Introduction and Summit Welcome

Welcome to The Parkinson's Solutions Summit 2.0. I'm your host, Dr. Ken Sharlin. And today I have Dr. Dale Bredesen with me. many of you may have heard Dr. Bredesen's name is associated with The Bredesen Protocol for reversal.

As well as prevention of Alzheimer's disease. Dr. Bredesen has influenced me greatly. He has trained many, many providers in his approach. there are many, certified medicine practitioners all around the world now.

And so I'm really looking forward to diving in to understand not just the medicine protocol, but how this can apply to other neurodegenerative disorders, particularly Parkinson's.

And a big part of our discussion today, Lewy body disease or Lewy body dementia, which overlaps tremendously with Parkinson's. So without further ado, Dr. Dale Bredesen, welcome to The Parkinson's Solutions Summit.

Thanks so much, Ken. Always great to talk to you. Well, it's great to have another neurologist, on the Summit. And we can kind of talk colleague to colleague and really dive into things.

and we're going to talk about some fairly complex stuff today. So, I don't I want to make sure we don't lose anybody. And I wonder if we can just sort of start with the real basics about rowing this Lewy body diagnosis or Lewy body disease into that broader spectrum of neurodegenerative parkinsonism.

And what's the similarity? What's the difference? How do we make these different types of diagnoses? Yeah that's such a great point. And you know, with with what's going on politically right now, of course, we're all hearing a lot about Parkinson's disease, about Lewy body disease, about cognitive changes and things like that.

Lewy Body, Parkinsonism, and Disease Overlap

And, you know, just to start with a 30,000ft view. as you know, we spent 30 years in the laboratory looking at what is the nature of the neurodegenerative process for all these things frontotemporal dementia, Lewy body, all that.

What is that? We understand something about what happens with cancer. You have somatic mutations. You ultimately have more cell growth and survival. Then you have cell death.

And that's what gives you cancer. But the idea of neurodegeneration, as you know, people have said it's about free radicals. It's about, you know, it's about mitochondrial damage.

it's about amyloid. It's about tau. It's about misfolded proteins. It's about aggregated proteins. It's about prions. You know, there are dozens of ideas, but there hasn't been one approach to say, this is the fundamental nature of what happens with neurodegeneration.

Telling us how to treat it, how to prevent it. Just as, you know, you're just as you've been doing you obviously written a whole book about this. So what what we found in the lab is that when you look at a neurodegenerative condition, two things happen.

First of all, you have to consider evolution. And so what happened with evolution? Just as we select it for performance over durability, which is what is associated with our aging, it's called antagonistic atrophy.

And species evolve. They continually select for performance over durability because of course, you're struggling against the rest of the world to try to get your food, beat your enemies, all that stuff.

Now, when you do that, you end up with great performance. And so we have tremendous cognitive performance, tremendous motor performance, tremendous motor control.

And you can, you know, measure these and show how incredible this is. But at each one of these we have such fine tuning that unfortunately we are over time susceptible to damage loss from things were exposed to.

So what we found is that you for each of these neural subsystems, there's one for or for for plasticity which is goes awry in Alzheimer's. There's one for motor modulation, which is, you know, dramatically needs mitochondrial complex one.

That's what goes awry in Parkinson's. And there's one for motor power, which goes a lot goes awry in ALS. In all of these cases, there's a whole set of things for supply.

There's a whole set of things for demand. So when your supply goes down, which happens as we age with fewer hormones, you are nutrients, more inflammation, etc., your or your demand goes up and that's due to toxicity, inflammation, things like that.

You end up with a system that's impossible. You cannot support that very complex system. And so the system now begins to involve. But you can support a smaller system.

And so when you and I treat these and all the many people who are doing this now and getting very nice results, really the first of seeing that you can actually make people better, you've got to reduce that demand, reduce the inflammation, reduce the toxicity, and you've got to increase the supply.

So that's the fairly simple overall concept. Now as you said now we have to adapt it for each disease. And right with Parkinson's and Lewy body I always think and you know you're really an expert in this area.

But I always think the of the paper that showed years ago, if you start with cognitive change and then you get some motor change, it's very likely to be Lewy body.

If you start with the motor change and then you get some cognitive change down the road, it's more likely Parkinson's and dementia. So I always try to look at, you know, when did the cognitive change, if a if any start.

