Alzheimer's, Dementia, C0V!D-19 Long Haulers, and Other Brain Disorders Transcript

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Introduction to Dr. Bredesen and Alzheimer's Recode

So today we're going to talk about memory and Alzheimer's. And I've a very special guest on. He's the author of The End of Alzheimer's. And this is doctor Dale Bredesen and received his undergraduate degree from Caltech and his medical degree from Duke.

He served as resident chief resident in neurology at UCSF, and then was a postdoctoral fellow in the laboratory of the very famous Nobel laureate, Professor Stanley, prisoner.

He was a faculty member at UCLA, and in 1998 became the founding president and the CEO of the Book Institute for Research on Aging and the adjunct professor at UCSF.

The Edison Laboratory studies basic mechanisms behind neurodegenerative disease processes and the translation of this knowledge into more effective therapeutics and tactics for Alzheimer's disease and other degenerative conditions.

This led to a publication of over 220 research papers, and he's established the Alzheimer's Drug Development Network with Doctor Varghese in 2008, leading to the identification of new classes of therapeutics for Alzheimer's disease.

So him and his group developed many different approaches. And the latest actually is something called the proteasome protocol, described as reverse sort of symptoms of those people with memory loss, mild cognitive impairment and Alzheimer's disease.

And it's a precision medicine based, protocol. I was published in 2014, and had, multiple enhancements and revisions recently. And I'm so honored to introduce Doctor Bredesen to talk about the root causes of memory loss.

And we're going to talk about this program called Recode. And we're going to talk about how this is really applicable to, people with all sorts of neurodegenerative diseases and of course, Alzheimer's disease.

I'm so excited to to talk to you about cognitive decline and memory. It's going to be exciting conversation. So, welcome to the summit. Thanks so much for sharing.

So much to talk about. This is a really exciting time. It is it is, you know, we met a long time ago, in person. And that's when I first heard about, the recode approach.

And I want to say that's 2017, met in Florida, and it was one of the first, first lectures on this approach on sort of this reversing kind of decline.

So a lot has happened between now and then. And, the race is on 17. And this is the most fascinating field of research that that I've seen, just because we're now using things, in our language, like immune health and diet and things that a lot of, a lot of neurologists and people just don't talk about.

So, first of all, let's talk about how the this precision medicine protocol was developed. It's kind of taken decades to even think about.

How Alzheimer's Works as a Network Problem

Right. It's a great point. So actually, I started up my lab at UCLA, in 1989. So and we the whole idea was to

understand the fundamental nature of neural cell death.

Why do neurons die? Why do you get these degenerative diseases? Why are they so common? And so we wanted ultimately to understand what's causing the problem.

And so over the years, we started to see a pattern, and we started to see that Alzheimer's is not what people were teaching. It's not just about amyloid or just about tau or just about type three diabetes or any of those simple theories.

This is really about a network insufficiency. It's really about looking at a, at a, an entire system together. And as you know, the big problem with and with classical medicine, unfortunately, is that it's now outdated.

People are looking at these. It's a literally like you're looking at the, the Titanic. Which is mainstream medicine, you know, has crashed into the iceberg of chronic illness.

And it's going down right in front of our eyes. And so we've really got to understand people as complex systems. And so we started to see that you could look at the signaling and how it was altered in people with Alzheimer's disease.

And you could actually trace molecular pathways into this beautiful system, this beautiful network. And you could see that it's insufficient. So as a as an example or as a comparison, imagine we take the United States of America, okay. That's a complex system.

There are all these things we take it as it currently is. And now we're going to ramp up inflation to 25%. We're now going to dramatically increase the toxicity.

The air pollution is now ten times worse. The toxins in the rivers ten times worse. Everything is ten times worse than it currently is. Then we start having some invaders coming into the country and starting to wage war on the country.

And then we deplete the support so that the companies can't do their jobs. We're not able to do commerce that's Alzheimer's. So in other words, it is an insufficiency of a complex network.

So now you can change the inputs to the network. The network starts to crumble. And so it can only work as a smaller network. And that's exactly what we're seeing in the synaptic networks of people who are developing Alzheimer's disease.

So synaptic network let's let's can can you define what that really means. Yeah that's a great point. So in your brain you've got this amazing supercomputer of course.

So you have about 100 billion neurons. And actually you have about ten times that many glial cells. So you all got also got support. You got the astro glia, you got the oligo dendro glia which wrap the myelin.

And then you got the microglia which are there that do the the immune part of this is gobble up pathogens and things like that but also secrete cytokines.

So you've got this beautiful network where each neuron makes on average about a thousand synapses or so. So a thousand or a little more. So so basically you've got almost a quadrillion synapses in your brain.

It's an incredibly powerful network that you have. So you've got tremendous computing power. And one of the major things that goes wrong in Alzheimer's is that the synapses start functioning less well and ultimately are lost.

And then ultimately, of course, the whole cell is lost, the neuron is lost. But classic studies by Professor Terry from many, many years ago, a famous neuropathologist, showed that what correlates best with the problems and the severity of Alzheimer's is synaptic loss.

So that is a fundamental problem. And so the question is why? Why are you losing these synapses? Is this something where something's killing them off?

And and the big surprise is that the amyloid that we make that's the hallmark of Alzheimer's is amyloid. And then tau is the second hallmark is actually a protective response to these various problems.

So you put pathogens in someone's brain. One of the things they do is make amyloid to cover and kill that pathogen. And professors Robert Moyer and Rudy Tanzi from Harvard showed a number of years ago that amyloid beta is actually an anti microbial peptide.

