The Three Levers of Longevity: Maintenance, Rebirth, and Cellular Death Transcript

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Opening and podcast introduction

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Even fungi have like 20-something base pairs. living things that are not viruses or simple RNA things. They have chromosomes and they all have to use telomeres.

So that's why we know it's something fundamental, like you said, to protect the tips of the chromosome. Welcome to My MD Unscripted, where healthcare gets personal and the script gets tossed.

I'm Dr. Clint Carter, ER doctor turned direct primary care freedom fighter. We're on a mission to challenge how our healthcare system treats patients in America.

In every episode, my expert guest and I dive into real conversations about what's broken and how we can fix it by making healthcare about people, not just managing disease.

It's time to rethink what healthcare should be and how we can make it better together. Welcome to the podcast. Hello and welcome back to My MD Unscripted.

I'm your host, Dr. Clint Carter, and I'm honored today to have as a guest Dr. Edward Park, who is a Harvard trained physician that spent nearly two decades studying the biology of aging with focuses outside of where you might expect with talking about telomeres and cell-derived exosomes was taking care of his father and we'll get more into that.

And then he started asking the deeper question, you know, why does the body lose its ability to maintain and repair itself in the first place?

Dr. Parku2019s origin story and telomere theory

And once we know that, is there anything we can do about it? So Dr. Part, thanks for coming on with us and give yourself a proper introduction and then we'll figure out how we got where we're at right now.

Yeah, I mean, I typically like you did the medical training thing and then you become a cog in a giant machine and because of the way that a lot of people practice medicine, it becomes sort of a cookbook.

And I realized, you know, at the time, 20 something years ago when my dad got brain cancer, it was just a cookbook. And he, as a physician, knew trial, this surgery, that chemo, and then eventually a slow agonizing death.

Last year of his life was just, you know, bedridden, unable to communicate. So he saw the light at the end of the tunnel and knew it was a train and he still kept walking.

So at that time I thought, well, why do people get sick? And I came up You know, it's like Dr. Google at that time. I was like, oh, T-limiters shortened, that's the best theory.

Because they would have these stupid theories like, well, social engagement. Because you are less social, that's why you get old. That didn't make any sense.

So they did something that was scientifically robust, that had animal analogies and models. And then gradually

over the years, you know, the first book I wrote, oh, 14 years ago, I don't know, explain my theory.

At that time, I don't even agree with my theory currently, I think that. Well, because, you know, at the time, like many people, I thought stem cells worked by going and honing and engrafting and changing.

And there may be some cells like mu cells that do that, but that's not the predominant method. The predominant method, we just found out, like, not even 20 years ago, was they released these tiny bubbles.

called exosomes, secrete exosomes. And the way, you know, like this gal I was talking to fell off a scooter. She had been drinking and she broke her wrist.

And I told her, well, when you get inflamed, the stem cells come and they release the exosomes. So you don't really need someone else's cells. All you need is the exosomes and that kind of reprograms what's going on.

Yeah, but the overall arching theory that occurs in all three books, this is the second book that I wrote for Hay House, it just talks about how without any money you can do lifestyle hacks, stuff that's common sense.

Sleep better, eat better, exercise. So that'll cost you nothing. And it's backed up by some, you know, science, kind of soft science, but they study like meditators, non-meditators, good sleepers.

Yeah. Yeah, that's where I'm at. So I have this theory, like you said, you know, aging can be hacked somewhat, but it's inevitable. You know, it's like you look at the lint trap every time you do a drying load.

There's something in the lint trap, right? It's not zero loss. You're always losing something, but there's a lot people can do to accelerate their aging.

and things that they can do to slow. And, you know, I went to Costa Rica this summer with my son and they gave ElectronX the cells to them and they have a clinic where they can extract and store your stem cells.

He may go there at 26 next year at 27 and store his stem cells because he's never going to be any better than he is at seven. Right. Yeah. I wish I'd, well, first of all, I wish I'd have done sort of hair restoration when I was 26, but also I wish I had thrown some stem cells into a cryo chamber.

That would have been unbelievable. And so, okay. So let's, let's talk this thing through. Let's, let's. go through the journey with you. So it was 2004-ish that you and you were taking care of your father.

He was going through brain cancer. And then, you know, telomeres were sort of the first, cause you still hear about that and it's not like it's gone away per se.

