Introduction and Guest Welcome

Welcome to the Parkinson's Solutions Summit. I'm your host, Dr. Ken Sharlin. Hope you've been enjoying all of the wonderful interviews so far. I'm very excited to introduce a special guest today, Dr.

Dale Bredesen. I've known Dr. Bredesen now probably since about 2016 is when we first met, when I had the honor of being in one of the very first courses he put on for the Recode protocol for Alzheimer's disease.

And I know we'll be talking about that as well. But today, what we're going to focus on is a condition called Lewy body dementia or dementia, with Lewy bodies and how that's similar, different from Parkinson's disease and other related disorders.

And then ultimately whether it might make sense to apply the principles of the Recode protocol in patients who have this really this family of degenerative disorders.

So without further ado, welcome, Dr. Bredesen I'm so glad you could join us today. Great to be with you, Dr. Sharlin, and thanks for having me on. Well, Dr. Bredesen, like I said, we met in 2016.

Dr. Bredesen's Background and Research Origins

I think at that point in time you'd published one or two papers related to reversal of cognitive decline. We're certainly in the early stages. Was before your association with with Apollo Health and it's just been so many changes that have happened since that time, I think for you and really for everyone thinking about Alzheimer's prevention, neurodegenerative disease prevention and reversal of some of the symptoms and signs of these diseases to really put people in a much better trajectory.

I wonder if you would tell the listeners and viewers who may not be as familiar with you a little bit about yourself, your background. You're a neurologist, obviously a world class researcher.

How how did this all sort of evolve and and, you know, kind of where things today for you? Yeah, Thanks for asking. So, you know as an undergraduate at Caltech and worked at MIT, actually while I was there as well, I was interested in chemistry and got interested in the brain.

I read a book called The Machinery of the Brain by Dean Wooldridge, and I got very excited about the relationship between computers and and human brains and how does this actually all work?

And then of course went off to medical school at Duke, and it became clear pretty quickly that we neurologists have a lot of people where we're not very helpful to them therapeutically.

And of course that was kind of disappointing and I could see that, my gosh, if whether you come in with Lewy Body disease or you come in with ALS or you come in with Alzheimer's as neurologists, we really couldn't do much.

And I actually thought about doing something else, like being a neurosurgeon or being a neuropathologist. But I was very interested in brain disease. And so came out to UCSF actually for residency and then ultimately fellowship and starting on the faculty.

And the idea was, okay, we can't you know, this was now back in the 1980s, we didn't have, as you well know, we didn't have something to offer these people like, hey, if you've got Alzheimer's, if you've got Lewy body, just take this pill, you're going to be better.

So I thought, okay, the best thing to do is go into the research lab. And I said, I had a research group. We worked

together for 30 years, published over 230 papers.

And looking at what is the fundamental nature of the neurodegenerative process, what's actually causing your brain to regenerate. And as you know, can there have been all sorts of theories.

It's it's about type three diabetes, it's about herpes simplex, it's about prions, it's about amyloid, it's about Tao, it's about mitochondrial damage is on and on and on.

But none of these ideas that people had had ever led to any sort of significant treatment. And so we were kind of going along and we were looking at the signaling that happens with amyloid precursor protein.

AP, which is the parent of the amyloid that collects in Alzheimer's. And we started noticing something really interesting that this thing looks like a molecular switch.

You can drive it in one direction where it actually supports making synapses and maintaining synapses. Or you can take that same molecule. And by interacting with different ligands or under different circumstances, you can drive it in the opposite direction.

So it's literally signaling pull back, pull back, pull back. And, you know, the analogy to me was really interesting. What later came as the pandemic in 2020, we were all told shelter in place and socially distance and don't go to work and be very careful.

And of course it was all about pull back, pull back, pull back. And our country went into a recession. As everyone knows, the same sort of thing that we see happen with this AP signaling.

Now, interestingly, it's on the downsizing side of AP, where you make the amyloid in on the upsizing. You don't make the amyloid. In fact, you make sap alpha, which supports Synapse production and synapse maintenance.

So we thought, wow, is it could it be that this signaling event is related to what we're seeing in Alzheimer's and in fact, what happens is in Alzheimer's, you're literally going from a growth and maintenance mode in your brain and in your neuroplasticity to a protective downsizing mode.

