Introduction to Reversing Aging

So you're just sitting here talking to Dr. Kara Fitzgerald and just talking about we're really entering a golden age. And so I'm so excited to have you on.

Kara, thank you so much for for joining us today on the Reverse Alzheimer's Summit. And, of course, just the phenomenon of Reversing Alzheimer's. People would say a few years ago, as ridiculous as they would to say reversing your aging and to my knowledge, you are.

Is it fair to say you are the first person to publish Reversal of Aging? Is that fair to say? Actually, there were two studies that that that preceded mine.

We were the first to have a randomized control study, and we were the first to have a diet and lifestyle intervention. But the trim study using growth hormone, metformin, DHEA, Zinc came out.

That was the first that was really the first one. And Horvath, the the you know, the developer of the bio DNA methylation clock, he was an author on that study.

And just to give you some context that came out in 2019, our study was underway at that time. But Horvath is on record many, many times saying biological age in humans, you know, really can't be reversed.

Like we can't there's really nothing we can do about it or there's really not much we can do about it. There were some you know, there were some there were some evidence that a healthy diet, a little more education, you know, might slow things down a little.

But it was pretty equivocal. And so he was just really blown away with that first trim trim study. And, you know, we were interested in changing epigenetic expression with our intervention, but I wasn't holding my breath that we would really be moving the needle on bio age.

I mean, of course we were going to look at it, but when we our study was underway, when we learned that it was even possible. So I guess to your point, it's incredibly new that we are we've got this evidence in humans now, and ours was really one.

Yeah. One of the first studies to demonstrate it. Mm hmm. Yes. And so, yeah, you actually used a, I would say, a more physiological approach, but it brings up some really interesting questions.

So, you know, when you reverse cognitive decline and it's pretty easy to say, oh, your MOCA score went from, you know, 20, we've seen them go from 18 to 30 and your MOCA score improved.

So that's pretty easy. Now, this is why we say reversal of cognitive decline. As I said that in the very first paper, I can't say that we're reversing Alzheimer's per se. We don't know.

We got to have pathology. Now, the good news is we've got blood tests. You can look now at p-Tau. How did you improve your p-Tau 217? Now with aging, it's quite different because aging involves a whole panoply of things. Mm hmm.

Biological Age, Epigenetics, and Reversal Evidence

And so the question obviously comes up in years. As I recall, you reduced three point to six years of my remembering that correctly. Yeah. Yeah. To more or a.

Little over three years compared to the control group, which is huge in a relatively short amount of time. Yeah, very

short. Yeah. Eight weeks. So the so the question is if you change the what you really did was you change the epigenome that gives you biological age.

Now, do you know if you change telomeres? You know. So the question is, when you change one thing, let's say just take the epigenome and you change it.

Is that really changing the organismal, aging? Yeah, it sounds like the sense is that you probably are because these people are healthier. They seem to be doing better overall.

What's your sense about, you know, changing the epigenome for aging? Are you changing global aging? That's a great question. I mean, so certainly in standard biomarkers, you know, and and, you know, quality of life questionnaires and so on and so forth, we would say, you know, of course, you know, we are using this intervention in clinical practice and looking at a broad swath of, you know, standard biomarkers and and physical parameters.

We can definitely see that. But like, to your point, you know, really thinking about the physiology of aging and changing epigenetics, changing the clock.

I mean, we can we could ask the question, are we are we are we manipulating? Are we favorably augmenting beyond the standard chemistries? And so forth, which are really fundamental and incredibly important, the hallmarks of aging, you know, getting to sort of like the root of the aging phenomenon itself and, you know, we we need to continue to research our intervention to demonstrate that.

And and we are continuing to research it. And we'll fine tune it and, you know, have a have a methylation diet and lifestyle or or younger you as we comment, you know, branded it 2.0 and stuff but what's extraordinary is that it seems that aging can be, you know, just going to the cellular cellular reprograming conversation that you and I have had aging can be manifested by manipulating the epigenome.

And when you so when you change epigenetic expression, you can see a manipulation of those downstream hallmarks of aging. So you're sitting, I think, really right in there with root cause aging.