So tell me a little bit more about how the telomere theory works. What are telomeres? Why do they matter? How might they help us? Sure. Sure. So the first book was called Thielner Time Bombs.

So basically this is not any kind of fringe theory. Every time a cell, a mother divides into two daughters, every chromosome, all 23 pairs has to be copied.

But because of the way that the mechanical reproduction works, the leader or the primer can't go right to the very tip end, so you lose about 50 to 100 base pairs every time a cell divides.

So over the course of time, it's called the Hayflick limit. Cells that are not telomerase positive will die off. That's called the Hayflick limit. So they got some chicken heart that was living forever.

It's because it was a stem cell. So what is a stem cell? A stem cell is two things. It's a cell that divides asymmetrically, meaning a mother and then a mother and a daughter.

asymmetric division like she stayed perfect mother but the defining characteristic is telomerase. So telomerase is

an enzyme that can be turned on transiently to just reprint more of the ends of the chromosome.

It's just a six base pair nonsense segment like in the old cassette tapes. I don't know if you're as old as I am, but yeah, blank tape leader.

Stem cells, exosomes, and cellular communication

Yeah. Right. That's all. So the theory is quite simple. I don't even think it's a theory. It's just a fact. And a hundred years now, they'll just say, well, this is what causes aging.

So your stem cells, they don't always have telomeres on. Otherwise the telomeres would grow. It definitely that'd be inefficient. Yeah. Right. get the intermittent activation of telomerase.

And that's why at 58, I don't have gray hair on top or reading glasses. So I think it slowed my aging over 18 years. I've taken it for 18 years straight.

So you're taking things to protect your telomeres that are basically just spaceholders at the end of your DNA strands. Because if you didn't have them, eventually you would start in the replication process when a cell replicates.

Yeah. I mean, all differentiated. Yeah, differentiated cells, like, you know, they don't copy a lot, but they do copy. So that's why you get old. So every time the cell divides, it shortens.

And if it gets critically short, then what happens if you're interested is that the chromosomes get stuck together because the cell repair machine ends.

So it splices it to another location. Yeah. And then that doesn't work. Well, the next time they divide, you'll tear apart the chromosomes and that's where the mutation and the death comes from.

Well, and so, you know, it's a built-in limiter to life to some extent, and stem cells are a little different. And so stem cells were sort of the next.

Um, sort of the next level of sort of anti-aging, you started getting into exosomes. I'm with you. I thought until like really recently, because I didn't look into it much as a physician, that a stem cell went to the heart and became a heart cell or went to whatever and became a liver cell.

That's not the case. That's not the case at all, which I think is hilarious because we use exosomes here for hair restoration and other things. And I didn't know what an exosome was.

I was like, well, yeah, but what is an exosome? And of course what we're having is plant-based. There's regulations and stuff for now. The thing that's really mind blowing is nobody knew about exosomes until 18 years ago.

They've had the electron microscope for almost a hundred years. So they saw these excretions of these little bubbles, but because they're, you know, invisible and because somebody once said that it's poop.

Yeah, everyone just believed it. But then when you start analyzing the bath water and finding these little bubbles and cracking them open, because that's an important step, they found out they contain protein, some mRNA, but mainly microRNA.

So my emerging theory, and I go to these big international conferences and I talk to the manufacturer that I've been using and he says, yeah, it's mainly microRNA because it's It's a very efficient hack.

So what microRNA is, you know, messenger RNA can be depending on how big the protein is. Right. That would be as big as the protein. Yeah, it's so big.

But microRNA, as you know, are 22, 23 base pairs. So it's really an inefficient way to block something. So why would that help? So like you said, in the heart or other organs, you have these stem cells that are not stem cells.

So the bigger question is, are all cells able to act stem-like? And I think the answer is yes. That's what the Kamayanaka factor showed us, that induced pluripotency is possible.

So there may not really be stem cells. You might be the temporary boss. Someone got fired or died and you're the temporary shift manager. And then that could be how it's working.

Like you can go to differentiate back to a queen bee, back to a regular bee. Anyway, the bottom line is that your stem cells, they don't do a perfect job of maintaining integrity, either genetically, the code, or the epigenetics, which is which genes are turned on and off.