Now, the problem is of course, it's not just killing the microbes. It's also downsizing your brain. So it's doing both. But therefore when we want to treat this problem first of all we want to get in early of course.

But second of all we want not to just remove the amyloid. That is an overly simplistic idea that has not worked, as you know, with multiple approaches.

So what we want to do is determine why the amyloid is there. What caused the problem is this insulin resistance is this reduction of trophic factors. Is this specific pathogens?

Is this specific toxins. You want to determine it for each person and then you want to address those. That's why it's precision medicine because you're addressing the actual causes of the problem.

And it's given, as you know, the best results of any approach to date. So the simple point here is all we're doing with re code is identifying and in and addressing what's causing the problem.

You know, it's so complex. And as you said there's a bunch of different synapsis that's going on multiple, multiple ways that it's beyond what's right whether it's nutrition information toxicities etc., etc.. Yep.

In another segment, I'm speaking with Doctor Daniel Inman. We actually talked about how difficult it is to put words into numbers.

Measuring Cognitive Decline and Biomarkers

So to quantify literally, you know, yes. You know, Grandma and grandpa has memory loss, right. But what like what is there is there number associated in it, other technologies that look at that.

Because what we know from MRI's and from, from cats and stuff like that is so limited. So how do we quantify the actual memory loss that someone has? So we know we're kind of going in the right direction?

Yeah, I think it's a great point. And obviously Daniel's done a great job. With not only people with cognitive decline from Alzheimer's, but also things like, chronic traumatic encephalopathy and things like that.

So you're right. And, you know, this is the future we will be able to get better and better and better definition of what's actually causing the problem.

Right now. We can get a very good idea by looking at things like how much inflammation, what pathogens are there, what toxins are there. But for example, we don't have a good simple way to measure intra neuron or intra brain intracerebral, nerve growth factor right now.

We'd love to know what that is. I'd love to know what the, you know, what the intracerebral ADP and other important trophic factor is, we can get now a serum, Bdnf.

But that again, that may or may not be reflective of intracerebral BD enough. This is where we hoped exosomes were really going to be helpful. They haven't proven to be, at least early on.

They haven't proven to be as helpful as we had hoped. So there's a lot more to know. And of course, the genetics, have been they have given us that idea that really gives you an idea about the proclivities, but it doesn't tell you the current state.

So putting all these things together, we can really get an idea of the proclivities you have to begin with. Where do you stand? Why do you stand there?

And what is it that we need to do to get you back to normal? I wish there was just one test, right? One thing to just look at and boom, magically, we have this sort of, you know, Alzheimer's score. And this is not the case.

And and that'll happen it will. You think so? I think it will. But it'll be a nuanced score. It'll it'll basically be telling you it's a lot of little pieces to it, but it'll be a score that say, here's where you stand and here are all the contributors.

So you're right, it won't be one scaler. That's correct. People are terrified to talk about memory loss. You don't admit they have memory loss. And when a spouse or someone talks, cause them out and say you should see the doctor for it.

There's a lot of fear behind it because there's this idea and sense that why would I want to know something that I have no control over? And this is a shame, because there's a lot of systems of controls that people can establish.

Over or over Alzheimer's. And that is all numbers, but also neurodegenerative disorders, right? Yep. Now, what do you say to people who are just terrified of thinking, man, I have this memory loss. I want to see my doctor.

I don't want to be all doom and gloom. I just want to, you know, keep going and the way that life is going right now and hope for the best. What do you say to those people?

Yeah. You know, I'm so glad you brought that up. If you could get across one thing to everyone, it's that you do have control over this. People have been told it's hopeless and that is no longer the case.

That's what we're all showing now every day. And so what I recommend to everybody is think about the four phases you go through. And that's presymptomatic sky subjective cognitive impairment, which lasts about a decade, MCI mild cognitive impairment and then dementia full on the end, you know, end stage Alzheimer's.

Basically, if we can just get everyone to address the first two. So if you get on prevention, if you're 45 years of age or older, please get on prevention.

And if you don't do that, at least at the first sign of anything wrong, do not wait. And you can have tremendous control. And you should never you should never get demented.

No one should get demented. This should be a rare problem. It's because, just as you indicated, people worry about it. They think it's hopeless. They're told there's nothing in the armamentarium.

And so what do they do? They put it off and put it off and put it off. And as you know, and you've seen again and again, there are so many wonderful stories.

And they're just our recent trial showed this 84% of the people in the trial actually improved, didn't just slow the decline, they actually improve their scores.

And so people are seeing this all over. Of course, doctor Heather Sanderson, is showing improvements in her trial as well.

Hope, Prevention, and Early Intervention

Doctor Sharon Houseman colon showing improvements of her patients as well. And just, you know, your patients as well, and on and on and on. So now I think it's up to all of us.

We've got to go into all the different areas. What about the people who are very late in the trial? What what are about people, we're interested in this as a Sarah trial.

Severe Alzheimer's reversal attempt. Is there something you can do for someone who has a mark, a score of zero? We've seen people go now from 18 to 30, a perfect 30.

We've seen people go from 0 to 9. We've never seen anybody go from 0 to 30. And I look forward to that day. Right. So Moca score 160. It was really, really bad.

And 30 is perfect. So we're looking forward to going from really terrible to two. Really. Well, I'll tell you. And you know, as you're saying this, let me let me kind of explain to you a lot of the biggest success stories that we have had here at Texas Center for Lifestyle Medicine when it comes to decline.