The amyloid, as you well know, is a protectant.

Amyloid Signaling, Functional Medicine, and Neurodegeneration

It is there to sequester or, in fact, as you know, in these plaques, you often see herpes simplex you sequester viruses, you sequester bacteria, you sequester fungi, and you kill them.

It's an anti-microbial sequester. And basically and so we thought, okay, we need to develop drugs that drive you toward the growth side. But of course, as we did that and we actually identified several drugs and we actually when it started a trial in Australia for one of them.

But I realized very quickly that this is not going to be good enough to just have one drug because there are all sorts of signals coming into the brain.

So if we're really going to change that equation, then we're going to have to look at all the different things. And so that, of course, got me interested in functional medicine.

And I really didn't know about functional medicine before that. And I thought, This is interesting. So okay, we need to look at what's the essence of this.

And what we found is that the two major players and I think these are important certainly in Lewy Body and probably in Parkinson's as well, but certainly in Alzheimer's is, you know, it's about energetics and there are dozens of things feeding into that mitochondrial function, you know, blood flow and oxygenation and things like that.

And then inflammation, the activation of the innate immune system, very much like the big issue in COVID. And so the idea is you need to change where you're in a situation where the energy is too low and the inflammation is too high.

You need to change that equation, which is exactly what the approach that we all take does. And so we realized, okay, these different neurodegenerative diseases, whether you're talking about Parkinson's or Lewy Body or MSA or Alzheimer, whatever, they're really about supply and demand and have a supply that's too low and or a demand that is too high.

And so we need to, for each person, identify all these things that are driving this. If your blood flow is not good, if you have sleep apnea, if you're dropping your oxygen saturation too much, if you've got a change in your oral microbiome that's giving you inflammation in your brain, you know, dozens and dozens of these things, you're going to be on the wrong side of the equation.

And over time, you will have a degenerative event. But the great news is we should now be able to take the unique biochemistry and genetics of each of these diseases, be they MSA or Lewy body or whatever.

Look at the Achilles heel is, you know, the Achilles heel in those synuclein apathy has largely been complex. One of the mitochondria with Alzheimer's, it's all about neuroplasticity and mitochondrial function and things like that.

With ALS, it appears to be more about the ability to re uptake glutamate, which is otherwise a cytotoxic you're, you know, driving them. So it has a different, a different Achilles heel.

And so for each one, when we make a diagnosis, it's really about telling you what are the likely things that are driving this particular neural sub network to have a poor supply to demand ratio.

And that really gives us a lot of insight into what do you measure, how do you treat it, how do you prevent it? And so very excited. As I as I told you before we started at the Pacific Neuroscience Institute, Dr.

David Merrell and Dr. Dan Kelly in their groups, we're now setting up the first Precision Medicine program for neurodegenerative diseases. And so this will include macular degeneration and include Parkinson's and MSSA and all the things you've talked about.

And I'm hoping it will be a be up and running by the first of the year. So very excited about that. And I think as you said earlier, you know, this is an exciting time.

Things are changing rapidly where we had just nothing to offer when I was training in the eighties, when I was in medical school in the seventies, there just hasn't been anything.

So we're in a new era. We're kind of coming out of the dark ages and I think it's so exciting to see as you know, to see people. And you were obviously the major contributor to the paper we all published together.

It was 15 different sites where we had people who had actually documented improvement in their cognition and you had more examples than anyone knew. So I think this is a really exciting time.

I'm just writing a paper up now on people who've had many years of improvement. The very first patient who came through is in April of 2012. She's now over 11 years and still sustaining her improvement.

So it's really exciting to see people get better and then sustain their improvement again. You know, this is a new era. It's a new era. And not to digress too much in the drug treatment for Alzheimer's disease, but, you know, it is a new era even for that.

And seeing how all these puzzle pieces fit together actually, I had clinic this morning. I saw a patient reviewed their spinal fluid results because that's primarily what we're doing on the precision medicine side from a diagnostic perspective and measuring the amyloid and tail levels.

And I said sort of the good news, bad news. You know, the bad news is, yes, this is Alzheimer's disease. Pathologically speaking, it's Alzheimer's disease, meaning this gentleman who was aware of some cognitive changes still at a fairly high Montreal cognitive assessment score.