And so you can do it favorably. When we look at cellular reprograming or even, you know, animal models, some of the work out of Sinclair's lab, you can see them create age by manipulating epigenetics and then reverse it or, you know, make them younger, biologically younger, physically younger by many different parameters, by manipulating favorably epigenetics and, you know, a scientist out of Stanford, Vittorio Sebastiani, is is demonstrating this in human tissue models and really getting ready to could when to phase one.

I mean they're in FDA talks to actually begin to bring some of this cellular reprograming technology to clinical trials. I mean, it's so cool. And so what I want to say is that I think we may be touching on our intervention, which, you know, it's standard diet and lifestyle stuff that we all know is is smart.

It's it's intentional. It was intentionally built like you could look at our intervention and say, this looks a lot like a mediterranean diet, but if you look at it even closer, you'll see that it's built brick by brick to influence DNA methylation, which is how we measure bio age.

There were very intentional pieces in there. And so the question is, are we touching on sort of root cause aging that can have these downstream effects on all of the hallmarks of aging?

And that remains to be seen. But I think that quite possibly we're heading in that direction. And there's a there's actually a new way to measure that.

So in Sebastian's lab, they are seeing that epigenetic reprograming influences all the downstream hallmarks of aging. I mean, he's on record saying that he thinks sort of the the the the vis of where you can influence aging sort of the fulcrum is right there in the epigenome I asked him is this he was on my podcast I asked him is this you know, is it root cause?

And he said, I don't know that it's root cause, but if you go in there and you manipulate epigenetics or you're changing aging downstream, you're manipulating all the hallmarks of aging.

And so they invent they they've got a clock in their lab that I would like to send our specimen to, where they're tracking the aging phenomenon across the genome.

So it's a much bigger clock than anything ready in prime time. And they're and they're showing that caloric restriction has the same it's manipulating the same regions as their cellular reprograming is.

And so he and I have been dialoging about some of the polyphenols, some of the interventions we've been using, and whether or not it could have a an influence on this clock that's really capturing, I think, the aging phenomena perhaps more closely.

And I would say that, you know, it's possible. I mean, that's a study that I am just really eager to undertake. But and I'll stop and let you. Well, that is fascinating.

I've always considered, for example, when I was at the Buck Institute, so my consideration of, you know, aging, you've got one person standing with a circle around and one person standing around has got a bow and arrow, one's got a revolver, one's got a rifle, one's got a bazooka.

Diet, Epi-Nutrients, and Fasting Strategies

You've got to avoid all of these things to age successfully, right? You've got to avoid atherosclerosis and you've got to avoid cancer. You've got all these different things.

But let's talk about some actionable items immediately here. So you brought up something. Everyone's going to be saying the same thing and I'm thinking the same.

Oh, you said you built this brick by brick. You were focusing on methylation. Okay, let's say that someone wants to use your bricks to date. What sorts of changes in their diets should should they should we make so that we can use your bricks to be a little bit younger, to have to have a younger us?

Yeah. So just, you know, going right in there and influencing our gene expression towards something better we want to be eating. So this comes as no great surprise.

We want to be eating a lot of vegetables of really good quality vegetables, dark, leafy greens. Yeah, well, right. You know, cruciferous. We want to be eating colorful veggies.

You know, I get some fruits in there, too, probably, you know, lower glycemic stuff, you know, like berries and so forth. Mushrooms turn out to be that we call these all epi nutrients.

And so and we and we say we should say that each each forkful of food could be packed with this epi nutrient information to sweet talk gene expression to move you towards a younger a younger you so you know all the veg that were mentioning mushrooms egg egg has something called choline which of course, you know, is is amazing for brain health.

It's also a methylation. It's a you know, it's an AP nutrient superstar. Most of us are low in it. That's exactly. Yeah, that's right. You know, if people are open to doing liver or taking liver in caps, this is another extraordinary nutrient dense epi nutrient superstar.

