Senolytics in Longevity Medicine: Dasatinib/Quercetin Trial Results Transcript

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Introduction and Guest Background

Welcome again to the Telomere Summit. This is Doctor Joseph Rafael, your host. Today, I'm very happy to have Doctor Edwin Lee on the program to talk about, all the great work he's been doing in his practice over the years, but in particular, very interesting study that he's just finished.

Hasn't, had all the data analysis, but we're going to get some interim results, on the effect of, similar to combination on patients in a clinical trial.

Doctor Lee is a bestselling author, international spokesperson and board certified endocrinologist in 2008. He founded the Institute for Hormonal Balance in Orlando, Florida, and practices functional endocrinology.

He is a co-founder of the Clinical Peptides Society and teaches many health care providers in the use of peptides. He also formed Save peptides.org, the not for profit organization to save peptides from the FDA.

His latest book is The Fountain of Youth with Peptides Discover the regenerative Powers of peptides. In addition, he is finishing up a six month analytic study in 21 patients.

As I mentioned, using the Saturn IB and quercetin. I should also say that as an avid triathlete and golfer and practicing what he preaches from an age management standpoint.

Great to have you on the show. Thank you Joe. I'd just like to start by getting, letting the listeners get to know a little bit about your journey, because this is a mostly a health care practitioner doctor audience.

You're very successful in your field. Sort of. How did you get from endocrinology, into age management and, and, in now into the, obviously into peptides, but also into the cell.

Alex. Sure. So, I got tired doing conventional endocrine, when I was a winter head in Florida. So I was the first endocrinologist in Central Florida, a small town.

And, I got very, very busy with, the first, 2 or 3 months. And after a while, I'm just getting tired of just doing bandaid medicine, and I kind of knew that there was another type of practice out there, and I was just really curious.

So I'll try and make you Joe. Probably about 14 years ago at Red Rock at the AMG meeting. I didn't remember I remember talking in the elevator and I was so impressed.

Like, wow, you did functional medicine in New York City. I was like, wow. Mean, it's like, I think I think you gave a talk, and I really got excited about the whole meeting.

I saw the light at the end of the tunnel, like, wow, there's something better than conventional endurance. So there's, huge cheers for medicine. Functional medicine.

So I took a lot of courses, and I started incorporating, what I learned into my practice. And then I decided I'm going to start in 2008. And I started the worst time ever.

That was the peak of the recession. You remember it was, oh, I already signed the contract, ten year contract and everything after that.

From Conventional Endocrinology to Functional Medicine

Just like if you want to win money, just do opposite what I do in Vegas if I go ready to go black. So, Okay, so in

2008, the whole economy is crashing, and I, I know nothing about, how to start my own practice.

And, anyway, I just, I remember asking my wife, like, a week before I started. Do you think I'll make it? Not knowing anyone here. Moving to Orlando. Not going to the hospital, not accepting assurance, dropping out of Medicare.

And I remember I asked her particularly that question. She denies that, but she said, I don't think you'll make it. Yeah, I saw that I had a similar experience when I did it, and I hate to say it, ten years earlier, in 1998, and it was sort of one of those field of Dreams things, you know, build it and they will come, and they just don't come quite as fast.

But they do come eventually. Yes they do. Yeah. It's it's word of mouth. So I, I kept telling my patient just to I just told my wife I just need one patient.

Unfortunately, some of my patients for Winter Haven, which is about an hour, south of Orlando, they came so that kind of helped out. And then, the practice grew word of mouth and, and, you know, now I'm being interviewed by you, and this is an amazing journey.

And, so, I'm not quite sure exactly how I got into peptides, but, I think I went to a conference and heard one person speak about, as I do a lot of research and got on the peptide wagon and eventually wrote a book and form your organization and, it's really it's been exciting.

Just a little upset with the FDA trying to take everything away, but, anyway, we're we're fighting to save some peptides. You definitely are fighting the good fight.

And the FDA, you know, has has certainly been, been, you know, trying to regulate things a little bit more tightly, but, Now you've, you've, you've moved along into also doing this.

So obviously the genesis of this, this similar clinical trial, in your, in your office, I mean, it's an exciting field. There's a whole bunch of things out there.

The, the the hallmarks of aging, talking about cell senescence, telomere length attrition and area. I'm very interested in, but, you know, as a busy clinician to, to do a trial with, with, with these, medications, it's, it's pretty accessible.

