medicine. Doctor Cleaver lectures extensively on longevity, analytic science, esthetics, and scalp and hair restoration, and actively trains medical professionals for a forum and LA Kind of Renaissance Group, LLC.

He has authored and published research textbook chapters and articles for international sports and anti-aging magazines. While at the Cooper Clinic and Cooper Institute, he conducted homeland security NIH research as a biologics expert with 15 years experience in regenerative esthetics.

He applies this cutting edge regenerative medicine science to minimally invasive esthetics of the skin, face, scalp and hair rejuvenation. Again, welcome Joe.

Looking forward to our conversation. Why don't you just start off by telling us a little bit about how you came to be in this field. I was like to hear the journey.

And then we'll get into a little bit more of what you're doing currently. Yeah. So first, thank you for having me. This is always an exciting topic. Telomere biology, analytics, longevity.

It's a pursuit of mine, probably for the last 20 plus years. And, you when I first, was introduced to, for example, a for him, I guess it's 18 years ago now, and, and I interacted with a lot of colleagues and they all had a story that, you know, they were convinced all physicians or medical practitioners, and they ended up with a kind of a cryptic type of symptomatology, like chronic fatigue or whatever it might be, and could not get the, satisfaction or the answers from conventional medicine.

And that's what drew them into regenerative or integrative, you know, medicine. And truth be told, when I was educated in University of Rome, in Rome, Italy.

Right. And the, you know, the heart of the Mediterranean. And that's really what started my integrative approach, because the, the, the, the subject matter, material in, in American medical school, at least in my, you know, alma mater, University of Rome was already it took an integrative approach.

And then when I got back to the States and, it might, you know, residency up in, in, in new Jersey and fellowship in Miami, down into the University of Miami in Miami, Florida.

I ended up in Dallas, Texas, and, I was at Baylor. I was a professor at UT southwestern and then went over to the

Cooper Clinic one day as a patient and ended up, being hired actually on staff there.

And Cooper Clinic was one of the original, I think, of integrative medicine, preventive medicine, clinics in the country started back in 1970. And, I was there for about 14 years.

And we the approach was always to, embrace lifestyle as a preventive, therapy. And so that mindset was always there. I never had any, you know, medical issues.

And I've always, was exposed to this all the way back to medical school, then Cooper Clinic, Cooper Institute doing research over there and and as a rheumatologist.

I did a lot of sports medicine at the time, and that's what, expose me, if you will, or, opened the door to using PRP biologics for MSK injury, etc. and that, you know, you've been in practice.

Journey Into Integrative and Longevity Medicine

We've I think we've been in practice. We were just talking about that probably about the same amount of time. And sometimes your practice, as much as you think you're steering it, sometimes it steers you and you you, it ends up morphing into something that you never imagined.

And that's what my how my practices developed and evolved over the years, to a point today that, I, have taken integrative functional medicine biologics, telomere biology, the hormone balancing, etc., and have expanded it into functional esthetics.

And that balance between, the looking good and feeling good really opened the door to similar ethics and longevity. So longevity was always part of the practice.

The, and the last five years of really taking a deep dive into analytics and understanding the real drivers behind, or the most, you know, the most potent, if you will, or important drivers behind, premature aging, inflammation, etc.

and, that's, that's how my, you know, kind of like my practice kind of, morphed over the especially the last five years. So I balance about 50, 50 esthetics, hair restoration, skin rejuvenation, sexual performance, etc., because all those things are knit together beautifully in a longevity program.

Yeah. I mean, that's, that's a that's a great story. And, I was just thinking when you said inflammation that, you were you trained in Rome, and, that term was coined by Claudio Franceschi, an Italian professor, researching in longevity medicine, I think back in 96 or something like that.

Right. So, yeah, he came from that sort of a mindset. You, you talked about, having a precision set, a lytic protocol. And, I'm curious about how you put that together with peptides, telomere biology and, to 65 and talk a little bit about how your, you got into that and what kind of things you do.