And then as you know there are the three major, you know, upstream before you ever get the motor changes people often get those three. They get some anosmia.

So they're having trouble with their smell. They get some, REM behavioral disturbance. So they may be flinging their arms around and they often get some constipation.

and I was actually surprised to see that just in these large epidemiological studies, you just take the people who had histories of constipation. They are at increased risk. Oh, yeah.

Right down the line. Diagnosis of Parkinson's. So I think, you know, that's kind of the typical way, as you pointed out. You know, there's Parkinson ism in both of these.

And also in that other third synuclein, an apathy where you have more of the autonomic problems so called MSA. So I try to look is is this mostly a cognitive thing.

Is this mostly a motor thing. Is this mostly an autonomic thing? Absolutely. And then I know it's it's the old expression are you a lumper or are you a splitter?

Biomarkers and Precision Diagnosis

And now the question is, is Parkinson's dementia really just another expression of Lewy body disease? And are they really more or less one in the same disease?

And in the long run? you and I, before we started, to hit the record button, we were having a discussion about what are called biological markers or biomarkers.

And, you know, with all of the new emerging technology used to amplify proteins and really look at what's happening at the cellular level and be able to get a simple blood test or other lab measurements, whether it's saliva, stool, spinal fluid, you know, we ideally go for the simplest thing to access.

even you brought up this, had brought up a sweat, test. will be much more precise in being able to make a diagnosis, but I wonder, you know, in how this will all play out that this sort of precision medicine will also allow us to choose treatments that are also personalized as well?

Yeah, that's a great point. And I do think, you know, there's there's a there's a push for, you know, omics and for looking and especially with all the I that's about now for looking at larger data sets and some really exciting things.

And one example that you and I discussed earlier, again, before, before you started recording here, was that you look at just sweat from someone with Parkinson's.

The finding has been that there are 400 different chemicals that they're finding that is as a signature in people with Parkinson's disease. And this, of course, came from a nurse who could smell Parkinson's and said, you know, there's something there.

and she was extremely accurate in the the blind tests that they gave her. And it turned out that you could then look at this, with Mass Spec. So I look forward to the day.

I hope it's not going to be too far in the future where this will be something that, again, you can do with a quick test and that you can do years ahead of symptoms, say, here's a person just like you would say, here's someone with prediabetes.

Let's make sure they never get to diabetes. If we can pick up many, many people with pre Parkinson's, pre old timers, that sort of thing, and make sure that they never have significant symptoms, that will be a huge step forward in an area where as you know, the neurology has been the area where there hasn't been a lot of treatment.

And I would say Parkinson's would be one of the few exceptions where you could actually make a difference in the symptoms, but really not much in the course of the disease.

So, I think we're in a very exciting time where there are going to be newer, better biomarkers, earlier biomarkers and more addressable biomarkers. And I would add one other thing, which is very exciting to me.

I think you mentioned lumbers and splitters, and I think that we have these different syndromes that are pretty clearly demarcated. So posterior cortical atrophy PCA which is interesting is another one that is an Alzheimer one.

But it's, it's closely related to Lewy body disease. Yeah. Primary progressive aphasia PPA cortical basal degeneration, which is really a Parkinson's like syndrome, which is which is a tau apathy instead of a synuclein apathy.

But 25% of those people have Alzheimer's disease as it turns out. and, and some of these ones that are, that are clearly demarcated, and so then to take those and say, okay, let's look carefully at the omics of those and see, you know, can we find them.

And as an example, we had someone and I would put Lewy body in there as well because it's a pretty demarcated illness. someone recently, cared for by, by Kerry Rutland, who's a superb health coach out of New York, who has a posterior cortical atrophy.

And actually, she's doing quite well, and has improved and, you know, reading again, using the computer again, navigating again. So it's wonderful to see dramatic improvements in her MRI.

And if you look at okay, what were her drivers in her case, she ended up having Bartonella, a tick borne illness which was treated. She end up having mycotoxins as well.

And then she had some metabolic changes as well. So now the question is, is it going to turn out that many people with PCA, have this sort of profile?

We don't know yet, but at least it begins to get okay. Can we look upstream at what are these things that are driving this as opposed to just, you know, we're all we've inherited what came from the 19th century, which was the era of neuropathology, so many advances of neuropathology and what ultimately became in the 20th century, synuclein and ofthese tau ofthese amyloid related tau.