One to supplement. The other is a medication. But tell us a little about the thought process and the experiences that got you to there. Yeah. So I read, Doctor David Sinclair's book.

On basically, longevity. And, I really got hope. I actually read the book twice, and, it's interesting. I got more hooked on. He was talking about synthetics.

I did a lot of research on that, and I. And it's interesting, there are some people who just basically follow his protocol and, has done basically, like a, a biological age test, looking at epigenetics.

But I was I wasn't really interested in that. I was really interested in the whole idea of senescence as we age. There's a tipping point where, we accumulate two of these called zombie cells with senescent cells.

And then from there, it just falls apart. And I was thinking, you know, why don't we do a, you know, small trial to see if this works? I reached out to Ryan, and, we, Ryan Smith from a true diagnostic.

And, he basically does basically, biological age. I said, let's see if we can get our biological age younger. Well, the, about 20, 21 patients in the trial, and, well, so marker will measure some markers for senescence, and we'll also look at our immune cells and we'll do some other, you know, blood tests to see where we're at.

And, so the test actually, all these tests was like evolving because we weren't quite sure. There's no perfect marker for senescence. It's it's a growing field that's.

And every month there's another marker. And if you look at basically, you know, every part of senescence changes and they release different, cytokines, a marker.

So it's let's, let's, let's step back for a second, just for maybe the potentially more naive, not naive, less experienced listeners. What defines a senescence?

Senescent cells is very simple. It's a definition where the cell stops, replicating. It just comes to a point like end of the line.

Launching an Age Management Practice

And at that point, it's supposed to go to autophagy. Our immune system gets rid of it, but it refuses to die. It's one like that apple that says, I'm not going to go and it turns on all these mechanisms to basically, to prevent from dying.

So we have three types of senescent cells. Number one is embryonic for organ, formation. I wouldn't be on that now. And number two is acute senescence, which we need for wound healing.

And number three is chronic senescence. So if you have too much senescence, what happens? Is it, it starts causing this release of toxic material, and it can affect your normal cells, and they can actually go to basically you have telomere shortening.

You can actually cause tumor cells, and you can basically, speed up the aging process that way. So if you have too much senescent cells, I call zombie cells for my patient in the brain that can lead to Alzheimer's, Parkinson's or generative diseases.

That goes for long. You can have basically pulmonary fibrosis, or you can have plus you can have many different types of condition in the heart. You can have heart failure, a heart disease, you can cause cancer.

So everything that really, Doctor David Sinclair from Harvard, he really put it beautifully that we should think of. Aging is a mother of all diseases. Yes.

Which is a paradigm shift, because we think that agents are inevitable. But if you understand why we age, address that, we can actually make a big difference.

And, there's three clinical trials that I know of in humans, that basically looked at dasatinib and quercetin and, so I figured, you know, let's, let's see if I can duplicate this.

A little bigger trial, six months study. And I'm really excited about it. So, if, if you're. Well, I can tell you about. Yeah. No. So that's that's the study.

That's the general hypothesis, which is if you, you know, target senescent cells or a fundamental aging process, then you can get, improvements in multiple age related diseases rather than targeting an actual single pathway.

I think they liken it to sort of going after the infectious disease rather than trying to cure the organ. That's, that's, that's a disease. And, and that's what the team trial is trying to do with metformin.

And, you know, we're looking at that. That's why, you know, sort of, decreasing, upregulated Ampk. And in this case with analytics, you know, we want to get rid of them, these cells that cause the all the nasty inflammatory markers like IL six and, you know, Alpha, IO1 alpha, etc..

So those, you know, so there's a pluses and minuses to those drugs. I mean the certain is a is used for CML. It's it's an anti apoptotic inhibitor. It tries to get rid of those, those pathways.

So I take it you had, how did you choose the, the subjects? And what was, what was the what was the what was

the primary endpoint? And that's a weak protocol for us.

So my protocol was that, we first had it, we went through the IRB to get it approved to make sure that this is safe.

Senescence and the Rationale for the Trial

And, so we had an IRB approved, patients. Basically, I sent an email out, say, hey, if you're interested, contact, contact me if you're willing to enroll in this, study.