So as we get older, all that kind of gets glitchy and entropy. I want to share this picture of trapeze artists. And that's exactly what happens is that the chromosomes will get stuck together if the telomeres are too short.

And then the next division, they'll be like trapeze artists ripped apart. They don't always come apart at the fingers. They come apart at the shoulders.

So if you look at something like the Gila cell, right, the cervical cancer cell, it's like this mutant. It's not 23 perfect pairs. It's a mutant. And that's the destiny of all stem cells.

If they don't maintain their telomeres. So the, and the telomeres are kind of, you know, we'll say God's way of, you know, protecting the DNA and, or, you know, putting a limit on how many times that stuff can, can replicate.

Yeah. But so that's the thing, like, it's like, if it's stem cells immortal, why aren't we immortal? Correct. It's not always on. If you talk about jellyfish that lives in the giant ocean.

It's always on. So the jellyfish can get really big and it's effectively immortal. So if you look at cross species telomerase activity, it very tightly correlates with how old that creature could be.

And it's true even in plants. Plants have seven base pairs, not six like animals, but even fungi have like 20 something base pairs. living things that are not viruses or simple RNA things.

They have chromosomes and they all have to use telomeres. So that's why we know it's something fundamental, like you said, to protect the tips of the chromosomes.

Yeah, okay. And so then you really started focusing on, when it became clear that stem cells don't differentiate, in, in, in turning to a different kind of cell in your body, but they do, as they release exosomes, they do things mostly in the reality, micro RNA, which really makes for micro proteins that really do things.

What's a nano vesicle. Exactly. And is that what you're talking about the exosomes? The term of art, you know, basically they're all. uh, extracellular vesicles.

So there are, there are less than 40 nanometers called extrameres. Uh, there's a cartoon you can find from ICEF, the International Society of Extracellular Vesicles.

There's many kinds of things, but The canonical one, meaning the one that everyone thinks is an exosome, comes from an intracellular pathway. In high school biology, we had the Golgi apparatus after the rough endoplasmic reticulum.

It's the final packaging area where they make the little bubbles. That's how COVID reproduces. That's how we make our exosomes. and they get packaged in this other vesicle of like maybe six to 20 little bubbles of exosomes and then they get extruded out.

So everyone's studying those, but I asked the question a couple years ago, what's the difference between the bigger vesicles that bleb off from the surface, and the tiny ones, we think of exosomes, that's roughly 50 to 170 nanometers.

And she goes, well, we do the analysis, they're kind of the same. But what we don't want is apoptotic bodies, because you can get blebs, which are just like hundreds of nanometers, even a thousand nanometers, or a micrometer, and it could be from a dead cell.

I mean, you maybe want that, maybe you don't want that. Nobody really knows. Like some exosomes, so typically, this is not your question, but typically the field has thought of what you see is what you get.

So if you look at those old micrographs of poop coming out, we think of those as the exosomes, right? But extracellular vesicles can be bigger and just come off the surface.

There are also a whole bunch of ways to make that work. Even I went to a great lecture by an ophthalmologist who's an MD-PhD, he said, The retina is the most metabolically active cell and retina can loan each other mitochondria through big vesicles, which is like fascinating.

That's wild. Bottom line is extracellular vesicles are cell communication. And yeah, plant exosomes, they affect us. Cow exosomes. If you look at the army fitness test, like people who drink unpasteurized milk are the fittest because they're fit as a cow, I guess.

They're strong. Yeah. Well, so a lot of our audience are patients, right? And so a lot of this has been sort of sailing over their head a little bit. So help me with what kind of patients have you seen improve with, say, exosome treatments?

I mean, I use it for some hair restoration, but not on me, obviously, it's a little too late. But what do you see it with? Well, I don't know about that.

I mean, you could still see some movement. It depends on if you have androgenic, scarring, alopecia.

Clinical uses of exosomes and regenerative treatments

Yeah, I mean, I can see some, but I have done some treatments where I became 20% less bald, which was just awkwardly bald, right? So I think in order to get hair like yours, I think I have enough scarred in old dead things that aren't alive anymore that it would be difficult.

But what kind of patients… Well, anything from head to toe. If you go to my YouTube channel, drpark65, And you'll find a short video, seven minute video, entitled Exosomes.

Are they a panacea from head to toe? So yeah, we do everything. In eight years since I included them in my practice, I was patient one. I met almost 700 patients, 1200 encounters, 3,300 treatments.