Amyloid, Synuclein, and Mixed Pathology

With these, now we can begin to say, okay, let's take the next step. How did you actually get this? And I think what's fascinating to me, we've all blamed as a field of neurologists.

We've gone through the period where we blame the disease on the amyloid. And I think now we're taking that next, although I know some people are just hanging on to that tenaciously.

because yes, it does have some impact on brain function. But the real question is what brought out the amyloid. It's clear it's an anti-microbial peptide.

What brought out the synuclein, which is another anti-microbial peptide. What brought out the towel? Also an anti-microbial protein in this case. So each of these is a response to insults.

And I think functional medicine is the best equipped to look at what are those insults that are driving you down these different pathways. And you mentioned earlier that, you know, more than half the people, with Alzheimer's.

Also, if you stain for Lewy bodies and synuclein and you can see the Lewy bodies, and I remember the late John Trojan asking such an absolutely fantastic neuropathology.

Just who pointed out at a meeting, you know, several years ago that if they looked at all in people who were diagnosed with Alzheimer's, most of them had all four.

They had amyloid staining positive, they had cow staining positive, they had synuclein staining positive, and they had TDP 43, which we associate with frontotemporal dementia in ALS positive.

So this makes some sense if we understand that we are responding to these various insults, and then we can identify those and address those, and we can look at the, you know, what's the profile?

Why do some people go down the Lewy body pathway. Some people go down the Parkinson's pathway, some people down the Alzheimer's pathway. Oh, no doubt about it.

I tell you, this is very anecdotal, but, we were, here at Shoreline Health Research. We were part of the, trailblazer ALS to, study that put, recently approved Inanna Mab, on the map.

now, you know, approved. But there still has to be an NDA or coverage, but nevertheless, we are a large enrolling site, and this folks, is, one of the anti amyloid therapies, recently approved for Alzheimer's.

The other one is Lacombe. we're looking at. But, in order to put individuals into the trial, it's where double blind, you know, randomized, placebo controlled trials.

So we didn't, of course, know who is on treatment, who is not on treatment. nevertheless, they all had to have Pet scans. That's, positron emission tomography scan.

In this case, we're able to confirm both the presence of the insoluble amyloid protein as well as tau protein. So for sure, when someone is randomized in the trial, we knew that they had Alzheimer's.

We had one individual little older gentleman qualified for this study, got randomized. Again, I don't know what he had. now, you know, again, very anecdotal, but, he really took a nosedive, after starting the study, starting to receive whatever investigational, treatment he was randomized to.

he got extremely psychotic, extremely agitated, to the point that he very quickly had to drop out of the trial, very quickly ended up in a nursing home where he was behaving extremely, you know, outrageous kind of behaviors.

and a neurologist who saw him out, you know, outside of the context of the trial, decided that he had Lewy body disease. Now, I don't know that. I don't know on what basis was used to decide.

Yeah, you had that, but perhaps he did. But we know that he also had Alzheimer's, without a doubt. and unfortunately, you know, his his trajectory was very different than the typical trajectory of Alzheimer's disease.

And I recently got noticed that he passed away. and probably from the time I met him, we was really just kind of in that mild cognitive impairment stage.

and so he declined rapidly in 2 or 3 years to the point of death. so this is a very interesting, you know, presentation. You know, I'm sorry to hear about that.

We have had several people where they presented with, you know, MCI, just as you said, mild cognitive impairment. they went on antibody treatment of one sort or another.

you know, whether it was the earlier trials of Solana's, Mab or donate memory or look, I, me, what have you and with each injection, they would clearly get worse.

And then slowly typically they would these would be about a month apart. And then I don't know in your trial with these two weeks apart or or a month apart.

Their monthly infusion. Monthly. Yeah. So that what they would do is they'd go downhill and it was typically a couple days and then they would slowly come almost back to where they were.

Then they would get the next injection and they would go. And the temporal relationship was so striking that, you know, it was hard to say, well, these things are helping them.

Treatment Responses and Clinical Caution

it really, you know, it really was we had one guy who went from a 22 on his Moca down to a six over two years, and I asked the wife, well, you know, didn't you?

Weren't you concerned when you saw each month that he was getting worse? And she said, well, the doctors know what they're doing. So, you know, she was going with, okay, this is a trial.