There was a fee for the study because a lot of these labs, I couldn't pay for all the labs available pockets. So, most of my patients paid about $1,500 just to be in the study.

And, so I have some people that, want to be able to study, but they had the same was diagnosed with cancer, so we excluded them. And, anyone with basically if like, I don't have many people with congestive heart failure or chronic, really bad, medical conditions, but, generally people with active heart disease, recent stroke or people with active cancer, you know, unfortunately can't give a study.

So, we're we're looking for people generally over 60 years old. And my oldest one was 87. And, so we, we collected 21 patients in a study and we checked, their baseline, chemistry profiles, CBC.

We also looked at CRP, IGF one, and we did a see basically, lymphocyte panel looking at CD4, CD8 and other parts of the immune system, which, was also an expensive part of the test.

We also looked at, your epigenetics, and we were looking at senescence. So we, we were going to do, basically the beta galactose, galactose, test, but, beta gal, but we decided at the last minute to switch to activate, and also we're going to look at a plastic linden level.

But apparently that company has never done this test. So it was like that would be a problem to do it. But we did the active in a which has been shown that if you have, senescence, it's elevated.

And so there's no reported studies that shows that if you reduce senescence you lower activation. So it's one of the cytokines that's released, with senescence.

And what I was saying before, there is no universal marker for senescence. You know, you have IE6 and you have all these other cytokines, but, it's one of those things that senescence is very complicated, and every cell does it a little differently.

And so universal pathway for senescence. There's an I and K for a but that's you know, that's a little tough to do. It's sort of a biopsy a thing to do.

And that's not universally agreed upon either. You know, this the speed of galaxies. There are a couple of available commercially, or at least one that I know of.

And there's some question about, you know, whether or not those are those are, but, in the papers that you mentioned, you know, the justice paper and the Hixson paper where they, they did the, the D plus Q protocols, they did measure those things.

And so I guess there are assays for that. But, you were looking to see if there was an overall change in, you also you had safety markers and then you also were looking, I guess, for an overall change in gene expression, which would be picked up by the DNA methylation, which, which clocks where you're looking at with, if you looked at all the clocks, so should I not say it's a, the Horvath, the groom, basically the the needed, that Dunedin probe that's from New Zealand.

So there's a clock from Yale. So they, they looked at all the clocks. So, we can tell you that, three people, dropped out of the study. But they all did the blood test.

They did a baseline three months and six months. The one person that dropped out, she basically, after his three month blood test, he drove from Miami to Orlando to do this study.

He was really into, during the study, but he says, look, I just got diagnosed with prostate cancer. And I didn't know his PSA was, like, in the one hundreds.

And he goes, I'm getting worked up. And he goes, can I be in the study? I go, it's unfortunately, your that's part of the exclusion. You have to be, but can you come back in six months?

I'm just curious. And he goes, yes, for something, I'll do that. And then I had two patients that basically had more of an anxiety issue. They didn't want to be on desk.

And to the they kind of got worried about it. They one had a little big headache, but nothing serious. But they went around saying, okay, but can you come back as six months to do your blood test?

And we just want to see. So we have three people that dropped out. So we were down to from 21 to 18 patients. And in general, everyone improved in terms of reducing the activity levels more at six months and up at three months.

So and we used the lower dose than most people because in their study there. But a lot of people use dasatinib 100mg and quercetin 1000. We I use dasatinib 50mg with quercetin 500mg so that was all provided for them.

And the reason I chose 50mg is that if you're on a P450 inhibitor, you can have side effects. So I don't know who's on verapamil, who's on a rhythm, myosin who's going to get a C pack.

So I said let's just be safe. The 50mg. Just go just go a little low and you don't need much of the basically set a letter to get rid of senescence. So you just have to get a low dose and it can do well.

So that's why we chose 50mg. So to them that quercetin 506 months. What's interesting is the science. We do say so now since we have improvement in the biological age, how did how did you show that you reduce senescence?

Reduce, active in a minute. Sorry. Yeah, yeah. But did you also, you know, I was thinking of the the the, immune subsets in that. Did you look at, any, like, Cd28 negatives or did you use the ones that we didn't in Cd28?

We just, cd4 CD8 couple other things, but I don't think Cd28 was in that lymphocyte panel. And, but all that data is going to, this bio, statistician, I think she's, basically at Yale's is going to look at it.