And we're talking about smokers from like single digits to the upper 20s, okay. And so and this is a this is a part of my career I never ever thought I would ever talk about, which is dentistry.

One of the, one of the biggest moments is that when we started looking at and working with people in the head and neck space. So dentists, counties, allergists.

And and people like that, we're noticing that there's a lot of hidden, inflammable and immune networks within the skull. Right. And the skull, of course, cradles the brain that, that is not seen.

And, actually, earlier today, someone we've been working with someone for for three years, and it took me three years to convince them to see a biological dentist.

They found a very terrible amoeba that's actually in the sinuses. Yes. And, and, and I will tell you that, you know, post operatively after that was taken out and there's a collaboration between the dentist and Adonis and the EMT in the O. R..

He, he spoke clear sentences for the first time in three years. And so, yes. And this is, this is, you know, day one post-op and, and thankfully, they were able to do this without general anesthesia.

I did a lot of drainage, but it's incredible. And but I have a lot of these stories that there's a lot of things that are hidden that's outside of my specialty, which is internal medicine.

But when we start collaborating, people will start seeing things. And as it turns out, we went back into the data two years ago. There's this, there's a sleep study that was done in large sleep study done for the patient.

And after we diagnosed with sleep apnea. And this is really weird, spike. Right at the, if your portion of the frontal lobe and the neural, we didn't really know what it was, but now we know that's a manifestation of something that was brewing there.

Because when you're also seizure, we don't really know what it is. It's some sort of signal abnormality. The MRI didn't show anything. The cats can't even show anything.

Wow. How do we can't just play it off? Right. And so and it's really the dentist that really became the hero. But then it's like, no, this is, you know. Yeah.

Right now, is this an account, the amoeba or an a Gloria or something different? No, this is something different. Actually. It's been, under analysis right now.

So this is a organism that's similar to. It's in the Gloria. So what we have found out is that it's not just that it's there, Sparky. It's within the oral microbiomes of there's all these different pathogens that are in there that, that now we're going to the dental community, and the dental community is reaching out to us because they're seeing these people who have underlying dementia, and they're cleaning the airways, and we're seeing that they need entire

neurocognitive workup.

So I think the collaboration in between and I use dentists and EMT is just one of the, one of the many people to collaborate with. But the collaboration, I think, is really key to tease out, these intricacies so we can all, you know, work together.

But I feel that there's so many things that I was not taught in med school residency. Right. And I just thought, oh, you know, you know, Alzheimer's disease of too much amyloid, being in the brain, not realizing it's an immune response is supposed to be there.

And how do we not have the ability to create amyloid? We might be dead, you know, much earlier on. Right. And so and so this is my my moment for me, for me starting when I met you in 2017, in Florida.

So now and that progression just gotten bigger and bigger because there's so many things that are out there that's contributing to it. And you know, one thing I really got to say about the Rico program is Rico program looks at everything.

It looks at everything from every point of view. And it really opens my view of what brain health really is. You know, it's the brain, it's the body, it's the skull.

It's the it's the spinal alignment, the connections.

Late-Stage Cases, Dentistry, and Hidden Infections

It's it's includes the liver. And the kidney is such a beautiful thing. And the brain is is the transmission of the body. We're kind of orchestrates everything.

And it's it's a beautiful thing to see people improve. But I think it's more beautiful when everyone can stand by and look at this patient and really appreciate what transformation they really have. And then a lot of times a week or two weeks later, they physically look different.

They look younger, wrinkles starts to start to appear to go away. And it's very transformative to see. Yeah. And you know, this is bringing together, you know, traditional Chinese medicine with modern medicine to say, look, you know what?

What can we see here that's actually driving? And so the thing that excites me is that this comes straight from the test tube, all the experiments we did over 30 years, they put you in this direction so that you can't ignore all these different things that all play on that network of neuroplasticity.

And if you ignore them, you're not going to get best outcomes. I mean, just as you were pointing out with your patient, with a Larry, a like organism, you got to get at that.

And the same thing with t identical, and PGR gingival and all these different things that can all play on that neuroplasticity. I hope that ultimately, it'll be simpler and we'll be able to do some very simple tests.

But right now you have to remember this is a terminal illness. So when people begin to have cognitive decline, only one of two things will happen. Either you'll figure out what it is and you'll help them and get them sustainably better, or they will die.

And so you really want to pull out all the stops, look at all the things, find out what's driving the problem, and then address those with a precision medicine sort of approach.

Absolutely. And I think what's beautiful about all this, it's really requires a lot of people, to be in a place where it's really patient centered medicine.

I think this is how all medicine should be really practice, right, with the with the integrative approach. But but let's talk about something that no one really likes to talk about, which is look at the naysayers, because I feel like there's a lot of people that look at the stuff that I do and the stuff you do.

Well, this is not, you know, a, a pharmaceutical trial any way, shape or form is not designed to be so and so. There is a lot of support from the medical community, and there's a lot of naysayers from the medical community, like, how do you how do you navigate that? Yes.

If it's okay, let me just I want to address one thing that you said just a minute ago before, and then we'll go into the naysayers. Yeah. But when I was coming through, illness was, you know, I'm much older than you are. When I was coming through, way back in the 70s and 80s, what happened was there were all these specialists, we say, okay, we're sending you to a cardiologist because you have a heart problem.

Okay? Now we're sending you to a renal doctor because you have a kidney problem. Now we're sending to them. Now, what's interesting is you still need all the different people, but now these people are all addressing one problem.

It has all these different fingers coming out from this problem. So okay, you have cognitive decline. We need to know what's going on with your heart and your liver and your gut and your sinuses and all these things.