And so this worries me. Now, Sally, who I wrote about in the book and actually who is in the documentary, memories for Life Reversing Alzheimer's, which is on Amazon now.

And, Michael Bublé did a great job with, with his narration. She talks about, you know, what happened with her. She had, eight. She went through eight injections, and with each one, she clearly got worse.

And so she said, after eight injections, that's enough. She's a nursing professor. So she she had taught about Alzheimer's for years, and she said, that's enough.

and and she was going downhill. Now she's done very well. got her Moca back up to 30. and really made a

wonderful comeback. She's now been seven years, on the protocol and continuing to do very well.

I should mention, you know, one of the things we found and we've just posted this, it's it's in review. So it hasn't been published yet, but it's posted and freely available publicly.

which is that we have people who are over ten years where they've improved and they've maintained it for over ten years. But what we found during that time is people will often have a secondary decline.

They'll go for a few years and then they may kind of fall off the wagon. Now, then, then when you look and say, okay. And in Sally's case, she had a after six years, she had a little bit of decline.

And then she was reevaluated by her physician, who found she had undiagnosed sleep apnea. And clearly that should have been picked up. But it was probably worse at that point.

The second thing she had was she had new Miko toxin exposure. She had a new leak in her roof, and that was, affecting her. And then, interestingly, she had a Cryptococcus Lauren Chai, infection, a sinusitis.

Those three things were treated. She went right back up again, did better again. So, you know, I think the the overall conceptual model that you are dealing with these insults, and that, you know, this is part of the response with the amyloid, with the alpha synuclein and things like that is holding up.

Well, the problem, of course, is there is this preon phenomenon where you're now amplifying the response, just as happens when someone gets, dic in the hospital.

we've got this phenomenon that's kind of a runaway. And so you've got to quell that. You've got to bring that back down. So I think that the these different models are kind of coming together.

but I think your point about seeing people get worse after these injections, although, again, as you said, you didn't know what he was on. But, you know, I don't know if the temporal component was there, but what we're seeing is very temporally linked.

Well, just be clear, folks. We're talking about two FDA approved drugs that had to go through the rigors of clinical trials and convince an FDA advisory panel that this was worthy of being approved and then ultimately approved.

but as the expression goes, sort of individual results may vary. And while the drugs have some modest benefit, in general, it's kind of like saying, well, if I take an aspirin every day, does that guarantee that I won't have a stroke or won't have a heart attack or whatever?

And of course, many, many people take an aspirin and still have heart attack or stroke. so we do want to I do want to be cautious and want to leave people with the impression that these treatments don't have any benefit.

But clearly, we are at the tip of of learning and what we can do and what these drugs are really doing. And I hope as we bring on more of this precision medicine and its biomarkers.

And so forth, we can really say this is the best treatment for you, but this is the best treatment, you know, for you. Yeah. In the so in the meantime, Doctor Price and you know, all of the things you've alluded to really tell us, you know, let me say that I love this.

You know, discussion about antagonistic play trophy and all that, because ultimately, what we're told when we talk about Parkinson's and Alzheimer's and Lewy body dementia and so forth, it kind of comes back to your infamous 36 holes in the brain.

If it comes back to saying these diseases sort of start out with common root causes, and then due to biochemical and of that individuality and various other factors, they start to split off.

But many still overlap. But it gives us hope that even today, if we can recognize these common root causes, that we can do things about it. and so that kind of goes into the breadth in protocol.

What do people need to understand about the drivers of this disease, like you mentioned, infection being a major driver. Yeah, that's such a good point.

Root Causes: Infections, Toxins, and Stress

and, you know, it brings up the idea that we need to more and more have places where people with any neurodegenerative disease, be it ALS, frontotemporal dementia, Lewy body Alzheimer's, what have you, a place where anyone can come like, you know, like the Charlene Center that you mentioned and have these different areas examined to find out.

Do you have specific infections? and, you know, this is where the diagnoses can help us. you know, if it's Alzheimer's, you're going to be thinking more about things like p gingival and herpes simplex and HHV six A and things like that.

and on the other hand, of course, what's been associated recently with Parkinson's, this DeSalvo Vibrio bacterium, we'll see, you know, whether that is a causal issue, whether that is just an associational issue.