So I don't know what the outcome is. It's a lot of data to look. Okay. So just, you're you've been living it. So, just to mention you were doing three days of her month.

Must you put them off for six months? So. Yeah.

Trial Design, Eligibility, and Biomarkers

So, some of my patients I know, even my staff initially got confused because they thought, like, three Mondays in a month like this. No, no, no, like January 1st, January 2nd, January 3rd for first, second, third.

So three days in a row. But somehow they interpret like three Mondays around, three days in a row. Yeah. These are the headaches that you run into. That that's, that's, that's that's very interesting.

And it was very well tolerated. They were mostly people with mild headaches. And we were just taking a night time. And, generally some people say, you know what?

Didn't get a headache to day three? The headache was really mild. I took it, my wife took it. We had no complications of or side effects of headaches or any GI issue, but, we just had two people that didn't like the idea

that they were on a, tyrosine kinase inhibitor.

That's such a. And I really enforce you have to take both together. You can't just like take this and then take quercetin or later you have to take in combination.

Yeah. And so you do explain why that is. Because the way the which they both the whole pathway of basically getting rid of senescence and they have this resistance to die and they basically have this, they have these proteins that they're releasing and all these chemicals to prevent, basically, plateaus.

And so if you can get a target kinase inhibitor in there and then basically quercetin also in combination does the Bre, I think BCL two bcr PCL two pathway, reduce basically the hypoxic tube pathway.

So there's all these different pathways and combination works beautiful because all the cell lytic said worked at one single pathway. It never really works.

You've got to do a combo of this for it to work. And that's you know all these there's biotech companies are spending millions or even billions of dollars to see what the magic bullet is.

They're really going for like, one single pathway. And unfortunately, they don't see good results. Right. Yeah. I mean, James Kirkland's group, has talked about that where, you know, you want to to not look for that single, that single bullet.

And that's why, you know, when you want to go after the cell, not the pathway, but, because that's what you're really trying to get rid of. You know, in the, in the justice study looking at pulmonary fibrosis patients, they, they got a lot of them felt better from a frailty stamp, but they're better functionally.

Did your patients notice anything in terms of, you know, how they felt energy wise or, you know, I don't know, what were they like in general? They overweight in general or what?

So most of my patients were really relatively healthy. They've been with me for ten plus years. That the their hormones are balance. They do a liver cleanse.

They're generally very healthy. They don't overuse alcohol. None of them actually smokes. But two patients are really interesting. They have very low activity levels to do.

So they didn't actually it didn't change. I mean, they were like like my number initially was like 0.54 activated a, and then it went down 2.5, 4.5 to A three months and then 24 to six months.

So the, the six months we see a big drop in basically reducing reduction in senescence. Those people, the two people, two women, it's hard to know who has a lot of senescence.

You just assume that everyone up to 60 has it. But I think people 30 that, you know, are very sick. They come in to see you and they have multiple system disorders.

It's like, wow, you probably have tons of senescence. Actually, there's no good marker. This is the marker active. It is for research only. It's not you can order available.

Right? Yeah. The, it'd be interesting to see what when you get your clock data back, whether people who are biologically younger by the, you know, various clocks, you know, either the intrinsic, the accent exchange the clock or the groom age or also have lower activity levels, whether they correlate.

Well, I mean, it's so small. And so you might not see, see it there, but what's the typical range for the active in. You said 0.5 for help. I mean how high up does it go.

What was your highest activity level? Just looking briefly at, if I recall, if the highest was like point seven or 0.0.

Okay. So that's a, that's a significantly kind of like one to like to like, like point one to like, I don't think anyone hit one, but like, you know, point eight or somewhere that so just realize that that's kind of the range there.

But, yeah, that test was done in Texas and it wasn't. I mean, we we basically had baseline three months, six months. I had a personally late my son and I personally labeled each one and, put it on dry ice.

Had I had a manifest, emailed everyone and it was literally they were all waiting by Fedex and to get it. And because that's a I don't have any backup.

This is for my, well, that's that's a risk of any degree. We had a store -80 degrees, right there. That was that called. So. So that's the first clinical trial you've done other you did us you did a CMT trial as well.

Right? In the past, I think. Yeah. Near 40. Showing that six months taking basically nitric oxide supplement called near 40 can reduce plaque both sides, because actually in everyone by at least 10% or more, after doing six months.