We need adequate protein. We need adequate hydration, green tea, curcumin, resveratrol, ludi, all in rosemary, dark acid in in rosemary, in tumor, etc., etc.

a lot of the time honored either nutrients used in in in classic cooking or in traditional medicine around the globe have are being discovered as having this epi nutrient capacity.

And I wonder if that's why they're, you know, anti-inflammatory, anti-cancer, you know, brain preserving antioxidant, they're pleiotropic and maybe it's because they're influencing gene expression in such a potent way.

So that is anybody can go into their spice cabinet. Easy peasy. Anyone, no matter how picky they are. Chocolate, by the way, coffee. We can all find a bunch of epi nutrients in our in our kitchen cupboard tonight and in our fridge really easy.

You know. So it just, you know, says empirically things that people were discovering thousands of years ago that were really wonderful. Yes, totally. They weren't so stupid.

They were actually kind of on to some good stuff. Yes. And so and what about the fasting part of this? What do you suggest for best impacts on your aging epigenome? So I know it's powerful fasting or caloric restriction, you know, just in Sebastian Joe's lab and what their what their what their demonstrate thing.

We in our study model, we only suggest a 12 hour on 12 hour off structure. And we're doing that because it's doable. You know, this is I mean, and we know the data out there, the economic data on if we slow biological age by a year.

You know, there's we stand to save, you know, trillions and trillions about. So we want is many people adopting these principles as possible and we want them to be doable.

So a 12 hour and 12 hour off which a friend of mine just called normal eating is is is a good entry point into the conversation. If you can do more, you know, if you can go to maybe a six, eight structure, if that's comfortable, you know, or a 14, ten, I mean, play around with it.

It needs to be something that is not stress inducing because you can have net you can actually have negative fallout. If you if you move outside of something that's, you know, easy or relatively straightforward and comfortable to do.

But if we the other pieces so we want, I think a larger fasting window is very beneficial. But here's the other piece and I think this is missed by a lot of people, scientists included, that window where you're eating has to be packed, in my opinion, with these epi nutrients, you know, and there's and there is, you know, we we outline quite a quite a few targets to attempt to be achieved.

And so that eating window has to be really important. It can't you know, it's can't be like sushi night at the restaurant for your calories that day. I don't know that you can anticipate getting the same kind of results with that.

So the absence of eating is and certainly is is important, but the information on our forks is, you know, as important. Interesting. Okay. This is really helpful.

You know, there's such an interesting relationship between the aging process itself and the various diseases we associate with aging from, you know, Alzheimer's to Parkinson's to cancers to, you know, things like that.

So that is really fascinating. Okay. So and people sometimes say to me, there's no reason to work on Alzheimer's. We're just going to get rid of aging.

And then Alzheimer's will never happen. Well, okay. But then I always point out, yeah, there are changes in the brains of these patients in their twenties sometimes.

So it's not purely aging. And yet there's some absolutely fascinating relationship between the aging process because as you know, you just see this dramatic increase up into your eighties and yet the big increase that people have reported in the last few years is in people in their forties and fifties.

So, I mean, what is that? I mean, what is your what's happening? What's happening in the what what's influence in those pathological changes in the in.

I mean, to be honest, I think these are functional medicine. Societal change is what has happened since I trained way back in the early eighties. You know, is we have more processed food, we have more exposure to toxins than ever before.

You know, we have more kind of messed up lifestyles and we have more we have more understanding of, you know, living among mycotoxins and things like that.

Now, to be fair, air pollution, for example, in L. A. has gone down. We've but we've got the California fires. We've got a lot of things that are driving us so that the way we look at Alzheimer's is, you know, too little supply or too much demand.

You're on the wrong side of the curve very much like what Dean Ornish found in atherosclerosis. You know, you got to be on the right side of that equation.

And so, you know, we've added negatives to the equation. Unfortunately. And at the same time, there are so many people who've got some vascular disease, some degree of sleep apnea, some degree in their oral microbiome.

This is. Very. Much a functional medicine. Absolutely problem. Well. Let me say this then, because I think that you've you know, you provocatively said that these things are happening really before the aging curve.