So it's a really it's a completely different way of thinking about medicine. But looking at these systems is critical. So apologies. Now let's go. Let's go back to your point about the naysayers.

Yeah. And I have to be honest, I I've been surprised at how negative the naysayers have been. But I, you know, I understand that the idea is, you know, if you consider yourself an expert and you're doing something the old fashioned way, the last thing you want is for someone to come along and say, no, you you have to change the way you're doing that.

No, you say, wait a minute, I'm the expert. Yeah. I don't have to change the way I'm going after amyloid, because that's what experts do. I'm going to, you know, go to meetings on amyloid.

I get grants on amyloid. I talk about amyloid, I study amyloid. So if you're telling me that that's not the answer here, I don't want to hear that. And so, you know, it's kind of funny.

It's sad for the patients. However, but you also alluded to the trials. Yes. We'd love to have a, you know, a phase three trial for everything, but that's that costs many, many millions of dollars.

Some of these, you know, some of these trials are 50, $100 million dollars for a phase three trial. So what we really want to do is start with the science.

How does it actually work? Get some preliminary data. Look at anecdotally, can we actually make people better? I thought when we showed for the first time and published back in 2014, hey, here are people who are reversing their cognitive decline and then, hey, they're sustaining it.

People would say, oh, okay, great. Let's let's try this. Let's see if we can make it better and better. But the response was, we just don't believe it.

Well, okay. Talk to the patients. Look at the data whatever you like. So we've just we've kind of taken next step. Next step. We've had ten. Then we had ten with better documentation.

Then we had 100. Anecdotally then we just published just recently the trial in Journal of Alzheimer's Disease. And this is where we took a proof of concept trial, 25 people who had well documented MCI or dementia, and showed that we could make them better.

And 84% of them improve their scores. And so now we're taking the next step, which is a randomized controlled

trial, and a very, very, honored to be doing this with some absolutely outstanding physicians with at six different sites.

So we're doing this with Doctor Anne Hathaway, here in California, doctor Cat Toups in the East Bay, doctor Christine Burke, in Sacramento, Doctor Craig Talia in Miami, Doctor David Hosty in Nashville, and Doctor Nate Bergman in Cleveland.

So very excited to be working with six absolutely outstanding physicians. And that's going to start, later on this year. That's amazing. And then where do you see this going?

I mean, you have you're collecting this data like what would be like the best case scenario for getting people to understand that there's a reversibility to a lot of the symptoms of cognitive decline, getting people to say they hope is okay.

We just have to have a very standardized approach to it. Like how where does that supposed to go in the next, you know, five years or so? It's a great point.

So there are different pieces to this immediately. The important thing is public health. So what we want to do is reduce the global burden of dementia.

This is a huge and this is $1 trillion problem globally that affects so many families and destroys families and destroys everything that families have done together.

It's really sad. So that's the key thing to get it out there, to get it into, into standard of care.

Patient-Centered Medicine and Scientific Pushback

Because as you know, the standard of care today, you go into a memory aging center. They're the experts. They take a small data set, they give you a drug and they tell you you're going to die.

It's a very, very sad. And it's really a now an outdated approach. So the first thing is public health. The second thing then is to begin to simplify.

Can we do things that will make it so that it's easier for more people to do this? And then the third thing is to now, now make this better science and more.

Can we now determine what are the critical variables? My my hope is in the long run will have equations. We currently use a computer based algorithm, but over time as we collect data we'll be able to put numbers on there.

So we can say to someone, 27% of your cognitive decline is due to this infection in your sinuses, 28% is due to your insulin resistance. You know, 15% is due to that.

And then we'll be able to, to adjust each of these things and see people get back to normal and then stay normal. We have people now who have been on the protocol for over ten years.

The first people started 2012 and they've sustained their improvements, but we have others that have often going off where they haven't sustained their improvement.

So we want to make it so that everyone can sustain their improvements. And we can tell them, here's what's actually causing it. Then, of course. So, to be able to adapt this for all of the other neurodegenerative illnesses.

And we've started, as you know, with the, the what we call the Arc project, arc just like two by two by two animals. The idea is to look with small numbers of patients with a larger, a deeper dive, larger data sets, and see if we can also reverse those.

And actually we started the first patients, in Texas. And we have some dark adaptation with people with macular degeneration showing that we can normalize their dark adaptation, improving their symptoms from macular degeneration.

So very excited about that. I want to be able to share the same thing with ALS, with frontotemporal dementia, with Lewy body disease, etc.. So and we've already seen good results with Lewy body disease, with our protocol.

But I think all of these can be adapted to get best outcomes from these other currently untreatable illnesses. So but the question is why? Because I feel like in the medical community, we have diagnosis codes that are very secular when it comes to a lot of brain health.

Right? Well, not just brain health, mental health as well. You know, you have like anxiety in the of ADHD, etc., etc.. And in the because of these diagnoses, there's stigma to them as well.

So a lot of people in the public, I feel like they want to know exactly what they have for an exact solution. What about brain health? How much overlap?

Okay. Yeah. And so what. So I guess my my question to you is yes, we know that in Alzheimer's disease there's there's tau protein is amyloid. That's the hallmark feature. But it's immune response.

What about all these other neurodegenerative diseases. What are the commonalities there and what are the differences. That's such a good point. You know so here's the interesting thing.

And this is now, you know, at the conceptual level, how can we take this to the clinic and and get improved outcomes. Because these as you know, these are all virtually all been untreatable illnesses.