Yeah. That's that's so you've been involved with, you know, adding to the medical literature. You know, I also did a recent study came out first published, published study on the peptide BPC, knee pain.

So I did a is a retrospective study. It's an intra articular injection with BPC in multiple types of knee pain, published this year. If you Google does BPC intra articular knee pain, you'll see my paper.

But if you go to PubMed and type my name on it, well that's interesting. So I can find why you can't find the. So you had, significant, beneficial effect on knee pain.

You were just you were injecting the BPC in your patients. Yeah. So this is a, peptide called body protection compound 157, and I'm not I'm not quite sure how many of your, people watching actually know what this peptide is, but, I think I call it a healing peptide, and it's truly amazing.

Results, Tolerability, and Biological Age

It's one of my top five peptides. I love and, I've seen people who've been told you need a total shoulder replacement, and I've injected peptides into their shoulder every month with also some guidance up to five months where they could not even put deodorant underneath their arm because they have three, like a labrum tear supraspinatus tear, infraspinatus tear.

Not complete, but just a significant tear where they they have no range of motion. Now they have full range of motion where they're basically back to normal.

So it's truly amazing how these peptides can repair. Well, this peptide can basically, repair tendons, torn ligament problems with, muscle and also nerve damage.

So it's truly amazing. I remember my son, he was in ninth grade, and that was in college, but, he injured itself during cross-country. You couldn't even bend his nickname a walk.

He had iliotibial band. Really? Just sore. And I said, look, I can inject a peptide. And he was in ninth grade. He goes, you are f injection. So I had to give like a five minute PowerPoint presentation out of is pure science.

And then he goes, okay, you're going to die. So my kids are like so tired of me talking about peptides. They just say blah, blah, blah. Pepto-Bismol. Cause I guess I talk about Achilles every day, right?

Right. So, so in this study we used, I used, I use BPC, inter articular I the dose varied from 2 to 4mg. I used to do, basically PRP and then peptide.

So improvement that I just said, you know what? I don't drop PRP. I just want to see this works. And I was mixing

tb4 BPC, and then eventually I just said, forget tb4, I just do BPC.

And out of 16 patients, 14 out of 16, had significant improvement. It was a subjective retrospective study. And I had a medical student, a pre-med student who wanted to publish, and he was I knew his father is an orthopedic surgeon, and this kid would not say he just wanted to publish.

He looks nuts. And he went to my office until we had a study going. So I was like, okay, call these people. We did, and maybe we'll work, but gather some data and write a paper together.

Great. That's good. I mean, you know, there's collect and sell you advanced clinical medicine by, you know, putting together these kinds of things. It's fantastic that you carve out the time, which I know is not not that easy to do.

So. Yeah. So let me mention I have one patient that, has been on to 65, for, he said in the beginning when to science started. I don't know when to science started 2007.

All right. Because he had a telomere length of 5.7. And he, he emailed me, that that was his lymphocyte 5.7. And then he's been, to 65 big believer always been taking that.

And, what's interesting is, he has I saw his biological age, is 87 years old. I guess what his biological age is younger, I would imagine. Yes, younger, but his 87.

So would you say he's 80 or. It it depends. You know which assay was used? Well, the the biological age was with two diagnostics. So it's an epigenetic test. Oh, okay. Okay.

Oh I see, so you're saying he's 65? His biological age is younger. I mean, wow, I mean, he also did a study that that's the the course. Oh, he did, okay.

But you don't have his the clock back on him yet on that after that, I have this biological age. I baseline three months and six months. Oh, wow. Biological age, has improved a lot.

Just a little smidgen. But his biological age is solid at 60 years old. Wow. That's impressive. And he loves fast food. A little off weight. It doesn't slow, but he loves to 65.

He even gave it to his dogs when they were alive. Yeah, I've heard of people doing that. Which would not be unreasonable. I mean, you know, there's a whole dog trial going on with rapamycin.

It's like three years into it, and they're seeing some pretty spectacular results. So, you know, dogs are age somewhat similarly to humans, so that that's a very reasonable thing to do.

That's a pretty impressive difference in biological age, I, I don't I don't see people, you know, too much younger than than, you know. Yeah. It's incredible.

I mean, I asked him what he thought and he said, 40s, I'll go. Okay. I don't know if you can see this, but here's, telomere length through, true diagnostic.