But in fact, if we were tracking aging, we would see this as an accelerated aging phenomena. So so it ball, I think it falls under that heading. We're seeing this accelerated aging phenomenon even in kids where, you know, we see hyper hypercholesterolemia and so forth.

I mean, it's it's pretty extraordinary, you know, or we see we see, you know, metabolic changes. We see prediabetes. That's the aging phenomena, you know, kicking in then.

And I think the more sophisticated clocks we have, the more we're going to really be able to tap into that. So I think, you know, I think that's. What is amazing to me because, yes, with your measurements, you're going to be able to say, well, here's you are 11, but you actually you've got the age of a 25 year old.

Yeah, that is concerning. You know, you can be aware of unfortunately, which is really tough.

Alzheimer's, Accelerated Aging, and Disease Links

And of course, this is what Robert Lustig has been preaching for years. He actually went into pediatrics to avoid dealing with adult diseases. When he got there, he said, oh, my gosh, I'm seeing type two diabetes.

I'm seeing obesity. All the things that I thought I was going to get rid of by going into pediatrics. So it's actually, you know, amazing. And you're right, I do I do agree with you.

These are 20 somethings that may be aging like 40 somethings. And you see the reverse 40 somethings that may be aging like 20 somethings. So this is, you know, as you are able to get these data which haven't been available in the past, you're going to see so many remarkable scenarios.

So let me ask another piece of this then. When you see people with better epi genomes, do they have better telomere lengths? Do when you start to do the right thing, does it just stop going down or does it actually get larger?

I mean, we always worry about if you're getting larger, is that going to be related to carcinogen versus what's happening with telomere lengths in association with this and what's happened with senescent cell accumulation?

So we're not we're look, we are looking at telomere length now. We didn't look at it in our original study. We're not looking at senescent cell accumulation, although that would be a lot of fun to it.

It would be fun to kind of cast a wider net with some of these labs that you're proposing. We need we you know, we just need to be mindful about the funding model.

But yeah, that would be that would be fabulous telomeres don't they're don't necessarily change. They they don't change quickly if they change at all.

I think a goal would be preservation, you know, of telomere length that would be a reasonable goal. Like when we we look at telomere length and we look at the pace of aging, those are the two tools that we're using primarily these days, the pace of aging being a third generation and DNA methylation clock.

And I would say that, you know, we're not seeing a we're not necessarily seeing telomeres track with slowing the pace of aging. And, you know, my understanding on the you know, my read on the literature is that we shouldn't be telomere preservation is is is probably the best we can do.

But I will say that we've certainly seen telomeres increase. I mean, we have but it's I'm not sure how to explain it. You know, when we do, I don't know that we can say here's the recipe for telomere preservation.

I mean, there are some products out there like the telomerase inhibitor, a POLYPHENOL, which is probably an AP nutrient, by the way. I think maybe it is, but so there are you know, there's there are some things that we can do that might inhibit them.

And yeah, you know, to your point, are we concerned about cancer when we see them very long? I don't I don't have a really good answer for that, you know.

Yeah. And then, you know, let me acknowledge the late great Dr. Judith Campisi, who was at the buck for years. I actually I recruited her to the book many years ago.

She's an incredible researcher, and she was the one who came up with senescence Associated Beta Gal and also the one who came up with, you know, the idea of of or did so much work on analytics and was involved in the unity at the beginning of that company.

So just tremendous researcher over the years. Do you think that the things that you're using in your patients do have a synergetic effect? Yeah, certainly some of those polyphenols have been identified as such.

You know, we're we're we're supplying, you know, foods with quercetin and so on and so forth. I, I, I, I, and I think probably the, you know, the sort of the entire cocktail of the program plus meditation, plus the exercise component, the fasting window taken together, I would anticipate, has a favorable influence, not just on senility but on, you know, multiple hallmarks of aging, you know, dysbiosis being a new one.

Inflammation, mitochondrial helped mitophagy. So I would anticipate that we're touching on multiple hallmarks of aging in our intervention. Yeah, really fascinating.