Right. So the, you know, the, the, the real deep dive in the last five years, as has led me to that, you know, that that cell senescent burden, and, and the impact in lifestyle, you know, fundamentally, we're all first and foremost lifestyle preventive medicine practitioners.

And that's the purest form of medicine we can prevent through the, education and lifestyle. We're so far ahead of the curve in, in maintaining quality of life and preventing the diseases of aging that, we we, we see more and more in the literature.

It's been around for quite a long time, just satellite X and the impact or what analytics do as a science to to, to initiate that apoptosis or destruction of zombie cells or senescent cells or my patients, when I call them a zombie cell, they never forget it.

So I use that all the time. But especially the SAS cell, that secretary phenotype, that is the real bad boy on the

block that we need some of the healthy, short lived SAS cells.

But those longer lived SAS cells that become very inflammatory, they create a lot of good inflammatory, substances. IL six. And so, you know, alpha etc., that are the real driver behind, the premature aging and those diseases related to aging, diabetes, dementia, etc., cancers, heart disease.

What we see with, the the with cell senescence is the in that lifestyle has a huge, plays a huge, role in, an increasing cell senescence burden in the body.

So when I started, you know, I was doing longevity for many years, and, and I've used the T 65 and telomerase activator for a, as a cornerstone of my therapy.

And we we have a much better understanding, at least I do. And how I link the impact of what telomerase does and the impact it has and the non-canonical aspect or function it has in mitochondrial health.

Yeah. So we were kind of grasping, if you will, you know, ten, 15 years ago, it's like, oh, everything's mitochondria, everything's telomere length. And we I think it took a while least for me to understand how they integrate together.

And they they're you can't separate them out. They, they're they're science as they are. Now that shows if you maintain telomere length, if you don't have dysfunctional telomeres, that mitochondria function better.

Mitochondria, a biogenesis biogenesis is optimized. You know, Ros oxidative stress is decreased. And it's and they all they all interplay between each other.

So but the end result is when you read the literature, it's like, oh yeah, telomere shortening is very important for, driving, you know, a cell into senescence.

And mitochondrial dysfunction is very important in driving a cell into essence. Epigenetics. You know, we can we can we can go through the list. But, when I started using, for example, to year 65, I didn't know until the I read the paper that you published recently, like I had to kind of, coax that patient and persuade them and and say, give it some time.

Give it some time over six, nine, 12 months and over time, you will even see the impact it has on, telomere shortening or telomere health and mitochondrial health on skin.

Health, for example. And, overall sense of well-being, overall sense of energy. And now that I now that we're, you know, fast forward to 2021, we really I realized that maintaining of telomere function, maintain telomere health has a huge impact on, mitochondrial function.

And so you had to get that patient over the hump if you were to say, hey, doc, I'm taking this for three months. And, you know, you tell me about these telomeres and telomerase and all this other stuff, but I don't feel much different.

I said, well, stick it out, you know, give it give it six, nine, 12 months and see how you feel. And if you don't think you feel any better, stop it and then see how you feel.

Because, inevitably they do feel a difference in sense of well-being, that sense of, really just energy every day. And it all started to make sense to me when we looked at the literature recently with our this pandemic and the and the and the virus that is, responsible for creating the pandemic and the impact it has or that virus has when it in the acute infection, it destroys mitochondria.

So now, you know, when you're looking at the literature that some of the literature has been published, say patients who have shortened telomeres, do do poorly, they when, if they, they, they, contract this infection, this viral illness.

Yeah. So if we're have somebody has a longer telomeres or somebody who maybe on a telomerase activator has improved mitochondrial health, mitochondrial function, they might fare better because the cell function system

functions are going to be added, I think.

And at a different level of functionality than somebody who has shorter telomeres. Yeah. So that makes sense. Yeah. I think, when those papers came out, I was really fascinated to see that it was sort of, sort of two mechanisms.

One, is there senescent cell burden is the sort of determines what your inflammatory state is.

Telomeres, Senescence, and Mitochondrial Health

And people with cytokine storm, it occurs because of an overreaction of the immune system. Because of this, these cells that are producing the versus molecules on the other side, though, it's your ability to for your lymphocytes to divide fast enough to fight off this viral infection.