You know, we'll see. you know, in ALS, people have looked more things like Acker, Mancilla and things like that. So, so the idea then is we need a place.

We need places where people can come. And so, I've been working with the Pacific Neuroscience Institute and Doctor David Merrill, Doctor Dan Kelly, very excited about that.

And we are setting up a precision brain health program where people with any sort of neurodegenerative condition can come. And as you indicated, infections are important, and I would I would take it again, look one step farther away, the things that the six major things you mentioned, the 36 holes.

And yes, there are all sorts of things that contribute, but the six major groups are, you know, anything that reduces your energetics. And of course, you see that so clearly in Parkinson's and you see it so clearly in Alzheimer's with reduced blood flow and sleep apnea and things like that, and then anything that increases your inflammation.

And that's where what you just mentioned with the various infections so important tick borne illnesses, exposure to some of the bio toxins that, create inflammation.

And so anything that's creating inflammation increases risk and something that should be addressed. And the third part is toxicity. Anything that is, these various bio toxins in organics like mercury, air pollution, things like that, and then the organics, things like glyphosate and Tal Ewing and benzene and anesthetic agents and things like that.

And then the other big three are anything that reduces neurotransmitters. And of course, the classic things that reduce dopamine, increasing your risk for Parkinson's.

so the neurotransmitters, and then the neurotrophic is things that reduce BDF so much associated with Alzheimer's, which is, you know, you can bring your body and up with exercise, up with exercise.

That really helps, in people with some borderline cognitive decline. and then finally, and interestingly, stress. And, you know, I have been so impressed, with a book that just came out by Doctor Neil Nathan, which is The Sensitive Patients, Guide to Healing.

I really I recommend it because there are so many people, it turns out, that have these changes in limbic response, changes in vagal response. And he has it is a triad with the third one being, the mix being the mast cell activation syndrome.

And what's happening is these people have been bombarded by these various insults, and their nervous system has essentially turned off and just said, okay, we cannot handle all this.

So we're going to limit your ability to respond, and we're going to basically keep you, he describes one patient literally would be walking down the hall and would pass someone who had a certain type of, you know, chemical exposure, for example, because of washing their clothes in a certain detergent.

And literally the person would fall to the ground. They could not keep their their tone because of their vagal issues and, and, and limbic issues. And this person got better when he did the appropriate things.

But I think in all of our practices, we're we're understanding now more and more that we do have to we do have to deal with that part of the problem that you've got people who are stuck in this stress or threat response.

And doctor, Dr. Clawson talks about this a lot with some of his, lectures on YouTube and things like that. So I think that these are, important pieces and we're all getting better at understanding, you know, when do we have to address this?

When do we have to address that, allowing us to get better and better outcomes to these people? That is, you know, just a few years ago were virtually untreatable.

I mean, you know, you look up, there's no no, effective treatment for Lewy body. interestingly, on the Mayo Clinic website, it says there's no treatment for PCA, posterior cortical atrophy.

Well, we're seeing some improvement in people with PCA. So, I think it's it's a new era and identifying these things, as you said, infection's so common in these people, it's typically those chronic infections.

But you get exacerbation when people get Covid, when people get the flu, when they get, when they get acute infections, they get an exacerbation of the underlying problem.

Absolutely. And I couldn't agree with you more about the stress component. So important. We actually interviewed Neil Nathan, Doctor Nathan for this summit.

So I know the author of these, interviews yet. But folks stay tuned or go back and listen to, Doctor Nathan's interview as well. He's, he's an old friend and, a wonderful interview.

You know, sometimes in the, in my clinic when I'm explaining these adaptive what really are adaptive responses of the body that don't necessarily serve the person as a whole.

I go back to that old scene from Monty Python and the Holy Grail, where the knights are trying to cross the bridge, and the gentleman's, you know, guarding the bridge.

And they they're sort of, you know, it's slapstick. So but they're cutting him up into pieces and he keeps saying, not dead yet, not dead yet, but until he's a tiny little stump.

So, yeah, he's not dead yet, but he's lost all function. And, you know, learning ways to turn that around by having a system like a Bredesen protocol, where we're breaking it down and we're looking at drivers of inflammation, and we're looking at, you know, drivers of oxidative stress, and we're looking at infections and we're looking at, you know, glycemic control, which I wanted to bring up because, you know, we're still in this epidemic of quote, diabetes City.