Now, one of the things that we found in in treating people with Alzheimer's is when you do the right things. And many of them are very similar to what you've been doing.

Yeah, that's improvements in blood pressure. You see improvements in glycemic index in their in their glucose control in general. Yes. And you see, you know, improvements in in their lipid panels, things like that.

Are you seeing that with your approach to you know, in other words, is that part of getting younger? Yeah, for sure. So there's two cohorts that we work with.

We have the the population. We've, you know, we've researched and so far those people are extremely healthy and our intervention is getting healthier.

In fact, our two publications, Biological Age, was younger than chronological age at the start, which is kind of cool.

So I mean, they were just really healthy and there was evidence that, you know, demonstrating that they were helping and they got and they got younger still.

So our cohorts right now are sort of are we're focusing on looking at the healthy population because we're we're interested in kind of isolating the aging phenomena.

So that's been really pretty exciting. And, you know, the we we studied a we published a case series in women who outperformed the guy. They were the series of six women.

They got on average about a 4.6 years younger using the same clock. However, it was done in blood and, and our first study was done in saliva. So it's not quite an apples to apples comparison, but I think it is really cool.

And these women were you know, they were biohackers. I mean, they really started they came to us, you know, excited to be early adopters in this. So they were very healthy at start and biologically younger as well.

When we use this in the clinical practice, in our clinical practice, where people are coming to us with, you know, different with autoimmunity, with, you know, cardio metabolic disease, with, you know, cognitive impairment of varying degrees.

Yeah, we see those parameters. We see people lose weight, we see blood pressure improve. We see, you know, antibodies and autoimmune disease drop. I mean, really not dissimilar from the kind of changes that you see in your protocol.

So yeah, we, we do in the clinical setting, we just haven't studied it yet. And it would be fun. I mean, I would honestly, I would anticipate if we if we look in a population presenting with like if we were to look in type two diabetics and run this program that, you know, we know already, you know, Nathan Price published on this, that there have a pretty profoundly accelerated biological age.

I mean, the chronic diseases of aging routinely are commensurate with an accelerated biological age. So I think diabetes was the most severe. And so I would anticipate our cohort would start out older.

But, you know, we would get them that that window, that change would be much bigger. Yeah, that's a great point.

Telomeres, Senescence, and Aging Biomarkers

So I guess that brings up the question of, you know, are these people who have these age related diseases, is it because I mean, is the disease causing the aging or is the aging causing the disease or are they actually both doing you know, is it both or neither?

I know that's such a great question. You know, that's a to bear a question. If you I don't actually drink. But I mean, that's such a great question. I mean, it gets to the heart of what is aging.

I mean, and it's a question that I will I asked you when I interviewed you a couple of years ago, you know, what is it? I mean, I think that there's I think that I think I think the wear and tear of life, you know, the influences on the epigenome there, those you know, those changes prompt the aging phenomena.

I think that we're also hardwired. You know, I think there's probably two paths we're hard wired, you know, after we're we're past reproductive reproductive maturity.

I mean, we're, you know, things begin to change epigenetically. And so I think there's two paths of information that we that we need to tease out to really answer that.

However, that said, you know, and that's where you get into Yamanaka factors and cellular reprograming I mean, they're touching on both of those paths.

You know, they're reversing, you know, I mean Yamanaka can reverse age to to pluripotent stem cell status. I mean that's a real reversal of the aging phenomena.

But we've got plenty of runway just addressing environmental inputs on the epigenome without even getting to sort of some of the some of the, you know, the aging phenomena that's probably baked into our genome slash epigenome.

You know, we've got plenty to do just getting ourselves moving and eating right and paying attention to pollution and stress. And, you know, our environmental inputs will keep us plenty busy and be able to, you know, help allow us to experience a health span commensurate with our lifespan and allow us to have a robust lifespan.

I mean, we know humans can live a good 120 years. I mean, we know we know that and we know that certain population organs like loose ends populations have a health span commensurate with their lifespan.

And we also know that that those blue zone populations, they've done a couple of studies. And actually there was a really cool French study, our biologically younger actually up to 30 years biologically younger.