He was really hovering just at the low end of the normal range or, you know, 25, 26. Right, folks, this is a scale that's measured up to a score of 30 and 26 to 30 is generally considered a normal score.

But we've known for several years it was established through a consortium that included the National Institutes of Health and Kaiser Permanente, the idea that we can not only diagnose things like Alzheimer's disease clinically, you know, a progression, a worsening of of short term memory or delayed memory, and then eventually involvement of instrumental activities of daily living and so forth.

But really more importantly, we should be looking at more precise ways of measuring and identifying this disease. And this is where looking at spinal fluid level measures such as amyloid and TAO or doing PET scans and things like that, or more recently now we're doing blood work even to look at that through companies like C2 and Diagnostics.

But the point here is that we can identify the problem before it's a huge problem, right? I would say it's like the the fire that starts in the kitchen when there's a little smoke and a little flame.

Let's catch it there before the whole house burns down. And now we have an opportunity really to call in the fire department right away. And we get the work of Dr.

Bredeson with the RE code protocol and potentially we can involve some of these new emerging pharmaco therapeutics. Don Anim AB as one example. Soon we hope it's not yet FDA approved, but probably by the end of the year or early early January 2024.

But anyway, we can do things now. Finally we can do things now. It's so important for folks to understand. So that is the silver lining when you hear the bad news that, you know, this is this is early stage Alzheimer's.

Absolutely. You know, when I think that what's happening is that we have an approach where we can do a precision medicine sort of approach and we can see people get better.

We had, as you mentioned, the drug approach, which doesn't really necessarily make you better, but it slows your decline. And I think bringing these together is the future.

So we can say, okay, we can come in now, improve your cognition, then we can slowly remove the amyloid. And I think of the amyloid as kind of being a long term cytokine.

Yes, it is there as part of the inflammatory response. It is there. It's something that can damage synapses. So in the long run, we will slowly want to be able to move that out.

But the idea of just doing that and nothing else, you're still kind of you know, you're leaving a lot on the table. You can do a lot better than that by getting their synapses functioning better.

So I think for the long run, it's going to be very exciting to be able to combine all of this and really get some tremendous and sustained improvements.

Right. So if I can just quickly summarize it and then we want to talk a little bit about Lewy Body disease. But Dr. Bredeson, I think is saying is if you imagine that you have a car, you've got your accelerator and you've got your brakes, and you need both to get a move forward, but you got to slow down, right?

But if your accelerator somehow gets stuck, you're in trouble. If your brakes get stuck, you're in trouble. Both are key parts. And what he's saying is with this amyloid precursor protein, the cell has the capacity to evaluate the cellular or environment and say what is needed at this moment in time?

Do we need to be in growth mode? Do we need to be in paring down mode and it does this by looking at all of the signals that are coming into the cell.

And that's really the key to the Recode protocol. When people say, well, what causes Alzheimer's? Well, a lot of things cause Alzheimer's, right? And he's talked about mitochondria.

He's talked about inflammation, and we talk about metabolism playing a major role in things like diabetes. But ultimately, the protocol is really about looking at what you many years ago coined, the 36 holes in the roof.

Right. It's many things. And we can't just focus on one thing where we've got 35 more leaky holes and we haven't ultimately accomplished anything but this.

Folks, this is possible. We talk in science about reproducibility, it being one of the key content concepts of scientific research. And Dr. Bredesen is definitely led the way.

But the good news is that there are enough scientific papers out there that have said, yes, these things make a difference and so with that confidence that you can engage someone who is familiar with this sort of approach and make a difference in your own life, you know, I would encourage all the folks watching this interview to take action for sure.

Yeah, I know you probably know Dr. Heather Sanderson just published her own trial that reached very similar conclusions to the clinical trial that we published last year.

And these things are all freely available online. So you can read them. And again, you know, seeing people actually improve, not just slow their decline, but actually improve growth.

Again, I think, you know, the future is bright. Yes.

Precision Medicine and Early Detection of Alzheimer's

And there are many, many treatments in addition to making these critical changes, which it's certainly not an either or. There are many, many studies, many treatments in the pipeline.