Remember his baseline, telomere length was 5.7 in 2000 and 7 or 2017, 2007. And then and, just recently, his, average telomere length was 6.8. So but there are two different assays.

And how can you tell. Yeah, he did two, one, two of them from wife length. And in 20 1617 they were average. 8.2 and 8.9. So different years. But anyway, they're, they're, they're just different measurements.

So I don't know how you can compare, you know. Well it's not yeah. It's particularly hard to compare a life length to the one he did in 2007. His life wasn't around back then.

It probably might be diagnostics. And, life length is about a number of killer bases higher. And then, you know, the I

say that the two diagnostics does is a, is a, is a deconvolution assay where they're predicting the liver, like, not actually measuring it.

They're relatively accurate. I mean, I see them because I have most of my patients getting to their legs. And I see that they're, you know, they're they're not far off on their assay.

So that's, that's still so pretty good. Yeah. And so you use to 65 in your practice, pretty pretty regularly, you see. What do you see with that?

Peptide Therapies and Clinical Case Examples

So I have devoted. Yeah, I, some people have been on it for ten plus years and, you know, they love it, but they also like the new stuff. The analytics.

Yeah. It's hard to tell to tease out the difference. Yeah. So when do you, did did Ryan say when he expects the results to come out of, we'll be fully done for this.

I want to know Microsoft's. You know, I don't know. We have the six months, and, anyway, I saw him six months, but I don't even have baseline, so I'm.

It's driving me crazy. So I'm the last one. Well, we we have 20 of them done for me, so. Oh, well, I think he's doing a cruel joke like that has been very busy, that's for sure.

Have you, have you, have you personally noticed anything since, being on it? Any better, positive or negative? I thought I had a little better in terms of cognition.

A little clearer, thank you. I actually stopped for about three months, and then I did another, epigenetic test should I lost, and then, wanted to see if my biologic age was sustained or if it went back up and, restarted.

That's the question. I'm going to call back all my patients in the study to see if they wanted to do another just twist on this, study. And that is repeat the dust and quercetin, but perhaps do something to increase energy.

We can measure energy levels and see if we can in turn on turn on two and one genes and see if the telomere length get even longer. That way. What are you, what do you what are you what are you using to measure energy these days?

You could do it. Intracellular energy levels through a company in Georgia called to infinity. Doctor. And, he he looks at all your blood, red blood cells, white blood cells, all the white blood cells.

And, it's a anyway, it takes about 2 or 3 days to see what it is. That's so good. So a good number to remember is it's like, optimal vitamin D level, like between 60 to 90 would be a good energy level.

I think we both were a doctor. She's lecture. That's, in Dallas. Yeah, that's. That is a good pearl to know that, if you're if you're 20 or below, which is what people over 50, 60 are going to be, that's the vitamin D deficiency.

And you want to be up in that 5060 range? Yeah. I'm going to start ordering that assay. I think, to see whether or not, because I've got a lot of patients on and they'd precursors to see whether or not they're, improving too much is not good either, because, I think one person who has, insulin resistance and he exercise it's really clean drives, has tried some crazy while his, fasting glucose is always 100 is A1, CS borderline 5657.

And I said I don't know what your energy level is. Maybe it's too high. And, if you're over 100, you know, we can cause insulin resistance. So, so he never gotten his energy level.

Yet the other guy, he's in Georgia, I thought I just talked to him today. So we're going to tell the kids all about his results. So he goes, yeah, let's check.

Yeah. Sounds good. So, it's great that you're able to combine the some clinical, research along with your with your practice. And, I'm really looking forward to getting, getting hearing back on this data.

It's kind of a, you know, you will be the the fourth clinical trial if it was, you know, published, I'm sure we'll get published, but, on on this, unless there's other ones that are cooking right now that we don't know about, but, it's it's a really interesting paradigm.

You know, I think that probably combining, telomerase activator, with this or in cereal with it because there's a, there's a fear that Michael Fossil and some other people like follows in fossils.

A telomere biologist and, and clinician is that if you take out senescent cells then you have to be they have to be replaced by other cells. And then that will cause senescence in their society.

Members have to, you know, get shorter as a divide to replace those cells. But if you can do that, then perhaps you have a very good sort of, approach with, you know, sort of just like the asset class in the osteoblasts.