Ibrahima Vivas is hypothesize and then subsequently I think we have Lascaux and French group have shown that you actually, when people with shorter telomeres do have worse outcomes, and you can bottom ten versus the top 90th percentile, you're, you're, you're have more severe disease because in the short term they're not able to the lymphocytes are not able to, to replicate enough to, to take it off.

So it's those two mechanisms, both tied together through telomeres and through senescent cells I think really kind of, you know, sort of brought it home.

So if someone is doing whether it's lifestyle hormone replacement therapy, you know, estrogen can turn on telomerase to a certain extent as well. Or actual telomerase activation, if they can keep their telomeres longer, they could potentially be less susceptible.

They could also be potentially used as a as a stratifying tool, which I'm doing in my practice to see, you know, if you have really short telomeres, is it something that you need to be more careful about?

I mean, it's obviously not backed by a large body of data right now, but I think that could be a direction to going. We see people that are 25, 35 years old without any of these other risk factors.

Obviously, age not being one at that age, succumbing to bad or getting that disease and sometimes succumbing to it. What's the reason for that? Well, probably because in some of those systems, their biological age is not that young.

So it's really the pandemic has brought, I think, us into this kind of area where we're looking at short term adverse effects because of something that's you know, impacting the long term aging process.

It's really fascinating. You, employ, which I don't that much in my practice. So I'm curious to hear about your your, you know, your clinical stories about it and your approach to, actual synthetic molecules like visitin and, and quercetin, and maybe and some peptides tell me a little bit more about how you go about that.

How you would your patient choices, what you monitor for it. To, to, give us a, our listeners an idea about how you approach it. So when, when I lecture and, you know, educating a medical practitioners, excuse me, or in my office trying to explain to a patient why they would embrace and want to be interested and, and, and start a, an analytic program.

Explain them to them. There's a, there's two sides of this equation. We have a healthy stem cell, healthy, cells in our in all our systems on this end of the equation.

And, they're on the other end of the equation is that senescent cell or the basically an apoptotic cell in the middle. We have a senescence cell. And we want to have two targets.

One is preventive. And that's where I think actually, telomerase activity 65 coming in makes sense to me that the

especially in the data that, that the you elucidated, looking at a decrease in senescent cells over a period of time, it makes sense to me that it's preventive.

And that's why K 65 is a cornerstone in my, in my analytic program that patients are on 365 days a year where as, early on. So that's the preventive part.

And some of these analytics are, you know, purely analytics are going to create and induce apoptosis. But they have double duty. They have several of them have double duty, if you will, for example, Visitin, it has some preventive capacity.

It also has a senility capacity. I use a lot of I use a lot of rapamycin rapid logs. Same thing. A decrease in mTOR can have a benefit or on both sides of that equation.

So we want to prevent or maintain a healthy cell. And that's through telomere length as you mitochondrial function and minimizing oxidative stress and maintain that those, those healthy numbers of the cells that we need to, to optimize organ and system function, then we inevitably we're going to jump over, you know, periodically, cyclically, we're going to dump, healthy cells into that senescent state.

And then what we want to do is, periodically rid the body of those senescent cells through a pure synthetic, if you will. So, what's a what are the pure solutes?

I'm sorry for the pure analytics versus the ones that. Yeah. So, you know, there are some fairly exotic analytics that, are not mainstream, that, are not, you know, are not honestly are not FDA, approve.

We do a lot of things that are off label. We do a lot of therapies that, that, that are, that aren't recognized the standard of care by the FDA or by other, you know, and medical, guidelines, if you will.

But there are peptides that are analytics. There are peptides that are mitochondrial peptides. So what has come to light recently is, there are several, publications that look at everybody is embracing nad, for example, every everybody I got me to do IVF NAD because it's going to increase my mitochondrial function, ATP, etc.

and improve my stem cell, function in health. That's probably true, but if we haven't removed the, the SAS cell, that is also going to benefit from, for example, an NAD infusion.