And so sometimes I joke that, you know, 100 is the new 70. I mean, so we've just by living a good life here and now, we can reverse and slow down biological age and allow our health span to be, you know, right in lockstep with our lifespan and extend both of them.

And then after that, we can, you know, talk about some of the you know, the what my friend refers to Sergio Young is the longevity bridge where, you know, we start tweaking cellular reprograming and, you know, Yamanaka Factor interventions and some of the more radical stuff that's happening in the, you know, in the research setting.

Yeah, that's going to be really interesting to see what happens with that. So, you know, when I was on the National Aging Council years ago, there was a report that came out from one of the other people on the council who showed that we in the United States get our first complex chronic illness, our first chronic illness about ten years before people in the UK, typically in our forties, whereas in the UK you get your first one in the fifties and so you end up with a relatively short health span and a relatively long six span just to get guess where we stand with our life span.

So let me ask you then, let's say we have let's say someone's coming to you. They have a 30 year old person and a 70 year old person, let's say, you know, could be even father son okay.

Or mother daughter, what have you. Are you going to be able to have a similar effect on the two of them in terms of reduction of their epigenome? Or is there a point where you say, you know, I really can't do it here beyond X, you know, beyond age?

God, I'm you know, there was a really cool study that came out looking at the influence of exercise on the epigenome. Exercise is really one of our most potent happy nutrients.

You know, it's probably right up there with kale. It's so potent and pleiotropic and it's in its influence. And one of the things that they identified and I love to talk about this is the older the individual was, the more epigenetic change, the more bang for their epigenetic change they got.

They actually had more impactful epigenetic changes. And specifically they were looking at tumor suppressor genes. So tumor suppressor genes protect us from cancer and they become hyper methylated as we age, which increases our vulnerability to developing cancer.

I mean, it's just this like, why does that happen? You know, like this raw deal and then oncogenes are turned on, you know, what's that about? You know, is it just sloppy epigenetics because of an environment or is there some sort of a baked in program?

But it just it doesn't seem cool at all. And incidentally, that's reflected in the tumor microenvironment. So like the tumor microenvironment looks like aging.

But it was so exciting to me to see that older individuals actually achieved more bang for their buck doing it epigenetically and exercising. So 100%, I would say we you absolutely start wherever you are.

You're at with, you know, a robust commitment and anticipate seeing benefit. Okay. Very interesting. So of course, with respect to cognitive decline, one of the things that has a an important benefit happens to be HRT, a bioidentical hormone replacement.

Clinical Outcomes and Hormone Therapy

How what is that what have you looked at that and what is happening with your epigenomic age when you are including HRT? So I know that the absence of HRT is or certainly estrogen.

I mean, if we look at menopause, we can see that what we can see physiologically, you know, once women hit menopause, their risk of developing all of, you know, the various chronic diseases of aging increases.

And we can see that epigenetically as well using HRT or bioidentical HRT, you know, has that to my knowledge, we don't have any specific publications on that yet.

However, it would be an easy data mining project for one of the labs, and my guess is that it bioidentical HRT would be associated with a younger biological age.

But I could actually get that answered and I'll email you. You can you can put it in the show notes for my for my talk because I don't have the answer.

This is always an issue because people say, well, you know, I had menopause ten years ago. And so I'm told, don't don't even think about HRT. But one of the things that fascinated me in our experiments was that you can actually look estradiol binding to its receptor has an effect, as you know, on hundreds of genes and one of the ones that increases is the alpha secretase that cleaves ap in the connection direction.

So we think of this now, it can go connection or protection and protection side is associated with Alzheimer's disease connection is associated with making and keeping memories and so estradiol definitely drives it in the connection direction, whereas NAF Kappa B drives it in the protection direction, it's an inflammatory marker, etc.

So you can really follow these molecular pathways beautifully to see why these things should or should not work, though. All right. Yeah. And I think we could conclude that, you know, there's a strong place for HRT.

Yeah, sure. And in your studies, you've got you look at all these wonderful things like their epigenome. They're they're biological, aging. One of the things that's been fascinating to me is how do you look at twins?