Yeah. and and, you know, I think I have the expression, you know, like you ain't seen nothing yet. Yeah. Because we're not we're not getting ahead of the problem.

In fact, I think I am not a conspiracy theorist, but I almost think that some of the, you know, all the excitement over the ozempic, you know, semaglutide type drugs is almost like, well, we got people heavy and now we have a drug

to treat and to get them thin, and then the cycle and start all over again.

And we just sold you on the junk food and the edible food like substances borrowing from Michael Pollan. And now we're just going to give you a drug to turn it around.

Not that the drugs don't have value. In fact, as you probably know, there was a paper in New England Journal not long ago about the impact on Parkinson's this day.

Perlmutter and I talked about that during his interview, but we would really hope to catch things at a much earlier stage.

Practical Prevention and the Seven Basics

We can, you know, save people the trouble of these diseases or reverse them when they're still in the early stages. Absolutely. And I think, again, as we get better and better biomarkers and more and more people to understand that the way to reduce the global burden of neurodegeneration is to get in early, get on active prevention.

we recommend that everyone begin active prevention at 40, get evaluated, get a clogged copy, get on active prevention. And you know, you mentioned earlier, about, you know, Parkinson's and some of these others.

The idea is to take the overall approach that we're all using, looking at these different upstream causes, the various contributors, and then adapt it.

When you look at Parkinson's and Lewy Body, you have to look more carefully at organic toxins, things like TCE and, and all the different toxins that are associated with parkinsonism, all that have an impact on mitochondrial complex one, when you're looking at macular degeneration, you have to look more at inflammatory components and things like, complement activation and things like that.

You've got to look at how much blue light exposure and things like that. So for each of these conditions, we need to look at and adapt that, you know, what are the things what are the upstream 36 holes, that there are actually going to be the most likely problems.

And I think doing that, we really should be able to prevent, and in many cases reverse, people who have these different neurodegenerative conditions where in the past it's been you wait till it's really late and then you give a medicine which doesn't do very much.

Well, one of the practical aspects to this doctor medicine, of course, is finding practitioners that understand this and can help people implement these principles.

And I know that's been a big part of your efforts, is helping to educate folks and really put a network together. So can you provide a little update on that?

Absolutely. Yeah. And so, so people can take Recode 2.0 training, and actually look forward to the day when we, when there's optimized training. And maybe this is something you're going to be doing.

I don't know. But but for how to approach Parkinson's but for cognitive decline, we have, Recode 2.0 training that's available. We've had over 2000 physicians trained and other health care professionals as well.

everything from health coaches, neuropsychologists, nurse practitioners, lpns RN's, you know, on and on and on, that have trained in this area. And so, we've got have been, as you said, all over the United States and all over the world, we've got ten different countries around the world.

there are people in Japan and people in France and people in Germany, in the UK and on and on and on. and so I think that more and more people will begin to see this at the same time.

we, as you indicated, the majority of people do not know this. So if you go into a, a typical university hospital today, people won't be looking for the upstream causes of your neurodegenerative process, unfortunately.

And I look forward to an update. And I look forward at some point to, the day when that will be the case. So our mutual friend, Doctor Terry Walls, sometimes talks about the walls protocol as sort of the kind of the public health version of functional medicine, meaning, Doctor Walls doesn't, treat a lot of individual people.

She does a lot of research, does wonderful, research, publication and so forth. It's really helped all of our efforts. but she'll say, you know, if you need that one on one, you know, go see.

And, you know, she does send a lot of patients me. But my point is, if we were to give folks watching our interview today, sort of that public health general principles, you know, where can I start?

What can I do just on my own? What can I do that could at least put a dent in this situation? I wonder if you could share 3 or 4 thoughts about what people can practically do before they necessarily dive into labs and individualized attention?

Yeah, this is such a good point because, you know, the idea to to have a public health, a big impact, without costing a lot of money, the idea is to have a multi-tiered system.

So you start with some basics, and then if people begin to develop symptoms, they go to the next level and so forth and so on. I think that's going to be the future.

I also do think the future is going to be, you know, drugs and protocol together. But what's easiest to do today? I wrote a book on this called The End of Alzheimer's and the End of Alzheimer's program.