I think that is a was an oversight, if you will, more, until more recently in my programs and or in my, in my main protocol. So what I'll do first is I'll start setting up my, my analytic program that is preventive.

And that is the 265 the flavonoids such as visitin, for example, before and before I go, come in with mitochondrial support to maintaining a healthy, stem cell, presence, I'll move into the analytics and then, clean them out through intermittent fasting.

We know that, you know, manipulating our caloric intake or intermittent fasting, etc. fasting, mimicking diets certainly is beneficial up to a certain age.

There's studies that show it really doesn't. It's not that effective. Every day, as we get later in life, and try to use intermittent fasting, but there's still probably some benefit.

So that plus, certain peptides actually that are, are drive p53 into that, senescence cell into that cell and, and induce autophagy. So now that I've cleaned up those, those senescent cells, those, those cells, then I'll come in with that, you know, really, potent or that really focus targeted might have conjugal support.

And there are peptides that do that. There are there are other, you know, mainstream, if you will, very well known. Nutraceuticals, such as carnitine alpha, etc., resveratrol that that support mitochondrial function.

But all along remembering that the mitochondrial DNA are so different as you know so well the nuclear DNA and

we have that they have that capacity to utilize, you know, for example, and 2 to 65 to shuttled back and forth between the nucleus and, and help, improved telomere length and function, but non-canonical aspect of to 65 or telomerase activator.

It does improve or it it will, help mitochondrial DNA, heal from damage and and there are other things out there. And so fascinating article I just saw on inter mitochondrial melatonin and its impact on mtDNA and all my patients.

I you know, I'm getting a little off track here because I just thought of that mitochondrial melatonin has a huge impact on mitochondrial function, mitochondrial biogenesis and healing that.

But that scarring mtDNA that mtDNA is has so much more damage on a daily basis than our nuclear DNA. So we have to keep up with that. So I just it's just another on the list in addition to, to to C5 or telomerase activator to maintain mitochondrial health.

And so you mentioned you mentioned quercetin as far as the peptides are concerned, which peptides because I'm not as it's not as what about peptides as you are.

Which peptides do you that have some similar activity? So one that, is a lot of anecdotal evidence and experience, is, something called foxo for DRI.

Analytics, Peptides, and Senolytic Strategies

It is a, it's a pure analytic peptide that shuttles p53 to that cell to induce autophagy. So, the, the Foxo Foxo four blocks p53 in, you know, autophagy.

But the, brilliant researcher out of Netherlands can't remember his name right now. He's he was in his this was, 5 or 7 years ago or so. Came upon this, this, peptide that was engineered to, to induce, apoptosis and not and it's very specific only to senescent cells.

And it's the the mechanism is through p53 and p53 will, you know, induce a pop ptosis. So in my program, plus minus, I'll explain that to a patient where we are with the, with the, with the, you know, the development of peptide such as fox.

So for dry and whether they, they, you know, it's, you know, we, we, we, we talk about, and discuss the upsides and downsides of, newer peptides. And, to date, it's been very, very effective.

And what I see now is, you know, after we start those, preventive, you know, analytics, we'll call them, clean up our, our, our senescent cells. Then we come in with mitochondrial, support with other peptides, other, you know, other nutraceuticals, etc., that, that fuel mitochondria biogenesis and function, etc., that, the, the, the testing that we do now, thanks to you and UCLA and, and we really didn't have testing before that is specific to, immune senescent burden that I believe is really the master switch of probably aging of all our other systems.

So it's, it's pivotal to, we can now get a senescent cell, burden assessment that we couldn't get before. And I think that's the only commercial available, lab test out there that does that.

And a clinical level. Yeah. So there's, you're referring to UCLA clinical, immunology or immuno phenotyping lab that we, samples to I've been doing in my practice now for, about 13 years.

And they look at the Cd28 negative, suppressor cells. So CD8 positive, see 28 negative cells, which are in a pretty large body of literature, thought to be sort of senescent or at least laid differentiated cells that are create nasty cytokines and accumulate in large number.