Dr. Cummings publishes his paper annually, I think, talking about what's what's going on. And we're even involved in a trial with a company that has a has a compound that's FDA approved in South Korea.

But it's it's MEK for a different reason that you'll see, but it's mechanism of action is very similar to SARS and Viagra, but it crosses the blood brain barrier.

It's called a phosphodiesterase inhibitor. And in this particular, we're doing the phase three trial and the phase two trial, the subjects involved in the trial actually had improvement in cognitive function, which kind of makes sense, you know, because not only are then you clearing amyloid, because you're improving the outflow of the brain, but then you're also bringing more oxygen and nutrients and so forth to the brain tissue that really needs it.

So very exciting time. Yeah. You know, we are at a point now where Alzheimer's is optional. You know, if you're a young person and you want to make sure not to get it, okay, when you hit 40, you know, get a conscious copy just as everyone knows, to get a colonoscopy when you turn 50, if you've turned 40 or if you're over 40, get a conscious copy, see where you stand, see where your blood markers are, and can you mention something very important?

It used to be we have to check CSF markers. Now there are for the first time biomarkers that you can get in blood like will 181. Soon there will be portal to 17, soon there will be GFP.

Currently, as you mentioned, you can get a better 42 to 40 ratio and I know C to N has this and also now Quest has offered this. So there are ways that you can really look and see this coming, which is so important.

You can see this for years ahead of time and make sure that you don't ever progress to that final stage where we call it dementia. You know, you can really look early.

The great news is you have a ten year period of sort of so-called MCI subjective cognitive impairment on average ten years, where you can do something very early on and reverse the decline.

So everyone should get on active prevention or earliest treatment. Please don't wait. And we really can reduce the global burden of dementia. Yes. And folks, you know, if if folks watching this have been to a functional medicine doctor, they may know that very often functional medicine doctors don't take insurance.

But I don't want those types of costs to get in your way. You should know, too, that even a center for Medicare and Medicaid Services has a specific CPT code.

That's a code that is assigned to a procedure or a type of office encounter that actually allows it. It's basically called a chronic care management code that allows for extended time with your physician.

And it is specifically it was specifically designed for cognitive screening and for your doctor to create a whole cognitive care plan by spending more time with you doing these tests that, by the way, that couple of tests that were just mentioned, they're generally covered by insurance.

So don't let these costs get in your way of checking your brain. Dr. Bredesen, I wonder if we can go ahead and move into the discussion of a disease that some people may know by name.

Some people may know because the great actor and comedian Robin Williams took his own life in the face of this disease and maybe the concern of what the future might look like for him, I don't know all of his motivations.

We talked about the fact that this disease very often is characterized by significant changes in mood, not just, you know, memory or movement, but Lewy body dementia or dementia with Lewy bodies, Lewy body disease.

As far as I know, these are more or less all synonymous terms is the second most common and degenerative dementia. Is that correct? Yes. Now, people talk about that.

So in some studies, vascular dementia is second and in some studies Lewy body. But yes, there are about a million people in the in the United States who have Lewy body very, very common.

About the same number actually as have Parkinson's pretty close So between Lewy body and Parkinson's. And as you indicated, I mean, these are all related MSA, those three are all these so-called synuclein ofthese.

And just as amyloid is an anti-microbial, so is alpha synuclein. And it is another anti-microbial. And it's interesting because we think of amyloid as being mostly extracellular.

There is some intracellular amyloid. We think of alpha synuclein, which was named because it was both in the nucleus and at the synapse. That's hence synuclein as being something that tends to be inside the cell in general.

And of course, but of course, cells ultimately die. And you now have, you know, you now have extracellular alpha synuclein. And so it is presumably doing the same sorts of things, going after pathogens, but also binding various toxins and things.

And it's interesting, you know, the Lewy body disease often is mixed up with Alzheimer's. Many people with Lewy body disease will have an initial diagnosis of probable Alzheimer's disease.

And of course, some people are thought to have Lewy bodies end up with Alzheimer's. And of course, the I think the final point here is that the pathologists will tell us and Dr.

John John Trojan Wolski, a very famous pathologist from Penn, has pointed out that somewhere around 60% of

Alzheimer's patients have some Lewy body. Yes.