So the concept here is the same for all of them. The idea is neurodegenerative disease is due to network insufficiencies. So you have a network that's important for neuroplasticity.

You have a network that is important for motor control. And that network is what's being what is what's failing in Parkinson's disease. You have a network that's it's important for motor power.

That's what's failing. And ALS of course support of your macula, that's another one. That's what's failing in macular degeneration PSP progressive super nuclear palsy is another one.

That one again, it's a cousin of Parkinson's but unfortunately untreatable with with typical Parkinson's drugs. Again, it has the same sort of Achilles heel which is energy related as does Parkinson's.

So for each of these you're looking at a an insufficiency of a different neural subnetwork. And so we can define that insufficiency. And it's really interesting because each of these subnetworks has its own Achilles heel.

As an example, anything that causes mitochondrial complex one to fail is going to increase your risk for Parkinson's or Parkinson's related syndromes like PSP, or MSA, things like that.

On the other hand, you look at Alzheimer's and we know the common things insulin resistance, changes in oral microbiome, as you pointed out, sleep apnea, things like that, changes in oxygenation.

That's all part of the energetics that are critical for this neuroplasticity. So each of these subnetworks is different. Interestingly, in ALS it's really about glutamate uptake because you've got this severe this cytotoxicity.

As Professor Rothstein showed many years ago from Johns Hopkins. So each of these things has a different set of, of problems. And a different Achilles heel.

And we can therefore define the network for each of these and determine what's going wrong and address those things. But so each of these Achilles heels is really depending on what set of network malfunctions I guess there

are within the brain, but one cause can affect any of these networks.

Right, exactly.

Clinical Trials and the Future of Recode

And it's just, you know, some some people again, be partly because of their genetics, you know, two people will go out and get exposed to, you know, to, to, you know, air pollution, and insulin resistance.

One of them will develop Alzheimer's, the other one will develop Parkinson's. So, yes, there are there are these various genetic predispositions as well.

But these things all contribute to network insufficiencies. You know, another part of this of what we actually talked about, headaches in general. And one of the things that we talked about was there's so many different types of headaches, cluster headaches, migraines, etc., etc..

And then the conclusion was, well, you know, one thing can trigger any of these headaches. They just have pretty names to them based on the diagnostic criteria.

So we're talking about the same thing in neurodegenerative disease. Right. Yeah. And when I, when I practiced neurology I treated a lot of people with headaches.

And well, what we would always do is, is this a tension headache? Is this a migraine? You know, is this something different? Is a cluster headache. What is it?

Define what kind of headache it is and give the appropriate drug for that kind of headache. But we virtually never knew. Well. So why are you getting a headache?

You know what's going on here? And so, you know, now, again, this is 21st century medicine. It's not about what it is. What's the diagnosis? It's about why it is.

What are all the contributors is, you know, do you have, an amoeba in your sinuses? Do you have, you know, do you have, oral problems? Do you have something else?

Right. And, you probably don't have a motrin deficiency or something like that. Exactly. Causing it. Right? Yeah. So, so a lot of the things that, we were taught, especially in medical school, are sort of these bandaid things that, yes, we're going to take care of the patients and make them feel better, put them on the back.

But, you know, looking at the root causes a whole different, you know, phenomenon, not just in neurodegenerative diseases, but all diseases and, and disorders.

Right. You know, and another thing that I really struggle with, and you may not know this, but ever since 2017, majority of what I do on the clinical basis now is pretty much like neurology at this point, because of the success of what, has occurred, when the last five years, in terms of what we've learned from Recode, in terms of, you know, everything and the collaboration and the practice, my struggle, and I want you to I specifically want one of you on the summit to ask you this phenomenon.

We noticed that, a lot of times when the patient actually gets better, they become more aware and therefore more depressed and to the family, it may seem that they're more withdrawn, or maybe they seem more quote unquote demented, but in reality, of neuro contesting, they're actually testing better.

Can you can you speak to this phenomenon? It's a great point. And I know in Marama, the assisted living facility, Heather had mentioned a patient as well, who clearly was better cognitively, but then started ruminating more and saying, gee, you know what?

What am I doing here in the world and that sort of thing. So here's the thing. There are a couple of important pieces to this. Yes, you you've made this improvement in their in their performance.

They are more aware, as you mentioned, but there was some interesting work from Yale a number of years ago, from a professor who showed that we've always think of depression as being a depletion in serotonin.

But her argument was, you can argue equally well that it can be an overabundance of acetylcholine. So we're pushing on these people to have greater cholinergic tone, because that is so critical for memory.

So we're giving them things like, alpha GPC and Sida, choline, and even sometimes things, like Hooper's DNA and things like that. We are improving their cholinergic tone.

One of the unfortunate side effects of that is you may get someone who feels a little depressed because of that improve cholinergic tone, even though they are performing better.

So what you want to do is now you've got to bring the other corollary systems up, you know, the complementary systems. You've got to make sure their dopamine is good enough and make sure their norepinephrine is good enough, as well.

And so that's part of it. The other thing is, yes, you do get some insight. And also you're, you know, you're struggling to to improve even more. So I think the important point is don't get discouraged by that.

You're clearly on the right track. You just haven't arrived at the destination yet. And so the thing that I always tell them, I know people get get tired of hearing it, I always say to all the patients when I'm talking to them, please keep optimizing because that's where you're going to get the best outcomes in the long run.

You know, good example. Someone gets some improvement. Now they backslide a little bit and we find, something's been missed. Now you you improve that.