And people with, CMV infection and, other stresses to their immune system, that's available through to sciences or through physiology. My, software company that looks at things, it has a software for measuring these kinds of things.

There's another company, that has a SAS activity. Marker infinity that I've not got much experience with, but I think more coming down the pike because I think it will be it's really I always like to practice sort of evidence and of one type medicine where you see what the effect is.

I can give a blood pressure medication. You see the blood pressure go down, you give us an analytic, you see the similar Excel, burden go down. And like you, as you kind of pointed out, the our paper should up in about 20% in senescent cells after, taking to 65.

And that is, you know, starting in three months and not six months and nine months. And so that's a good marker to follow. And it's it's a shorter term. One is three months.

Telomeres take a longer time, I think, to get a good idea about which direction you're going in. But yeah. So those are the two that I'm aware of right now.

There are other companies that are working on, you know, beta glycosides, you know, SBT gal, markers to look to see, you know, whether or not these things are being effective.

But I think, you know, the whole one of the whole issue, sort of with, with a license as a therapy for aging is the whole concept that if you're taking out these cells, other cells, stem cells within, the tissue issues that you're looking at would be a muscle, liver, you know, brain have to divide to replace those cells.

Their telomeres get shorter, and then you're going to then get back to a senescent cell burden that may be even a little bit higher. Michael written about this, in obese geriatrics recently, 2019.

And so the scenarios are great, but you then have to help the telomeres on the cells that need to replace them so that they don't go down that same pathway.

And I think that's maybe behind some of the failures that have occurred in some of the, the earlier, studies done by, by some of these biotechs where, you know, they removed a large number of senescent cells, but then later on, the mouse died, which is not something you want to see happen.

I don't know if that's behind the biotech's failure with their osteoarthritis drug, but, you know, I think that is something that they're going to have to start keeping in mind when they're, when they're, when they're looking at removing, senescent cells.

So is foxo for dri, a, an IV peptide, a subcu oral. How do how do you take that and what kind of, yeah, it's, right now it's, it's IV, and it's a short course of, three infusions.

We've tried it, subcu. But the, I think it may be the, the pH. But it can. It's pretty painful. Yeah. So we we don't we don't usually do. Subcu diluted in, D5, and run it in over, you know, 30 minutes to an hour.

And the if we extrapolate from the lab studies, lab animal studies and, and convert it to, human dosing, it when I'm using a new any, any, any, you know, therapy, I'm very cautious and conservative.

So I'll cut that dosing in half. And, I haven't had any side effects at all. I know that, there are patients or have people have experienced, if, a too high a dose, they can experience a cell lysis syndrome.

If we think about if we're destroying all these cells too quickly, too fast, that, that's a possibility. And I did have. Hi. I gave a talk. I think you were there actually, at, Cell Surgical, back in June or, May in Vegas. And.

Yeah. And, I remember one doc raising his hand and he kind of he gave he actually self-administered. He had it done and, he experienced, some similar side effects.

So, using it, you know, cautiously, it has a, I think it in so far, it looks like it has a significant impact on cleaning up that, that, that that senescent burden.

The studies in animals are pretty fascinating. They, you know, they they, they genetically engineered mice to age over a short period of time, as you know, pretty typical studies, and, took the age mice and, treated them with foxo for and within ten days, their, you know, their gray fur that was falling out started to restore in about 2 to 3 weeks.

They got on their exercise wheel and, and started exercising 50%, greater time than they did before. And, in about a month or so. You know, these are age mice.

So older systems are starting to, decrease in function, renal function that was diminished to about 50%. Was restored back to, you know, to original 100%, renal function as is similar to the, the young mice in the study.

So this is an animal study, and we're still very early, but it's a fascinating peptide as a, as a pure analytic, that, if you know, you understand, your patient understands, the, the, the, the product and understands the, the therapy and so, and the physician understands that therapy that, that's, that's something that's a, you know, a possibility to, to trial or, or have part of your analytic program, but, yeah.

So that, you know, I was going to say the 65 and the rapamycin, you mentioned before. And, you know, if we if we wipe out these cells or our body's just going to go in and replace them faster, potentially, we, you know, that that's that's a theory.