So, you know, there is this kind of gradation where you can have mostly Alzheimer's, you can have mostly Lewy body. And we see a lot of people who have some as well.

And it's interesting to me because we think of Alzheimer's as more temporal and parietal, and we think of Lewy body as more parietal and occipital. But we do see forms of Alzheimer that are more parietal occipital, and the famous one is posterior cortical atrophy. Yes.

So one of my questions has been, are these the people that are going to have more likelihood of having more Lewy bodies? So when you see this and by the way, we've had people with Lewy body disease, Lewy body dementia, who've responded nicely to Recode, you have to focus a lot on the toxins because Parkinson's, Lewy body, these tend to be toxin related illnesses or Alzheimer's.

Some of them are toxin related illness, but many of them are pathogens. P Gingivalis herpes simplex, things like that. And again, as you indicated earlier, 36 holes in the roof.

So there are lots of things that can contribute. But in general, the people who have Lewy Body look a little different. They often have visual hallucinations, as you know, although I find that those are often more you know, they're a little bit later when you see the early ones, they don't necessarily have that, but they often have a history of toxin exposure.

They will often have this waxing and waning as you know, which has been called Showtime.

Understanding Lewy Body Dementia and Related Disorders

They have times where they're really good and times where they're not so good and then more of a waxing and waning than your typical Alzheimer's patient.

And then, of course, they can have sometimes delusions. They think that someone was, you know, came over earlier in the day, but they didn't really. And then interestingly, they also have this late sleeping pattern.

Well, they have someone that, you know, all of his life or her life. They got up at 630 or seven or 730 in the morning, and now suddenly they're sleep until ten or 1030 or 11 and something has changed.

What's going on? And so that's another piece of this. So they do have an Alzheimer like dementia. And of course, the the other thing to note is that they have a little bit of Parkinson's.

It's almost as if someone took a little Parkinson's and a little Alzheimer's and put them together. And that's really what Lewy Body looks like. And so they often will have slowness in their gait.

I mean, you know, their mitochondria aren't firing on all cylinders, so they're going to be slowing up. Their facial expression changes, their speech can change.

And they have what neurologists would call some Parkinsonism. Let's go ahead, please. Yes, just. To say and I would mention cortical basal degeneration is a rare form, but it's also related.

In that case, you don't have the synuclein and you have tau, but they have Alzheimer's changes in about 20% of them. And they and Dr. Craig Tornillo has a remarkable case recently where he has done some beautiful and very successful treatment, again, identifying critical toxins that this person was exposed to.

So, again, I think this area of of neurodegenerative disease that has just been hopeless for decades is really opening up. Right. And I was going to say, oftentimes the folks who have Lewy body dementia may have mood changes, personality changes early on, some falling and the Parkinsonism starts to appear late a little later as things begin to evolve.

So that with what we call Parkinson's disease, dementia, there's I have read some papers that think that maybe that's even the same diseases as as as you know, as Lewy body, although the localization, as I understand it, of the alpha synuclein is in exactly the same.

Yeah, but that being said that one of the fundamental differences is that with Parkinson's dementia, the movement disorder piece is an earlier presentation than in the Lewy Body folks.

And that's, you know, such a good point if you look. So it's interesting to me what the neural pathologists have told us for years, and of course they've told us that Alzheimer's looks like it somehow comes in through the nose because of where it spreads.

When you look at Parkinson's and as you said, then the Parkinson's can go on to dementia. That looks like it's coming from the gut through the Vegas to the brain stem and then going from the brain stem up on the other hand, when you start with the Lewy body, you get the motor later, as you said, and you start with the cognitive changes.

This really looks like something that's coming through, the sinuses that's coming. You know, you're and it may be that there is some sort of pathogen or there is a toxin that you're breathing or something like that.

This is really starting more cortical cortical. And then to some extent, you're getting some brain stem pathology. But the two look quite complementary.

They are kind of different starts and different spreads. But of course, with this prion spread, you actually have a protein, in this case alpha synuclein that is beginning more of itself and spreading throughout the brain, presumably, you know, to fight the pathogens, to fight that, you know, whatever the insults are that are starting this.

Yeah, we interviewed the chief medical officer, Dr. Todd Levine He's with CND LifeSciences. So we do a lot of this in one skin punch biopsies. And I just want to remind folks that this is a sue nuclear property.