Though the end of Alzheimer's program has more of the basics. Here's the, you know, nuts and bolts. Do this, do this, do this. That's the easiest thing to do.

and and then beyond that, there are some we we always think of this in terms of seven basics and then specifics on the specifics you can look at based on your testing.

and there is, you know, there are now tests that you can get biomarkers, you can get what we're calling a brain scan, which is p tau 217 along with GFP and NFL.

so you can, you can do that online, through, you know, you get mobile phlebotomy and you get that you can see your, your result there. But let's go back to the seven basics.

And these many of these apply to Lewy body as well in Parkinson's. So it's diet. We we typically look at a what we call keto flex 12 three which is a plant rich, mildly ketogenic diet.

Getting people away from that insulin resistance that you mentioned earlier. that's been such an issue with diabetes. so diet, exercise, sleep, stress, brain training, some detox and then some targeted supplements.

And those are the basics that can help most people, especially, early on. The earlier you start, the better. Then the specifics have to do with looking for those infections that you mentioned earlier and looking for those toxins.

And when we're looking at Lewy Body, we want to tilt this more toward toxicity. There seems to be a lot of toxin associate, Lewy body disease. And so we want to look, very specifically at the organic toxins, at the bio toxins, and at the inorganic, the metallo toxins and things like that.

And we see it again and again. And people have talked about even things like exposure to carbon monoxide and things like that, that increase your risk.

So finding out if you have toxin exposure is so important so people can get started. you can even look at, there's a

freely, publicly available, paper, where we talked about the rationale for a, precision medicine protocol for cognitive decline.

that is, freely available online. It's in the Journal of Alzheimer's Disease. and we go through the rationale for the different pieces. So I think that there is a lot now that's publicly available.

and people can just look to see, you know, where, how to get started.

Training, Access, and Getting Started

And then I always think it's a good idea to work with a health coach at some point, even if it's someone you're not seeing, you know, every day or every week, someone that can help guide you and kind of get you going in the right direction.

I think the health coaches are so important for best outcomes. And then, look, if you have symptoms, you want to see a physician like Doctor Charlene or like someone who is, who really has good experience.

And I always ask, find someone who can tell you, yes, I have improved symptoms in patients, not just follow them as they went downhill, but I've actually seen people improve their symptoms with these biomarkers that you were talking about, things like P tau 2017.

You can actually now follow to see is the person going in the right direction. It is a very exciting time. And you know, our colleague Dan at Florida Atlantic University, reported on a case and course.

And again, individual results may vary. but, Doctor Richard Isaacson, who headed up the Alzheimer's prevention Center at Weill Cornell in Manhattan and now joined his brother, Stuart is a Parkinson's expert in, Florida.

But he had a case where he used the passivity ad to test and was able to track over time, that the individuals scores were getting lower and lower, consistent with the disappearance of these pathological proteins.

So great to see. It is it's wonderful for all of us. And it's really, not just, you know, tip of the hat to Doctor Isaacson, but also to show that, you know, we truly are in the age of biomarkers.

And as you probably know, the Alzheimer's Association just released their most updated position paper. basically going through what are the diagnostic criteria?

And really going back since about 2013, they started to embrace these biomarkers. And there's been a little shifts. But it to my knowledge, the most significant change in the 2024 criteria is that they have embraced the blood biomarkers now.

But, the tell 2017 and all that. So folks, I want you to understand that, you know, I think I can speak for Doctor Bredesen to say that we want this information to be available to everyone.

We don't want this information to be just for sort of the rich, the elite or whatever, those who can afford it. we want. Yes. There are things that can be a little more time, a little more, demanding and a little more, demanding a professional, you know, expertise in those things can, raise sort of the price tag of the service that's provided where the the cost of the tests.

But that being said, there are things that you can do at every stage, at every level. and even Medicare has their, cognitive assessment, CPT code 99249283.

I think it is. But at any rate, that technically allows the practitioner to run a panel of blood work and to sit down with you as a patient and really go through not only what are the findings, but a cognitive care plan that allows the physician to be reimbursed for those services.

Now, is it the medicine protocols, the Charlene protocol? Probably not quite as detailed, but it is a value nevertheless. we just need more practitioners that know that that code is out there and then have the toolbox to actually show the patients what to do and then to help them implement that, maybe with a health coach, an excellent choice.

Yeah. That's a that's a great point. You know, one of the things that's exciting is with these biomarkers, you can actually see, just as you said with Doctor Isaacson's patient, you're literally seeing that the person had underlying Alzheimer's.