I it I, you know, I, I don't I don't know if it's true or not. I think it may be. So that's where that, that preventive part comes in where I set that up first.

So that's 265 the rapid logs, even visitin, and there's a long list of things that, help maintain stem healthy stem cells. There's, certain certain other peptides.

Some of the time a sense can do it. Some, some of the secreted dogs actually improve and maintain cell efficiency and function. Yes. So and speaking of side effects, particularly with the Rappeler, there was a recently, study published, in IPF, looking at IPF patients to and again on Linux, I think it was this afternoon.

They didn't see too many side effects, but I know that other other practitioners, like Peter Attia, has mentioned that whenever he takes in, some of his patients, take a rapid along, they get, ulcers in their mouth.

Have you seen that with your patients? And I wonder if giving them to 65 potentially prevents that from occurring. That's a good. You know, I haven't seen it.

I, I'm pretty conservative with, with rapamycin, I embrace it. Well, I'm sorry, what's your dosing on? Unwrapping mice when you get it. Yeah. Now we're just going to be I'm just going to mention the, you know, the the study is in the elderly with, the vaccine response.

They, I think they did 1.5mg daily. I did the three dosing schedules. One was five milligrams weekly, and then the last one was 20mg weekly, if I'm not mistaken.

I haven't looked at that study in a while, but the you get some immediate mTOR response with the daily dosing and you get some with the five milligrams, and then this is 20mg weekly.

I don't dose anywhere near that, but that's probably the best benefit for my, if I recall correctly, in the elderly patients, even having a improved immune response to vaccinations, I believe about 12 months out, that, it was the higher dose, actually.

And one of the side effects was, after this ulcer. So I'm using, 2 to 3mg about, every weekly, on my patients. And I haven't seen any, any side effects at all.

I mean, I haven't seen the ulcers at all, but he made a good point, I think.

Viral Burden, Rapamycin, and Clinical Monitoring

I know, the, the podcast you were referring to where when he was in residency, he was he was injecting bupivacaine into his. It was after the. So he's already prone to it, but it makes me think, one of the adjustments I made based on your, discovery of the immune burden from, you know, hidden viral loads, you know, such as CMV, is, I think, maybe part of the reason why.

Because I clean up those viral loads now, it it with the anticipation. Somebody has a smoldering EBV or CMV, HPV. And I learned this when treating these patients in our pandemic that we're, you know, struggling in with right now to treat.

And if we give an antiviral I've had patients, two patients that came back about 3 or 4, 4 to 6 months later and said, you know, I've had HPV on my pap smear for four years now.

And this was all of a sudden it's gone. I was like, well, that made me think, why can't I we, I can't we develop a program prior or even or build it into a synthetic program that, we'd want to clean up that, that, that viral load or that viral burden.

And in our, in our patients as part of a simulated program, because CMV is such a huge, burden on the immune system shortens telomeres drastically. That, it makes sense to me to trying to address that, but so which do you do you use, Valtrex or a fancier?

I mean, those are antivirals for, you know, most of the herpes viruses are not necessarily effective against, CMV, and they're usually the more toxic ones, like Jan's, like a beer and and, fastconnect that work for, for, at for, for CMV and probably, you know, so you're right.

And so you see some benefit from which are the antivirals they're using some of the, some of the antivirals that, that, are being used off label and, in the pandemic, I which ones?

I'm just curious, you know, it's, it's, for example, ivermectin is a is a very potent antiviral. Okay, okay. Know, I thought it was when you were, that you were referring to, Yeah.

I mean, interesting, whether that has, affect, you know, ivermectin has had a colored history recently. I mean, starting out with being fantastic and there's a biological plausibility for this antiviral and multiple biological mechanisms through which it might work, but then ultimately, you know, depending on where you stand on this, you issue, you know, not being necessarily proven to be effective.

So, but I, you know, it could be that it's helping to reduce viral burden in somebody that has I mean, I measure all the herpes viruses in my patients to see because of a study that showed that the more herpes viruses you have, the faster you're telling nutrition.