We'll do everything. Nasal is one of our favorite for facial and neurological stuff to get to the brain. And we can do IV. It just depends on the brand because some I wouldn't trust at all.

I get every week or two, I get an offer from a new manufacturer and a lot of them just fly by night and they're not in control of their quality. So anyway, so our low hanging fruit I would say is probably joint disease.

Yeah. Pretty pervasive, so knees, shoulders, hips. Do you inject it directly into the joint? We do, yeah. That's awesome. Yeah, and then we also do a lot of nerve damage.

A great example of how exomes work is they have this blocking of microRNA that can cause things to regenerate. Nerves can regenerate by these two microRNA that block the blockers of de-differentiation and they block the blockers of myelin production.

So a lot of what we consider adult cell behavior is just because the immature behavior is being inhibited. So for a short time, I think the exosomes in these cells act primitive and act like, you know, the boss or the queen bee and that's what's going on.

Well, that's beautiful. So the ability to put the stem, the exosomes in the area where they can do the most good and basically turn on. what the spell did when you were a child or…

I think that's how it works. That's just based on nothing but my intuition and some science. Well, I mean, but it's the same idea with, you know, epigenetics is the fact that all of your genes aren't turned on all the time.

And the ones that are turned on oftentimes due to stress and lifestyle and the American diet, et cetera, et cetera, are inflammatory and survivalist. And definitely not what we're talking about.

I'll give you a great story. In Hong Kong this summer, at the stem cell conference, ironically, these people don't even talk about exosomes. They'll admit that that's how stem cells work, but only 5% of the posters were about exosomes.

That's funny. Well, everyone's in their silo, but I want you a great presentation of this brilliant Chinese doctor who cured diabetes. So what does that mean?

Some people in the pancreas have an autoimmune reaction where they don't make insulin anymore and they can record- Right, type 1 diabetics. Yes. Yes. They're so brittle.

They can die any day. Every day is a struggle. So what he did was not even using the Yamanaka factors, he used chemical induced induction of IPS. So to your point, it's important the audience understands what IPS is.

Induced pluripotent stem cell. Nobel prize like 14 years ago or something. Basically the thing is any cell in your body can be turned into a stem cell and even a very early, early undifferentiated stem cell.

So now if you go to this conference, like they have vendors, they have sheets, one sheets, cookbooks. It's like how to be God, how to make a kidney cell, a liver cell, retinal cell.

They know every step, what chemicals to add and which switches to turn on and off. As we're alluding to basically the fate of a cell, its identity, what act it's playing as an actor, it's just a bunch of switches that were thrown on in sequence.

And you can erase that like a new pinball machine set up and that's it. You can go back to zero. And so the other interesting presentation was by a Japanese researcher on public opinion.

And they asked 3000 Japanese would they be, cause they can make induced pluripotent stem cells from any cell. So they can get, you know, skin from Pamela Anderson and Liam Neeson and get, you know, the young versions of them into a sperm and an egg without anybody ejaculating into a cup.

Nobody has to ovulate. They can make artificial sperm and egg. We're not artificial induced pluripotency from anyone. So I thought the meta question was, you know, the Japanese government is sick of having like Indonesians, Australians become Japanese citizens because they're very close society.

Right? Yeah. Right. I asked the question, Hey, would you guys be okay with companies like McDonald's, Walmart or countries? you know, doing the brave new world thing.

Cause Korea has the lowest birth rate in the world, but Japan's way down there too. So they're basically dying by the wealth trap of industrialized nations, you know.

Anyway, IPS, induced pluripotent cell, any cell can become a stem cell and then very early one. This guy, Dr. Dang Huy Kwing, he basically took cells from seven people and he did differentiate them too early and then did every

step to make pancreatic islet.

But using chemicals, and then he put those cells, not in the pancreas, but in the rectus sheath. And sure enough, those seven people are cured. And the beauty of that is instead of taking someone else's cell and de-differentiate, they'll still have the major histocompatibility.

So it'll be rejected like an organ. He took the seven people and took their cells, like skin cell or whatever, and he de-differentiated to a pluripotent cell and then made the islet cells.

And he didn't bother putting the pancreas, which is hard. He put it in the record key. These people are just cured. They're scooping Ben and Jerry's into their pie holes.