We quantitate so many different things. How do you look at skin? Skin is one of the most important things to so many people. Does my skin look better?

You know, that sort of thing? Yeah. And so how do you quantitate aging of the skin? Is there a measurement that you do or something you do to say, okay, your skin is three years younger than it was becoming?

I think there's a measurement coming. So so there are some, you know, pretty neat scientists at a a skin care company that was started by four PhDs who who developed a skin biological age clock, you know, and they've published on it.

And they've actually they actually used it to test various peptides. They used it to pull together, you know, a product that they developed which, which and they're in there researching it and they're publishing on it and they're, you know, they're looking at beyond biological age, you know, mechanistically what's what's occurring.

I mean, there's some just really cool science. There's another there's another company that's doing something similar in skin science as well and looking at mitochondrial fitness, you know, and skin health and just showing

some really cool results.

So there, you know, there are tools that can measure, you know, wrinkles and and sun damage and so forth that that could give predict that that can produce a biological age like Matty spores have these tools.

And these tools actually have been used in different in the research setting. So I think there's a a place for those. But I'm very excited for, you know, skin age clocks to be available to us clinically.

I think that would be really fun. Yeah. And clearly know different parts of skin are going to have different effects, but you know, it's something that really impacts people.

People know is my skin is going to be looking better, that sort of thing. Yes Very. And we know I mean and we know certainly anybody who adopts a healthy lifestyle habit will see these changes anecdotally.

I mean, the fact of the matter is we are hardwired biologically to be able to predict somebody's age. So we can I mean, we can look I mean, that's been published on so we can look at another individual and say, you know, your age and your why and we can look at the, you know, in your why age.

I mean, we can actually perceive accelerated biological aging or decelerated aging. So adopting the healthy lifestyle, which, you know, when people say you look great, you look younger, it's probably actually trip.

It would be fun to actually test it, you know, and to have that hard evidence that's awesome for people. But really, when people notice why you look great, you know, wow, what are you doing?

The reality is they're they're picking up that they're biologically younger. I mean, there there's just evidence for that. Yeah, such a great point. Okay.

And then the other thing is, is cognition, of course. Did you do have you done any studies when you're looking at younger age? Do they do they seem to be better cognitively or do they even mentioned, hey, my memory is better, hey, this is better, that's better or what?

What's your sense about that? So this would be more in the clinic setting and not, you know, and not what we're studying in the in the research setting.

Certainly we see we can see that in our MCI patients, you know, where we're tracking with MOCA and, you know, in other tools that you've you know, that you've been advocating forever.

In fact, you know, one of our clinicians is is trained in your model. Sure. And I mean, I think all of us are using components of it. But one of our clinicians who does it primarily is, you know, really follows in lockstep your program.

And we layering in younger you nutrients and so we will see that in that population for sure but we haven't we haven't studied it in the research setting.

If you go back to the if you're at the Buck Institute again, maybe you can bring us on board and we can somehow lock into practice and be able to, you know, be able to really answer your great questions because it would it would be so fun.

Yeah, it's as I say, it is. We have gone from the dark ages, you know, a decade ago where there's nothing you can do about these things.

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You know, as as as Linda Marsh of the science writer said, I don't think we've ever seen failure on such a level before when you talk about neurodegenerative disease.

And of course, the same issue was true for aging where like, what can you do about it? It's just, you know, it's going to happen. And now suddenly all these things have opened up and you're seeing people get better.

You're seeing people stay better. We just have a paper that's coming out, showing over ten years of sustained improvement in some of the patients. So I'm really excited about that.

You're reversing biological age so that brings up the other question, which would be the next obvious question. If you're reversing the age, how much of a Benjamin Button effect can you have?

You know, 3.2, four years. Yeah, you go can you go farther if you keep going? You showed that. As you said, you showed it quickly. What if someone does this for five years?

Can they get five, six, seven, eight years? You're where do you where what is the max? And there I said, okay, so I want to make two comments. One is that, you know, we we can see that Alzheimer's is an age acceleration phenomena.