And now they get even better improvement. Or someone says, well I'm not complying as well as I used to because, you know, I've gotten better. That's all I need for now.

Well no, keep on going. You may be able to get better and better and better. So again, we're defining the neurochemistry of synaptic function so that we can make it better.

And you may have little hiccups along the way like a little backslide or compliance issue or a little depression. And just keep on keep on optimizing.

And you'll get really good results right now.

Extending the Model to Other Neurodegenerative Diseases

And I do want to address that, you know, shout out to a lot of the caregivers listening to this, because it is a tough job, trying to manage your own life plus your loved one.

It is very, very tough. And we recognize that. Right. And so, and the reason I asked you about, you know, when people get better or they start getting all depressed is that the caregivers suffer a lot of guilt, looking through that process.

But yes, yes, the memory could be a little better and the function could be a little better, but, you know, he or she is more withdrawn. Seems to not talk as much, not be more interactive.

And there's a lot of guilt that really comes, when when people, you know, start to get a better process. But I agree with you. Is that to, like, keep going.

And then the other part I think is there's a fear of false hope, not just from the caregivers, but some of the patients themselves, too. Right. And as the patients become more aware of their surroundings, of the people that are there and they become more kind of the understanding, the patients themselves have a guilt because they, the patients themselves feel like they're a burden for their family and their caregivers.

So it's like a bidirectional, you know, guilt thing that a lot of times we have to just, you know, counsel and overcome and that's that's, you know, it's great that we're seeing people get better.

But for me, that's probably the toughest part to, to, to, to, to help manage and help people, you know, meet the expectations, you know, and I would argue that there is a lot of false hopelessness.

I mean, yes, I understand people always say false hope. The term reality is there is now a lot of false hopelessness in cognitive decline. So, you know, you go on to a website for a foundation or you go to a memory and aging center, or you go to an expert and they all say the same thing.

There's nothing that can be done. Forget it. It's hopeless. And that's no longer true. So I would argue that false hopelessness is a problem that is on the increase.

I also you mentioned the caregivers. I think that's an important point. There's such an important part of all this. There's a very interesting paper that's now in press in the Journal of Alzheimer's Disease that will be coming out, and it's talking about the health of the caregivers and different responses of the caregivers.

Some would get angry, you know, some would get verbally abusive. You know, some would just kind of put kind of, everything would be shoved under the rug.

They tend to get different types of health problems. So we have to take good care of the caregivers, as well as taking good care of those who are cared for.

Yeah, absolutely. I 100% agree with that because some of our, more severe cases are more difficult cases. Are those people actually without any caregivers?

Yeah. That unfortunately sort of part of the system. And unfortunately, it's really hard to manage those people as well. And, and for those caregivers listening to this, if you are supporting someone through any sort of brain issues whatsoever, they're lucky to have, you got to pay yourself on the back at least once, maybe do it now, you know, and, and understand that, you know, through love, everything can really be accomplished.

But I agree with you, doctor. And the false hopelessness is much, much bigger. I think, and especially what we see. And I'll, I'll give you this this one story is we have your biggest fan, one of my patients.

His name is Carl. He says hello. He knows we're talking today. So. And and Carl, has multiple copies of your book, The End of Alzheimer's. Multiple. Give it to the church members.

He goes on these walks and, and and a couple years back, he was in an Alzheimer's board, walk. And, the staff members respectfully told him not to pass out anything, that's related to, like, the end of Alzheimer's.

And I given this false hope, and I'm just, like, cover my head on, like, are you serious, Carl? Like, you went out with your church to go on the Alzheimer's walk, and they're not going to let you, like, at least provide some hope for people.

And it it drove me a little insane. Just kind of hearing that. But that's the that's sort of the delicate situations that we're seeing right now in the public of this false hopelessness.

That's there. Right. Well, and what's interesting is we all want this to be blessed. Anything that's new, if you get a new treatment for cancer, a new treatment for Alzheimer's, a new treatment for lupus, we want it to be blessed by the experts.

And we always think that the experts are completely objective. But the experts are, you know, they're they're being paid by drug companies. They know they're they have their own, nonobjective reasons.

And so we're waiting for blessing. And so, you know, you hear you go to this walk and they're like, well, you know, here's what we believe. Because this new approach, even though, you know, like read the papers, we've published this, it's in peer reviewed journals.

It's happening. Their argument is it hasn't been blessed yet by us and therefore we can't talk about it, which is sad because it is offering false hopelessness.

Right. And I think that that validation is what people are kind of, you know, searching for and coming around. But I, you know, and I want this summit to really be an educational experience for people and having people experience, the process themselves and decide for yourselves.

Right. And in the last, the last thing that I want to go over with you is, what I've seen, ever since the global pandemic hit. So through the coronavirus, I learned a lot about sharing.

Depression, Caregivers, and False Hopelessness

I learned a, the the amount of brain issues, long haulers and stuff like that, you know, almost called acute onset of Alzheimer's in some people. It's it's startling.

And what we're seeing is that if you have something like Mark on the time Alzheimer's, that that that gets worse over 40 years, we're seeing that gets worse over three months now with with coronavirus.

Right. And so what what do you think you gleaned from, the global pandemic and how affects the brain right now? And how does that really affect your view of, of what, brain brain health is?

It's a great point. And as you said, you know, we're seeing brain fog everywhere now, so many young people. And so we're really having to take the same principles that we've used for Alzheimer's and move it basically 20 years earlier for everyone who has brain fog.

Riley that evaluated, please find out what are your contributors. Because as you know, many of the same things. And here's what's the interesting parallel.