So if you have, you know, one and two, I mean, most everybody has three. Most of it has four. Not everybody has five. But as you get older, you get more of them.

A lot of people have six. And if you have four, versus one, you have a much deeper attrition rate over lengthened. And it would be great if we had, I mean, I mean, I don't know where the other is, I think published on ivermectin and herpes viruses.

I, I believe there there are some publications, if you dig deep enough into the literature, that is it has never been really of any, interest, up until more recently.

Right? Yeah, sure. Because how it came to light. We're not talking about treating therapy, you know, as a therapy in the pandemic, but as an off label therapy for certain, smoldering viral, loads.

And, you know, in your patients and, and, and that, we've seen, we've seen patients improve and these are, there's a combination there's and there's various, protocols, that may or may not use ivermectin.

That and we use some homeopathic, homeopathic therapies, sometimes, you know, the equivalent of the acyclovir, routine is a, the, a potent antiviral. And, and we've treated patients, for example, with chronic fatigue that have that wastebasket, the diagnosis that is, you know, it's, EBV chronic fatigue type patient with, with, surprisingly, good results.

So, it's kind of taken, you know, the, the cover off for me and, and and being able to, you know, insert that therapy, into a similar tick program. Just makes sense to me if I, if we can improve those telomeres, telomere length, certainly even in CVS. You.

So you you know better than anyone, but I, I are we are we addressing that that viral load will be in a long term, longevity program. Yeah. Right. The virus, that people carry is I think very certainly with regard to immune system, very important.

When you know how rapidly your immune system and you're dealing your length is, is is attacked, you know, we talked about the microbiome, which is obviously very important, but the virus, I think is actually going to turn out to be almost equally important.

And there's probably a whole bunch of other retroviruses, line viruses that we don't yet know enough about, but probably are having a significant impact on our aging trajectory.

You know, they're looking for a vaccination for, for CMV because of the, you know, the burden of infant, death and, deafness and etc. and then, of course, in transplant patients and in immunocompromised, HIV patients, they, you know, they'll be great to be able to stop that virus in its tracks.

But so far, it's eluded any, any vaccination program. It's a pandemic. It's just not as fast a pandemic as the one that has hit us recently. True. Yeah. Globally pandemic.

So, are there any other peptides that you, work with that, that, impact? You think, set a license or or, I know we people have talked about it and, being, a telomeres activator and a telomere length and, I mean, I saw the data on that was from, you know, some very from older, Russian studies that I would write surprised when I remember looking at in that field where, like, measurement technique was pretty not probably not great.

But I heard it talk about using it and some other people talk about using. Have you had any experience with that? You know, that comes up a lot. I certainly speak, and, lecture a lot on peptides and, and, inevitably, you know, telomere length longevity and creep into the, into the discussion.

And if we're talking about peptides, some you always get a question on every talen and what I referenced is a study from the Buck Institute. Adam, Marion County was done about eight years ago, and they were looking at, they were looking at the, telomere length and, the, and, how quickly telomeres can, lengthen what is the, mechanism or what is the, the, the therapy, if you will, that is used to lengthen telomeres.

And then they went back and looked at the I said, okay, we can lengthen telomeres in this rat, to make them a, you know, the equivalent of an old rat to a, you know, a 12, you know, a 12 year old, you know, human, equivalent of telomere length.

But they went and looked at the fragility of those telomeres and realized that they may be doing more harm than good because you can grow telomeres, and the length is not necessarily what is not the most important thing.

It's the quality of the and the structure and the function of those telomeres. That or I think the underlying most important aspect of telomere length.

So their conclusion was if you lengthen telomeres artificially, in a short period of time, the the quality of those telomeres, can be impacted significantly and may cause more downside than upside.

So when I create a program and we do to 65, for example, at length and telomeres, but it's a natural telomere. It's a

it's a substance that improves telomere length and quality of life and function of telomeres.

And it's done in conjunction with, lifestyle over a period of time.