They're all good. It's crazy. So, gosh, that's a brave new world, man. That's a, that's a whole nother. Like you can breed people to be workers or artists or whatever, warriors.

And you can fix what's broken as it breaks or before in an individual's organ by organ. You can, this is. Super interesting. It's a little bit playing God, but in the end, I suspect there's a limit to our power.

Induced pluripotent stem cells and organ regeneration

But so far, this all sounds like a science fiction, I'm sure, to our listeners. Level three really to pull on wellness and longevity and in fighting aging is synolysis or the cell death.

So much of what happens, you know, is that the cells that should die don't. So tell me about, tell me about how that sort of has got you excited here as of late.

Um, you know, this is the problem. You know, you get somebody who's doing a really, really bad job, right? At your restaurant or your factory or your bank.

And the question is, do you have someone to replace him or her? Like, are they doing the best they can, even though they're phoning it in or it's a bad job?

So that's where the only real viable hack is banking. Because, you know, there are lateral central legs, like curcumin. There was one called FOXA4-DRI, which may work a lot for cancer, but, you know, I haven't tried it for that.

But there's all this, basically, it goes back to the telomere, the chromosome. So the major thing, it's not that complicated to understand, is that every time a cell is about to divide, There's something called a checkpoint.

So it basically says, Hey, do we have the right number of chromosomes? Are they paired up? If that's not right, like last division, the trapeze artists got messed up and there's, you know, too many or too few chromosomes.

Then the cell will activate something called P53 and P53 just initiates the cell suicide program. Right. But the, so I think, you know, this. The idea of stenolysis is not a very welcome one.

Unless and until you can bank. Like if my son goes to Costa Rica and he banks his stem cells from a bone marrow aspiration. Then, you know, a hundred years now he can get a vial out and then he can make whatever cells he might need through a process.

Cause the whole thing is it's got to be self-friendly, right? Cause of organ rejection, cell rejection. Right. Right. X-ray don't have that either from plants or animals because they're so small.

There's no proteins on the surface to tell them that you didn't make that. It's not compatible. So that's why they're so much better than like Wharton's jelly or, you know, all this stuff, these tissue products, they have self antigen.

So they're going to be rejected. In the meantime, they'll make exosomes, but why bother? Well, you know, and you say it's hard to fire that employee until you have someone better to replace them.

But as a business owner, I would say, and just bringing this analogy all the way around to health is like, if you have a cell. doing a bad enough job, you're really, to some extent, better off without it.

Now, if a significant percentage of your workers, let's say you're in heart failure, you don't just get rid of the heart, right? You've got to do it in a stepwise way, but ultimately you got to get rid of the cells that don't work so that you can replace them with the ones that do.

That's for sure. I'm just thinking in sort of a meta way, but yeah, there's something that you're alluding to called a secret sorry, senescence associated secretory phenotype, S-A-S-P.

So when a cell is damaged epigenetically, it's acting badly, it's inflamigenic. It's called, people call it glamo aging. So yeah, if you have a misbehaving cell, it certainly probably is better to target it and senolize it.

Yeah. But I don't know that that's really a thing. I think that's an anthropomorphization. There's many ways that cells can secrete inflammatory factors because they are in danger.

It's like the cell danger response, I think, is a… anthropomorphism, but let me put it to you this way. Okay, so let's say you kill all the old dysfunctional cells.

Do you have any niches or pluripotent stem cells to replenish them? Maybe not. So you may be accelerating the aging process in the absence of replacement, right?

So that's what I'm saying. The bank may not run. The Krispy Kreme may not make donuts anymore. And I think this is illustrated the best by a Dutch woman who died like six, eight years ago.

Her name was Heinrich Van Andelschlipper and they did a bone marrow biopsy or a blood test and it showed that her clonal variants of hematopoietic stem cells, meaning your T cells that fight infection and cancer, showing that two variants, which means, you know, you and I have like 10,000. Yeah.

The way that we can respond to new immunological threats. So that's just one lineage, like the bone marrow white cell producing lineage, but it was true in our eyes and in the liver or heart or kidneys.

My point is that it's not only mutation, it's depletion, right? Sometimes there's no lint in your lint trap because there's no, it's all your streets are threadbare, right?