And if there is a question that you have Lewy body disease within the appropriate clinical history and physical, if that's what's suggested by the presentation, then the skin punch biopsy can confirm that.

And they did present some data where they could actually tell differences within under the microscope between Parkinson's Alpha synuclein and Lewy body disease and multiple system atrophy and what's called primary autonomic failure as well.

Or they could actually distinguish based on the microscopy, what the person had. But it's always important to put it in the clinical context. That's fantastic.

And do you have a sense for and we've just started doing these on the patients where we suspect that there may be Lewy body disease? And do you have a sense for the sensitivity and the specificity in these in this particular test?

I do. It's actually been reported very high, 96% on both sides, sensitivity and specificity. Wow. It's an excellent test. Excellent. I don't even I really don't order that scans any more, although there's some proposal that's come up recently about a new staging system for Parkinson's where we could combine looking at alpha synuclein with a dopamine transporter, concentrate patients through the dad scan and have a more accurate neuropathological staging system.

But for now, the skin punch biopsy is to me a better way to go. Yeah, he's. Fantastic. So. So one of the things I think that's really important is we've been having this a pretty intense discussion.

It's been a lot of fun, is that we focus a lot on kind of a disease centered approach. We say, Well, this is Alzheimer's, this is Parkinson's, this is Lewy body dementia.

And there is truly value in sort of naming the disease. We our brains gravitate. We want an explanation and we

want to understand. We want to have that sort of please help me know what's going on so I can understand also not just what's happening now, but what might be happening in the future.

But we also do it because traditionally we want to assign a drug to that diagnosis. Right? But the paradigm now really is starting to shift, and it certainly doesn't in any way disqualify the need to understand what is the primary diagnosis, which is the at least the primary driving pathology.

But now we want to say what are what are all of the things that underlie that? Why is this happening? Because that's really where the empowerment comes from.

Absolutely. And I think that is the fundamental difference between 20th century medicine and 21st century medicine, 20th century medicine. And we we I was taught to make a diagnosis.

What is it? Is it Alzheimer's? Is it you know, is it Parkinson's? Is it Lewy Body? Now, as you said, it's all about why Why did you get this? And, you know, just as an example, we had a patient recently who was doing well, who turned out to have positive P Tau 181.

Diagnosis, Biomarkers, and Personalized Treatment

So some but but modest So so yes, there's some Alzheimer's component. That form of signaling that is telling your time to get phosphorylated and that's part of pulling back the new rights that is present.

We know that, but it's not very overwhelming. The person also had a history of CTE and you know the right history for it. Some of the right symptoms were the person also had some clear vascular changes, vascular damage.

So this person had cognitive decline. But, you know, was it Alzheimer's? Well yes, partly. Was it CTE? Well, yes, partly. You know, was it vascular dementia?

Well, yes, partly. But so you have to now take those all into account. And again, I think being being able to say, why is this so that you can now address all the things we're certainly removing anything that is continuing to push you downhill if you have Lewy body, you're presenting and you're starting to go downhill and you're, you know, living in a place where you've got a lot of toxic exposure, you need to remove that and you need to detox and and again, but at the same time, you want to support the dopaminergic system, you want to support the cholinergic system.

Obviously you want to stay away from classical anti psychotics that can really be damaging to these people. So I think we're getting a much more symptomatic and biochemical view of what's going on here than just the old idea of it's just about pathology.

Yeah, this person had Lewy Body. One of the things you shared with me, the last time we saw each other in person is that you, your company is leading the way and opening up clinics around the country.

So more and more folks are seeking out this kind of care can have access to that. What's your tell me give me a little update. Yeah, it's a great point.

So, you know, the big change is going to be this this precision medicine program, I think, in Los Angeles. But the first couple of clinics, one opened in Sarasota.

And of course, as you know, there have been over 2000 physicians that have taken the training at various times from 2016 all the way until the present.

And in these are in ten different countries and all over the United States. But as you well know and you saw this in the paper where you were a coauthor, some are getting better results than others.

And it depends on, you know, how you're doing it. It's I would say this is much more like a surgical procedure or

than it is like classic prescription pad medicine.