There's a lot of parallel between Alzheimer's, and Covid 19. Although one is clearly a viral illness and one is much more than just a viral illness, as we were talking about.

It's it's many things that are have to do with this net, but they have a number of things in common because, you know, for example, you low vitamin D, you're at risk for worse.

Covid, Alzheimer's, you have low estradiol, obesity, insulin resistance. ApoE4 is another one. All these things just go right down. But what they have in common is in Covid 19, you have your your innate immune system activated.

So you have this inflammation, but your adaptive system has not been able to clear. You're supposed to be able to hand this off. Now the innate system goes down.

Your adaptive system takes over. You. Now get rid of all that inflammation. You have a very specific approach cellular humoral response. And now you clear the virus. Everything's good.

But the problem is because of the strategies of the virus itself, your own genetic background and your own biochemical background, you're in a state in which your innate system is still on, and your adaptive system is not a clearing, so you die of cytokine storm.

This just keeps boom in that system. Now in Alzheimer's, what happens is you also have the innate system active

and you have it cleared, whatever the problems are.

And of course it can be mycotoxins and all these other things. Whatever it is, you haven't cleared it. So therefore you don't die of cytokine storm because it's a chronic illness.

You die of cytokine drizzle. You just have this chronic, ongoing, ongoing, innate system activation. And so our job is to find why that is, get rid of that and then bring up that adaptive system, support the body.

And that's where there's a lot of relation between what happens in Covid 19 and what happens in Alzheimer's disease. But I agree with you. It's really making all of us even more aware of optimal brain health, the cytokine storm versus cytokine drizzle.

I'm totally going to use that. It's it's a beautiful way of of looking at it and really helps me really understand this this this concept. Right. Yeah.

When, whenever I, we have our patients with any sort of neurocognitive issues and they actually get, the coronavirus, there is sort of this temporary, you know, decline.

Right? Yes. And then when they get over it, they come back, some people come back faster than others, right? Yes. And so and I feel like that's the same way with just people developing disease and disorders.

There are things outside of genetics that, are responsible for how people are reacting to, whether it's viruses, whether it's toxins and things that cause I'm nervous and stuff like that.

And so, you know, we label this, you know, epigenetics, you know, beyond the genetics and surrounding genetics, what do you think are the top epigenetic factors and lifestyle factors?

And, reducing either the risk of Alzheimer's or improving cognitive health that people listening right now can just start doing at home, like right now, some simple things.

Yeah. So you bring up a really good point. So let me just address what you said earlier first, and that is this whole issue of getting worse. So you have someone who's who's now on the upswing.

They're doing better with their cognition. They're they're beginning to heal that underlying problem, whether it's SimCity or or Alzheimer's. They're beginning to improve.

And now, boom, they suddenly get Covid 19. And they usually take a step back. And again, because their innate system is now reactivated, their adaptive system hasn't cleared yet.

And so you've got this. You know, it again, it it supports what we're all saying about Alzheimer's disease that that's part of the problem here. So we now want to help them get over this.

And yes, they'll often take a little step back. But then in the long run they'll start coming up again. And that happens with any insult. I see it with kidney stones, with gallstones, with operations, with a lot of anesthesia, with a new infection, with new exposure to toxins.

As you know, all these things, you take a step back. And so now you got to figure that out. And, and Julie G, for example, has talked about her own story where she was doing really well.

And then it turned out that she had undiagnosed Birbiglia. So to come back to your point, though, about what are the things that we can do today? The most important thing, is to get evaluated because this is, as you said, this is a complex situation.

And so you want to know what are the contributors. But I understand, you know, a lot of people, their their their local doctor may not be doing this. You can always get a Cognos copy.

So I recommend everybody please get a Cognos copy. We all know to get colonoscopy when we turn 50, if you're 45 or over or if you've had Covid, please get a Cognos copy, see where you stand.

COVID, Brain Fog, and Epigenetic Lifestyle Factors

And, and be able to address those things. And it starts with the basic seven things which are diet. You know, you want to have a, a mildly ketone, a plant rich, mildly ketogenic diet high in phytonutrients, you know, make sure that it's anti-inflammatory, all those important things, hopefully omitting, grains, dairy, sugars, things like that.

And then, you know, any sort of gluten or dairy, that sort of thing, and then exercise. And boy, there's there's great stuff on exercise that wasn't around even a few years ago.

Things like, these are these, K2 bands that have been very helpful and improve muscle mass, improving therefore, your insulin sensitivity. What exercise with oxygen therapy.

Very helpful, especially for people who have vascular components. So lots of important things, both on the aerobic side and on the strength training side.

And then third one is sleep. And again you could write books on sleep. And of course, professor Matthew Walker has done that as, as has Arianna Huffington and others.

Tremendous amount that you can do to make sure that you've got the right. And of course, wearables are going to help so many of us to see the problems before they ever come to see whether you have a low heart rate variability or whether you have poor oxygenation at night, that's one of my biggest concerns.

Low oxygenation at night affects so many people and of all people, I just found out recently it was affecting me. I didn't know this for years. And so okay, now I know that this has to be addressed as well, to avoid problems.

And so sleep is the third thing. Stress management stress a huge issue. You just look at people with high stress and that's been, well documented. They have smaller brains.

So, you know, let's let's reduce that problem. And number five is brain training. Professor Mike, Nick, started this years ago. Fantastic. Brain HQ has done well for so many of our patients.

And then sixth thing is targeted supplementation all sorts of things. And people say, well, you know, supplements aren't a cure for Alzheimer's. Of course they're not. Nobody's saying they are, hopefully.