They just didn't have much in the way of symptoms yet. But now you're seeing the biomarker showing that this process, this ongoing pathophysiological process is now going down and is no longer in the range, as he pointed out.

And it gets to the point where it's no longer in the range you associate with Alzheimer's disease so that you had something off long before. It's almost like, you know, seeing something way down the road and preventing an accident.

so I think these are going to be valuable, and I think we will also get more information, seeing what has actually helped, what actually move the needle, what didn't move the needle, that sort of thing.

So that's very exciting. We should get to a place where neurodegenerative symptoms are relatively rare, that people get this picked up just as you'd have your blood pressure checked, just as you'd have your cholesterol check those sorts of things, you know, you will have these markers checked and you say, okay, you know, you were going to get Alzheimer's 15 or 20 years from now, or Parkinson's or Lewy body.

But now we're going to make sure that you never get that. And I think that's going to change things. It's definitely and this this is a lovely way to even circle back around to the anti amyloid therapy that we started talking about.

Not that I'm telling everyone to go out and do that. But when we look at the data the way that, the Donanemab trial, was structured, and when they looked at the subset of individuals who just had mild cognitive impairment but didn't meet full clinical criteria for Alzheimer's, their outcomes were twice as good as the people who were already in that Alzheimer's stage.

Does that mean that the treatment is the right treatment for you? Not necessarily. But what it says to doctor reticence. Point is that early intervention, you know, early intervention is key.

Early intervention with diet, with sleep, with movement, with a stress resilience practice, with identifying and eliminating toxins, including infections, as well as heavy metals and other chemicals.

Cleaning up your your environment, cleaning up your body, getting rid of this stuff makes the the biggest difference it's made when we catch this early.

Absolutely, yeah, and I look forward to the day when there's just a simple neurodegenerative panel where you and I know there are some neurodegenerative panels now, but I'm talking about one that would be the earliest changes would probably include a, you know, sweat related, mass spec test, which can be done fairly quickly.

It would probably include p tau 217 it would probably include, you know, markers for ALS as well, that sort of thing, and say, okay, you have a clean bill of health.

You're not headed for any neurodegenerative conditions down the road. Or someone say, oh, yeah, you were actually headed for Parkinson's, but we're not going to let you get there.

Symptomatic early. this will, you know, really change health care. I think it's it will it will happen. We're starting to see evidence of that already.

So it's a very exciting time. I hope we all live long enough to see it come to fruition. I do as well. Dr. Dale Bredesen, thank you so much for being part of The Parkinson's Solution Summit 2.0.

I know people are very excited about your protocol. have many patients who, come to see me. You've either already read your books or watched you online, or maybe had the opportunity to hear you speak in person.

if folks do want to get involved with Apollo and your Protocol and what what should they do? What should they. thanks for asking, Ken. So lots of ways.

So you can go to mycognoscopy.com. So that'll that'll look at the testing and how to get those. then there's getabrainscan.com which is So this is just to give you the details of the most sensitive PTL is through that group, and that's through, a company called Neuro Code.

They have the most, currently the most sensitive, PTL, NFL, and GFP. you can, you know, you can go to, looking at one of the books, there's, there's, the, The End of Alzheimer's, End of Alzheimer's Program.

And The First Survivors Of Alzheimer's. you can look on Facebook, Dr. Dale Bredesen. same for Instagram, same for Twitter. So any of those social networks looking, I guess now at Twitter now called X, any of those, networks to get more information and you can look at our published papers.

we have many published papers that we published, over 230 papers. and a number of these are available publicly, freely available online. so lots of ways to get additional information.

And I encourage everyone, be active if you are 40 years of age or older, and especially if there's any history of Parkinson's or Lewy body or Alzheimer's in your family, get evaluated, get a Cognoscopy or see an expert like Dr. Sharlin.

get evaluated so that we can have a world that has less neurodegenerative disease. It has been a huge, huge problem, as you know. Well, thank you so much.

Look forward to seeing you soon. I think, by the time this airs it will be past. But I think hopefully we're going to see each other this coming, October in Washington, DC.

So fantastic. You're scheduled to speak as well as myself. Have a wonderful rest of your day Dr. Bredesen. You too. Take care. Thanks. Bye. Right.