There's nothing. Yeah. So she only had two remaining clonal variants at 114, which tells us that if we accelerate her senolysis, right. She might be left with nada, nothing.

Yeah. You know, it's like Jack LaLanne was held as a horse, but he got knocked off by a common cold in Palm Springs. So he's like, I'm macho Jack LaLanne and I can do finger pushups, but he had no immune system to fight whatever cold he had and he died.

That's, you know, it's. It's a balance, getting rid of the fellows that are doing harm, but they're also, how much good are they doing and how much harm are they doing?

Where is science in this field? Yeah, because that's where, you know, the way science, I realized going to two exosome conferences, one stem cell conference, scientists are very OCD, anal retentive.

They only care about paying their postdocs and their PhDs. Yeah. uh, candidates and it's just, they have a narrow focus, right? So they're not like that creative.

And so they don't see outside the box. I mean, the stuff I wrote about my STEM cell theory of aging, it's just going

to be like preposterous. It's like saying, well, an egg falls, then a bowling ball will fall.

Yeah, no shit. It's like the same thing, you know, but they're very slow to adopt change and new dogma. That's why for 80 years, they thought exosomes were cell poop, which they're not.

Yeah.

Senolysis, aging tradeoffs, and stem cell banking

So they're very slow to change, but the biggest questions, you know, I use the analogy like creation, like Brahma and Hinduism is like the stem cell, right?

And I think ordinary cells has been proven by IPS can become mother omnipotent stem cells, queen bees. And then there's maintenance, which is telomerase, that's Vishnu.

But Shiva is the, Shiva is the difficult one. Cause if you start killing off everything, you might just be dead. So part of the privilege of getting old is that you get to live longer, but it's not a pretty sight.

You get a lot of inflammation, you get wrinkles, you go blind, lose your hearing. So this is the same process. It's one disease with a thousand faces.

It's all driven by telomere attrition. So we can mitigate that by taking a telomerase actuary like I've been taking for 18 years. But the real hack here is what they're not working on, right?

These billionaires that want to hack aging, they hire the wrong people. They hire people who have their own acts to grind, their own revenue source, their own boat payments.

So you just got to think of it like an open mind, like a beginner mind. So how would you hack aging? Well, every person that's born needs to just save their stem cells.

freeze their placenta, and then 100 years, 200 years, 500 years, they can use their own replacement parts. Because the science involved with thawing a piece of placenta Extracting the stem cells.

That's here. That's been here. I mean the chimera manufacturer that I use for 10 years They have a single donor going eight years now and and they made hundreds of files.

So what does that tell us? Yeah, even though Heinrich or vandal slipper didn't have an immune system Now they have the science to make whatever stem cells you need hematopoietic whatever cardiac All you need to do is freeze your own cells and then in 100 years it can be trivial.

You can drive up to like a kiosk in a Ralph's and there's sperm cells, you know? So this is the hack. Then you don't have to worry about senolysis anymore.

You can kill off the bad ones because you can replace. So I just think that there's a constant entropy. It's just you're always like death and taxes, you know?

Taxes are just always going to eat away. You can't own your house because there's property taxes, right? So there's all this great thing going on. But if you know how to store your stem cells and the future scientists, the current scientists can differentiate that.

Then that's the real hack. Cause then you don't worry about, you know, death. You can replenish. But the thing is right now, if you kill off all your stem cells with chemo, I mean, look what happens when you get chemo, right?

You get your hair, you get GI problems, brain fog. That's because you, you pay Rob Peter to pay Paul. You basically killed all the rapidly dividing cells.

So it teaches you. It really does. What's funny is you're making me feel guilty because with my first two kids, we did freeze their stem cells, not knowing where science would go.

It's easy. It's a couple hundred bucks a year to maintain the freezer that they're in. And then the third one, we were like, yeah, but they'll have stem cells from their siblings.

If they get sick, they'll have, you know, I'm like, well, shoot, I should have gotten direct exact stem cells from her. Of course, she's only 15, almost 15. So she's still pretty young version.

Yeah. Yeah. So we can see most pretty good. Yeah. Siblings will share, but genetically speaking, in terms of rejection, they're not close enough. Not that close enough.

But for most of our listeners. Let's say, what would you have them do? You would have them at this point in the game. If you wanted to slow and or halt aging, you would have, obviously you have one book that talks about just the basic lifestyle modifications, which a lot of the functional medicine guys are focusing on.