Do you go and then you get rid of the bone and then you got to go. Somebody go in there, put bone back in. You know, and and that may be an interesting paradigm to test.

Got some. Got me thinking about this. Now, I might add to things I do up here. But I'll wait to see what your results are. That'll be that'll be interesting to see.

But look, it's been really interesting to to hear about what you're doing. Is there any other stuff on the, on the horizon for you that you want to tell us about and or any pearls that you have for, you're now an old hand in this field just as I am.

You know, we're, So, really been impressed with epithelial and, and this is a peptide, actually, from the pineal gland. Doctor travels in from Russia.

Basically published a lot of paper with his colleagues. He had a paper that 15 year review on, on using epithelial in humans and basically showed a reduction in cancer, people with heart failure, heart disease live longer because they looked at people with coronary artery disease.

And, so it was a three year use of epithelium. They basically 50mg, not all at once, but just in divided doses. And then basically repeated in six months.

So they did 50mg twice a year, and for three years. So I had to call doctor Kabat-Zinn somehow I had, doctor from Russia. She wanted to shadow me and learn about peptides.

And one Monday morning I come into the office and this beautiful blond doctor, it's like in my office, about six months or so. Russian doctor. I'm like, this is like kind of it spills or what's up anyway?

So she she she goes, look, I just want to learn about peptides. And later I said, can you hook me up to Doctor Tabassum Saint Petersburg? And I really she, she did that and I talked to him and I said, can you use it more than three years?

And he goes, yeah, my mother is over 90. She lives in Chicago. And she didn't use it for 30 years, so it's 50mg twice a year. Is that the dose? The 35mg.

And you basically can do a subcu shot or I am shot for like, ten doses. Just to make it 50mg. And then you repeat it and then basically, six months. So.

And so what is epithelial for? Look, Italian is a for me. No acid peptide from the pineal gland that actually controls cell, cycle. And apparently, we had a speaker on clinical peptides.

Society is really into, into outside of human medicine. He actually looks at, just a bigger picture and he says it's estimated that about 450 million years liquid has been around in reptiles and amphibians because they control their cell cycle.

So if our cells are programed to only go so long, and then if you give epithelial actually basically turns on the cells to go a little longer, well, ever.

So he says, basically for fish and frogs, amphibians, they all have AP power. And so anyway, he's, I never can verify it in their, you know, paper, how long everyone has been around, but I guess he did some research.

An estimated about 450 million years. But one of my patients, I've had a lot of people with macular degeneration using epithelium. And it's a degenerative disease.

That does mean better. And I have some people, their eye doctors are, like, truly blown away, like, what are you doing? Why is yellow and you're doing the same?

This is five milligrams for ten days and then six months. Yeah. The file that we get we use is to go from TaylorMade. So you could have 50mg I get it now from Brooksville.

Or I think it's from Brooksville. And it comes only we only can do 45mg this cost effective. So within 45mg twice a year. In divided know you're saying you don't do it all at once, took you or you do it all. Okay.

Five milligrams nine. The shot every other day. We do it every other day. And then, so I say it's going to be over a ten day cycle. And pretty simple to do.

And, actually, one of my I'll tell you two stories. One is, my patient with Parkinson's for bad Parkinson's, and I used to do ancient people, stem cells, exosome therapy.

I've given tons of, like, I. V. and, it really didn't make a big difference until. I mean, I use peptides, like, I'll have a zoom on him. And then I said, look, I want to do of on you.

And he goes, okay. And he goes, it wasn't until you saw the cinematics with that one, because that has made a big difference. And also the other things we're doing.

But he really a his wife goes, his gait is flowing. You can see it's speech used to be slow. It's rapid. It I remember the way he used to speak. He still has a little tremors.

But it's it's he is taking one medicine to control that, and I think that's temporary, but, anyway, I think he has mitochondrial disorder, so I'm using some other mitochondrial peptides to help with that.

Because if you permit mitochondrial disorder and Parkinson's, you'll see like 60,000 articles on that. So the thing is that, yeah, if I can get mitochondria to work better, reduce senescence, and also to basically help with peptides and increase his telomeres, I think it'll be a great way for him to get better.

Epithelial Peptide and Future Directions

So that's that's really exciting. And number two is I've had, you know, even though I'm in the chronologies, I really don't see many diabetics these days.