Telomere Quality, Eye Health, and Closing Thoughts

And, you know, if you look at a list of things that lengthens telomeres and you and you said, well, what's the difference in, you know, arsenic can lengthen telomeres.

So we have to be very careful, and use a therapy. And, something like 60 or 65 that has been studied quite extensively. And in, in lengthening telomeres in a healthy manner.

I had Tom Dell on, recently. He's all adjusted to the, to 65 study on macular degeneration. And I want to just ask you, because there's a very plausible mechanisms for, you know, why it work to improve, the macular, function, through removing senescent cells and lengthening telomeres.

But do you when you, I mean, you do a whole program, but do you see any, benefit in eyes. I, I was curious to talk to the doctors. Prescribe a lot of to 65 whether or not they see improvements in distance, vision or presbyopia.

He said that they did pick up a signal with presbyopia, but, not so much with, with distance. Anything anything from your patients anecdotally? Well, I can tell you as a, my experience, is in the and a handful of patients that have been on tilt to 65 for a long period of time, short term.

No, I haven't, I haven't seen anything significant such as that. But the, after certain age, when our vision starts to start to accelerate in terms of, our visual changes and our visual acuity, seems to maintain, that visual acuity, and my I, I'd wear contacts, but I have not changed a, a power in my strength of my contacts in about eight years.

And, you know, and I and I, I'm in the age group that usually this is annually. You keep changing your, you know, the power, the strength of your your corrective lenses so that my yeah, you know, attest to that, right?

Yeah. I mean, I've had patients say stuff like that. I've also had some actually documented cases of, reversal, the presbyopia not to complete, no glasses, but, to see any of the lower, diopters, well, this has been great.

I wonder if there's anything, you know, so any closing thoughts that you have about, about, so license, your approach, and to, to tell our listeners any pearls to take away with them?

You know, I, I think we hit on a lot of the pearls already idea. And just the more recent ones that, you know, brought to light, you know, the, the viral issues and, the, the focus on to 65 as a, as just a it's a cornerstone.

It's a workhorse long term and and I, I knew it I know you knew it until that study came out and you published it, you know, and that that was something that, I, you had to really assume that had to be a, a, a hypothesis that, I was always waiting to be to to be, or to to to see the proof.

And now we have that proof, and it makes so much sense. You know, chronologically, timeline the way, tell about, telomerase or telomere, function works.

It it makes total sense to me. And if we have, we're going to fare better in any, especially any infectious disease. If we have optimized I look telomere length to me as a surrogate marker.

I certainly they have inherent function, but I know it's an in it's an inherent or it's a subject. It's a it's a marker of stem cell health. It's a marker of mitochondrial health and function.

And they're certainly going to fare better, especially when we start looking at the impact of, for example, this pandemic. And it has, how it destroys mitochondria in just a short period of time.

So if you already have deficient mitochondria and mitochondrial dysfunction, the dilemma rage dysfunction leads to PGC one, alpha dysfunction leads to decrease in mitochondrial function, etc., etc..

And along with p53, I mean, there's a triad that few people address. There was a study back in 2011 looking at telomere function and I just mentioned.

So we optimize our, our telomere function. We're optimizing that our, you know, our defenses and immunologically and in every system in the body. Yeah. That's study by, Egan.

So he, Adam a lot of it it's been, you know, great to put the telomere, health together with mitochondrial, dysfunction through the master regulators of biogenesis BCG, one alpha and beta, and that's a that's a that's a great, that helped me put things together, too.

Well, I also would say that, I'm a little bit envious of you because you've talked about lifestyle medicine, and you, you walk the walk and talk the talk.

You have a great, place out there in, is it, Sonoma where you're located is where you do, you do, you know, enjoy wine, big wine, teach, doctors about how to, do esthetic medicine and anti-aging medicine, and then also just really enjoy life, the way you learn how the the Europeans and the Italians do.

Something I'd like to get out there, check you out in there. Well, the guest house is always open for you, Joe. All right. Thanks, Joe. It's been a pleasure talking to you, too.

Good seeing you. Thank you so much for having me. All right. Take care.