And then you would have probably, uh, telomerase, uh, Activator, how do we do that? They can go to my website, rechargebiomedical.com and learn about 65. I mean, if they do a little deeper digging, they can probably disambiguate what it is.

But the generic versions they sell on Amazon, I'm not sure they're as effective. I've heard different things, probably not. There's a process of micronization that makes them more bioavailable.

But yeah, I really, you know, if you look at pictures of me from 25 years ago, I look much worse and it actually caused a visceral fat extinction in me.

So I lost 15 pounds without diet exercise after 90 days. So it can do a lot of good stuff. If you look at the molecule, it looks kind of like vitamin D or a steroid molecule.

Yeah. It's kind of magical and it just seems to do a lot of good things for people. So that's just the maintenance. You know, in the future, yeah, you can find a reputable place to store your placenta.

Um, and then, you know, even nowadays you can go places, they'll store your stem cells because you're never as good as you are today. But there are a lot of lifestyle things you can do, sleep better.

Bad sleep is a huge accelerant of aging, bad nutrition and bad thoughts. So we go into that, you know, you would know what's good for you. You know, what's not.

So. Well, and most of my guests are talking about functional medicine and ways to do what's good for you and stop doing what's bad for you. And a lot of it is, if it wasn't common sense, it is becoming common sense now.

but directly affecting our telomeres along the way, sounds like a fairly, what's the barrier to entry there? Is it expensive? It is. You can use it like aspirin.

Like if you have like a body fatigue after workout or a rough night drinking, you can use it like aspirin. Just keep it in there. Yeah. Cause it's, the effects are pretty immediate.

It makes the sleep much more efficient. So the dreaming is rapid and healing is faster. So it's really just cost at this point, cost and hassle factor that getting as telomeresic activator.

Yeah, if you want to get regularly every night, like I do, then it's cost. But if you just want something, you know, a little helper once in a while, if you're having a tough day, you can use it like aspirin.

I mean, you don't use 12 bottles of aspirin a year, right? And it's oral, you're taking it orally? Yeah. We take it with a little snack at night before sleep.

That's easy, man. Yeah. You feel the effects right away. It's not subtle. Yeah. I mean, obviously there's a limited amount of money and resources out there, but if I had something that made me feel better today, I'm going to kind of want it tomorrow.

Yeah, it's like that. On the rare, rare times in the last 18 years, I've forgotten it somehow. It's just like, usually it's like Groundhog Day. You just wake up and it feels like the same day over and over again.

But then you can kind of feel yourself aging one day at a time if you don't take it. It's weird. That's crazy. Well, gosh, man, that's hilarious. So. What an interesting conversation we just had.

And I feel like it was probably a whirlwind for a lot of our folks because there's a lot of, there was a lot of science jargon in there. Yeah. Okay. They can revisit and all my books are aimed at like an eighth grade level.

So I explained the science in a really simple way. So people, most people at least are like, Oh, I get it.

Practical longevity advice and closing remarks

I think I get it. So I practiced in New York, Texas, Florida, Utah. Hawaii and California where I live. So we will put, we will put your YouTube and links to your website in the show notes for sure.

Um, uh, is that the best way, any other ways you want us to get people to get to you? No, I have a sub stack and I can subscribe on YouTube, but mainly they just join my newsletter.

Cause as you know, I also have a podcast. So we interview people about functional medicine, mold, autism, all kinds of things, you know, Lyme, glutathione.

So all these topics, we're doing the zeolite guy next week. So it's cool. All right. Well, hey man, thank you so much. Um, what a fun conversation that was.

So, so it's Dr. Ed Park. He's, we're going to put information in the show notes, how you can get ahold of him and go check out his website and his practices in enough States.

He's probably close to you anyway. So thanks man. And, uh, you have a wonderful night and thanks for coming on. That was great. Thanks Dr. Carter. Okay.

Have a good one. Thanks for joining me today on My MD Unscripted. I hope today's conversation opened your mind and inspired you to imagine a better path for your health and therefore life.

If you found value in this episode, be sure to subscribe, leave a review, and share it with someone passionate about transforming healthcare. Real change starts with real conversation, so let's keep on going.

Until next time, stay well, stay curious, and never stop pushing for better.