We can see that standard blood biomarkers that have been you know, that have been that are associated with biological age. So they're you know, they're weighted in an algorithm to cap to capture biological age and standard blood biomarkers.

We can also see that in DNA, methylation, biological age clock. So we can see the aging phenomena. And therefore, you know, it stands to reason, you know, that we can absolutely get in there and change, you know, in Reverse bio age as I mean, it's got it just has to track with the work that you're doing.

And I know you're you're bringing these measurements into your your research. You just must be looking at epigenetics at this. Moment, thanks to you. So we are working with the true diagnostics to look at epigenetic biological age basically.

And we will see where things stand at the beginning of the trial and at the end of the trial. I'm so excited to see that. And even beyond the clocks, you know, there's the methyl loam is far greater than the clock.

So there's much else to look at. And we're doing that now. We're actually working on a what they call an Epigenome-wide association study with our our our first cohort.

And we see that we've influenced the course of hundreds of genes, and that's going to be just really fun. And I think that gets into the nitty gritty of metabolism.

And, you know, what's happening, you know, very, you know, preclinical. It's just very exciting to me. So yeah. So Benjamin Button, like how far can we go?

How far do you go? Yeah. So with art with the people ask me that question. I just it's so funny. I, it, you know, with our diet and lifestyle intervention, we, you know, we aren't going back to pluripotent stem cells, you know, with our diet and lifestyle intervention.

I think what we get to do, to your point, is shut down that sick span. And, you know, we get to extend. So let's have a very robust life span and have our health span walk in lockstep with that.

You know, let's all live to 120 and be vibrant and strong and active and cognitively intact, you know, and just, you know, all of that. I think that is, you know, one of the potentials for this diet and lifestyle program, perhaps layering in, you know, some of the like rapamycin, some of the analytic combinations that people are using, you know, some of the other slightly more radical, if you will, interventions, you know, maybe exercise some stem cells and so forth.

There's a there are a lot of tools that are nearing that are ready for primetime or nearing. And then at some point, you know, the Yamanaka Factor intervention, you know, will very likely, you know, enter use clinically.

Yeah. Yeah. And I gather from David Sinclair's work that, you know, he's as he's saying if you wait too long it doesn't seem to do what you'd like it to.

I guess you may be able to go from 40 to 20. You probably can't go from 70 to 30, that sort of thing. Now, you know, we'll see. Time will tell. And maybe, you know, maybe ultimately, you know, these cars that we drive around called human organisms are, you know, are going to be you know, you keep on replacing the right parts.

You keep on doing the right thing, checking the oil, etc.. And that, you know, you can you can keep them going for a very long time. You know, we'll see.

I think this is why I say we are going from the dark ages to a golden age where we've got blood tests now for Alzheimer's. We've got better ways to do we we've got, you know, the measurements of biological age.

You know, we are beginning to understand the relationship between biological age and chronic illness. We're starting to understand why it's going awry, what's happening with what we're exposed to, where we live, where we eat.

I was just talking earlier to Brian Karr about just changing the way we build homes. As he points out, he's driving down the road, it's pouring rain and all these houses that are being built, all the wood is just sitting out there in the in just wet.

And so, of course they use the same stuff. And now you're living in mold food. Do you really want to do that? I mean, is that really the best way? So I do think that, you know, public health measures, you know, reducing the processed food, you know, reducing the fire, all that sort of stuff is going to help.

So so we're kind of getting to that point of it's not just about taking antihypertensive and everything will be fine. So this, this is really a golden era and obviously you're contributing hugely to it.

And so, Kara, just absolutely great to talk to you, as always. Please keep up the fantastic work. I look forward to hearing more and I'm very interested in everything you're doing with reversing age.

Obviously, you've got the stuff, you've got the book Younger You, you've got the studies on women, you've got the studies on on both. It's obviously, you know, works for everyone.

And as you've pointed out, seems to work just fine for older people like me, as well as younger people like you. So that's so fantastic to hear. So thank you very much for your great work.

Thank you for joining me.