But I've had a type two diabetic who I had to put on insulin, and he's just continued to gain weight. And it's just like he's had problems with the vision.

Blind spot on the right. Chronic kidney failure, his AP phthalate. And what's amazing is his got off insulin and he goes, I just wanted to tell you, I'm Italian, and I have not touched a bowl of pasta for, like, three years.

And finally, my blood sugars were going so low, I got to stop all my insulin, and he goes, I did an experiment. Check my blood sugar before and one hour after eating passed, my blood sugar was under 100.

And he goes, do you think everyone could help? And what's interesting, it's the pineal gland and the retina. And I think the pancreas is all close together and in the embryo and organ.

So I think there's crosstalk receptors there. So I think for the endocrine function, this is really exciting because the one person that talked, I come back to the society, he's had very he's had kids teenager with chronic pancreatitis that has failed everything and somehow goes all the way to like Wyoming, small little city, Jackson Hole, Wyoming to find this guy.

And he goes seven out of eight, kids who have pancreatic insufficiency with elevated enzymes. I can't flush stool down the toilet. They failure to grow can't absorb anything.

Epithelial is basically, sniffling through their lives. Their pampered parent has got back to normal. I've seen this guy, his kidney function. Not normal to, and, his eyes, he goes, I to the eye doctor.

Every six weeks, I get an injection. I know what I can see. I sit there in the waiting room for an hour, wait for the eye doctor. Come in. You can see, you know, my eye exams.

And you can see over time, my eye is getting better because my right blind spot is gone out and my left eye is now 2020. And so the issue is retinal issues.

I it doesn't really help with cataracts or the front part. And the back of the eye is really interesting. So I like to do like, I have a friend that has read my wife has read Minds Pigmentosa.

They have like, eight kids and four of them have the have the gene. And, you know, they're just like, I would love to do it on them because in Russia they did published a paper.

So, you know, I actually had Tom Dalen on the, I interviewed him earlier and he's an ophthalmologist. He published a paper with Cal Harley on 65 and, markers for early macular degeneration, showing that taking to 65, reduced the marker on the eye, test.

And, you know, he explained the retinal, epithelial pigment is very, highly, you know, turns over very highly metabolically active and telomeres get short there.

So telomerase activation could well be something that is, you know, can can help it. And, you know, there is, as you said, some potential evidence that epithelial can like, telomeres.

Maybe that's what's happening with it. So it's certainly a possibility. Be interesting to see. And you got to be real interested in epithelial. I'm, I'm going to have to, investigate that a little bit, a little bit further.

My problem was I just the papers are all from a long time ago, and, and, and I haven't seen anything since then. And they stopped publishing anything recently on epithelial, and they're actually, I guess they have nothing else to publish.

So they've done all the research, so they moved on. Still, they had to go. But in America, this is all new. So those who like yeah I know Russia's like, oh, we've done that in 1990s.

It's a very interesting. Well, again, I mean, thank you very much for sharing, your exciting results from your study, your, your all your clinical insight and the case studies are really fascinating.

There's certainly things I want I want to look into. It's great the work you're doing down there and taking the time to, to add to the the body of knowledge that we have to treat our patients.

You know, better. I think it's fantastic. And, I assume you're going to probably. I'll see you out in Las Vegas soon. Yeah, I, I'm I'm, I'm debating, about this one, but, anyway, I'll see you in one of those conferences.

Okay. Whether it's in April or in Vegas, but, in Florida or Vegas, but, and you, you can tell anybody that's want to how they can get in touch with you if they want to, send patients to you or anything like that, just, patient wise, I am.

I'm slammed. I'm okay. I don't know if you're just, but I am, I am, I we I've had people like we had a turndown. This woman from Italy, one that's come to see me.

I don't know how she heard about me, but, it's like, I'd love to help you, but, I'm only one. I have a nurse practitioner, and I think the next available is, like, seven months.

Yeah, I, I, I mean, it's great. We have a it's a good problem to have. I need a young doctor. I can train. So if you want to, have him call me. I love to train them.

And they can help me in my practice. Sounds great. Well, sounds like that decision in 2008 was, actually a really probably one of the best decisions for you.

You really, you really helped me make that decision, so. Thank you. Well, you're welcome, and I'm glad to see you made really good use of it. All right. Thank you again. Thank you.

Can I just.