You're looking at lots of different things. You're doing the right things at the right time to the right people in the right way. And if you do that right, people are getting much better.

And if you're not or if you don't find the things that are driving this, then you, you know, you have more of a problem. And I should just say parenthetically, we've had people where they would improve for years and then start to backslide.

And we look to say, okay, why is that? And find new things, address those and they come right back. And this just happened to a woman who had was in her seventh year doing great.

She started to have problems again. We found out what the new things were and she's doing well once again. So, yes, the first clinic was then in Sarasota, the second one in Jacksonville.

The third one is probably going to be in Salt Lake City. That's just being set up. So the the hope is that we will have these all over the country or interact with with clinics that are already present like your own so that that we can get.

Of course, the goal here is the same for all of us. How do we get the best outcomes? How do we truly reduce the global burden of dementia and ultimately of other neurodegenerative diseases as well?

So again, things are really moving rapidly in, you know, in this era we're really coming out of the dark ages of an inability to treat neurodegenerative conditions.

It's so true. And you know, to your point, this is not a 14 day course of antibiotics once and done. This is a lifelong commitment to change. We're co hosting with Dr. Sandra Scheinbaum, who has functional medicine coaching academy.

Because I have such a huge commitment to the idea that health coaching is absolutely integral to the success of the kind of work that we're doing. Absolutely.

I think it's one of the most critical components because people, you know, help the coaches can really help to get best outcomes, to work with the patients to make sure that they are doing the right things, that they're doing the things that the physician has suggested.

And it's just a it's a critical piece going forward. Dr. Bredesen, if someone wants to find out more about you, about the Recode program, about finding a practitioner, what what can they do?

Where do they start? Yeah, lots of ways. So as you know, there there are a few books out there. So there's a book called The End of Alzheimer's, which was on New York Times bestseller list for four or five months.

It's in 33 different languages. So you can you can find it very easily on Amazon or wherever you are Barnes Noble, wherever you like to buy books. Then there's another one at the end of Alzheimer's program and a third one, which is called The First Survivors of Alzheimer's.

You can also go on Facebook, Dr. Dale Bredesen also what used to be called Twitter and I guess now is called X and and then also on Instagram and then I would encourage people, please, if you're 40 or over, please get a cognoscopy.

It's much more pleasant than a colonoscopy, having done both of them. I can tell you that.

Clinic Expansion, Coaching, and How to Get Started

And you can, you can find out where you stand and please get on active prevention or earliest treatment. Let's make it so that dementia is a rare problem.

And then you can also check with the Pacific Neuroscience Institute and and also through the so called Gray Matters clinics. So there are lots of ways and again, so much that we can all do.

If you're interested in taking the training, there's Recode training, Recode 2.0 training now, which is over 30 hours from all sorts of experts. Chris Shade on Toxins and Neil Nathan on Bio toxins, lots of great stuff.

And let me just end by sayingone of the biggest, as Dr. Sharlin knows very well, one of the biggest one of the biggest challenges for people who are just getting started is to find the right diet, get getting the right food, go into the right place.

It's it's something that people often say, well, I just don't want to do this. So we worked with nutrition for longevity and it was called so-called for Elle.

They've done such a great job with all sorts of medically appropriate diets and they have produced the KetoFlex123, you can see this if you go online, KetoFlex123.com and they have ready to ship meals and I've eaten them myself, they're fantastic.

I've actually been really excited about what a great job have done with making these things delicious. They help you get into ketosis in a plant rich, mildly ketogenic diet.

Really fantastic. So I would encourage people give those a try and nutrition for longevity has just done a great job there. Wonderful. Dr. Dale Bredesen, thank you so much for participating in the Parkinson's Solutions Summit.

I know that folks who've been enjoying this today have gotten lots of rich information and can start to take some action steps to change the trajectory, whether it's their Parkinson's, their early stage, Lewy body disease, multiple system ad free Alzheimer's disease.

We've got to think of these as having common root causes. And ultimately it's about personalized and precision medicine, figuring out what your imbalances are and working with a team of providers, all of which are going to be experts in their own area but focused on you and helping you to correct and maintain that new trajectory indefinitely.

Right. Fantastic. Thanks so much, Dr. Sharlin. Always great to talk to you. Bye bye now. Thank you.