But getting their optimal biochemistry for your brain is huge. And they can be helpful, no question about it. And the armamentarium is huge. Being able to target the right ones for the right person.

Very helpful and then the final thing, of course, is detox. And I was never taught as a neurologist, that these toxins were causing cognitive decline that we ultimately call Alzheimer's disease.

I didn't know about trickle theses or glo toxin or oak or toxin a, I didn't know that air pollution was an important one. We knew that mercury could be a problem, but we didn't realize how commonly this could be associated with Alzheimer's disease.

We didn't realize about the, about the organics, the glyphosate and tali leads and benzene and formaldehyde and all these things. So now we are getting a much a better kind of crystallized view of what it is that's actually driving for each person.

So I recommend everyone please get checked out. Please. There's so many things you can do for yourself and just go by your own outcomes. If things are going well, great.

Keep optimizing. If things are not going well, please consult with your practitioner because he or she can help you.

Now let's dive specifically into the Rico program.

So what's it like of being a person going through the, Rico program? What's what's the journey look like? Yeah. So it's it's in which straightforward and trying to make it more straightforward.

So you simply you can go on you can go on my cog Moscow B. Com or Doctor medicine.com or Apollo Health Co, any of those. And you can go and you can sign up for Recode.

It's simple. We do Recode is for reversal of cognitive decline. And pre code is for prevention of cognitive decline. So if you're truly scoring really well you have no complaints whatsoever.

Then just do prevention. But for anyone who has any complaints or who's concerned or has it running in their family, please do recode. You can actually then get your labs directly from online and then you can have and then you'll have access to.

We've trained over 2000 physicians, obviously showing you've done a great job with all the people who have come. Your way so that then we can link with people where you are.

We have people all over the world, in ten different countries and all over the US who, you know, who are, doing this, successfully and getting people to improve.

And of course, we're all interested in the same thing, which is continuing to evolve and get better and better and better as we learn more and more. I'm I'm never saying this is the final answer.

We're always looking to get better and better and better outcomes. So that's Recode. Pretty simple. Go online, get your blood tests, go to a practitioner who knows what he or she is doing and get a good outcome.

Now that's amazing. And what's really exciting about all of this is that for those people who go on record, you're using that data to compile even more data for the future.

So you can be predictive of what factors cause what and how much. And that that's the beautiful part behind it, right? It's not just about, you know, you individually as a person going through it.

It's also about the contribution factor to the greater good and to the study of medicine, study of of brain health. What is the perfect age or is there a perfect age to start either Rico or or pre-Code?

Yeah. If for anyone who's 45 or over, I would recommend please get on as early as possible. The farther you go, the harder it is to get a complete reversal back to normal, as you know.

So please get on early if possible. Now, one caveat here 95% of people who develop Alzheimer's have, as you know, sporadic Alzheimer's, where you have genes that increase your risk, like ApoE4, or various alleles that increase your risk, like ApoE4, and others trim too.

And there there are many of these, but that 5% that has true familial Alzheimer's, where you have a mutation in AP Prestel one or Presnell and two, they can start earlier.

They may get it. Some of these families get it in their 20s.

How Recode Works and Who Should Start

And so whenever if you are truly in one of those rare families that has it go in 15 years before the onset of the symptoms in your family. So if everybody's getting this when they're in their mid 30s, go in and if they're getting in their mid 40s building when you're 30.

Now we are dealing with just a few people who have familial Alzheimer's, and it's in their family. And we're seeing

some good results so far. But we don't know yet.

It's it's a different thing because the genes there have such a powerful effect. Wow. That's amazing. So to think about this from a preventative standpoint and understanding that the early start, the better the outcomes may be, is is truly inspiring.

And I want people to listen to this, is that there is, a fear behind neurocognitive disorders like Alzheimer's disease. But if there's something that's out there, that can either prevent it or that can reverse some symptoms of it, you got to be able to just just attach on to that and understand at least what it's what it's about.

And, you know, not all doctors understand this process. And you're right. I didn't learn that through med school, you know, and not all people are going to be want to be part of this process, but that's okay.

Sometimes you just have to advocate for yourself. And yeah, I really want to thank you for writing the first book, The End of Alzheimer's. Which is one thing that a lot of patients come in, they have things highlighted and we kind of go through. It's actually a lot of fun.

And we actually at Texts Center for Lifestyle Medicine, I did an entire book review of your book in online format mode that we have for our patients, on our, on our memory programs, so they know exactly what each thing is talking about.

And it's becoming, it was becoming wonderful. So for for and people are listening to this. If you if you want a full review of Doctor Peterson's protocol, just go to WW WTC university.com.

Scroll down to the root causes of memory loss. And I go through, my review of the, of the entire, book of the End of Alzheimer's and what each of the sections is that we can't really cover on this short segment of our interview as well.

But thank you for coming on. I mean, this has been an incredible talk. Leaves me with a lot of hope, and I hope people listening to this are going to be developing that that hope for mental ality of the other things that we can do right now, for yourself, for your brain.

So thank you for coming on. Yeah. Thank you. Chang. And, you know, this is the time for updating of all this is you mentioned 2017. So that was five years ago.

So we're now looking at a second, a second edition of this with updating everything there is, you know, there are tests possible. Tao a beta 42 that weren't available before into, you know, in in the blood isn't instead of just the CSF.

But epigenetics tests all these things that weren't available even a few years ago. So we'll be able to do better and better with following people and get better and better outcomes.

So thanks again. Chang.