How To Protect & Strengthen Your Memory And Thinking If You Have MS Transcript

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Introduction to Dr. Bredesen and the interview

Welcome to another episode of the M. S. and Autoimmune Summit. I'm Doctor Terry Wahls, your host, and I'm pleased to tell you that we're going to be interviewing Doctor Dale Bredesen.

Now Dale Bredesen. I graduated from Cal Tech, received his medical degree from Duke. He trained with the Nobel laureate, Professor Stanley. Prisoner. It was the founding president of the Institute for Research on Aging.

His research is that of the 230 published peer reviewed papers and the first reversal of cognitive decline in patients with the Alzheimer's disease using a precision medicine protocol.

He's the author of two New York Times best sellers, and is the senior director of the world's first precision medicine program for neurodegenerative diseases at the Pacific Northwest Neuroscience Institute.

So welcome, Dale And I'm so pleased to have this interview. So Doctor Brodersen, I'm so glad we have the chance to have this interview. You know, because cognitive decline and brain fog are increasingly recognized as an issue for people with multiple sclerosis.

And the longer we have our illness, the more likely it is that we're going to develop brain fog. So I know you are so well versed at dealing with Alzheimer's.

Shared mechanisms behind MS and Alzheimer's

I'd like to talk a little bit about how what you see is the similarities between the cognitive decline that happens in Alzheimer's and the cognitive decline that happens with MS..

Yeah, such a good point. Doctor Wahls, thank you very much for for having me. So in Alzheimer's, what is seen is that the three major players are anything that induces inflammation.

And it's typically innate immune system. It's typically not adaptive immune system. And you've got this chronic activation just as we get in trouble with cytokine storm in Covid we get in trouble with cytokine drizzle.

In Alzheimer's disease it's a long term mild activation. So to some extent you could argue that Alzheimer's is essentially a chronic mild innate encephalitis.

And that that's what's driving the problem. Then of course the second one is reduction in energetics. So blood flow decreases sleep apnea, mitochondrial dysfunction all that.

And the third big one is toxicity. And so mycotoxins, bio toxins, inorganic toxins like air pollution and mercury and things like that. And then and then organic toxins like glyphosate and, and toluene and benzene and formaldehyde and all those sorts of things.

So those are the three big players in Alzheimer's. Now, you know, the big difference, as you pointed out, people with Ms. do have brain fog, and they do have cognitive effects over the long haul.

And this I remember years ago when I was first taught that with Ms.. You can impact the number of plaque form formed without necessarily impacting the fact that people could still get cognitive decline in the future.

So they seem to be to some extent have at least some non-overlapping mechanisms. And the big difference between Ms. and Alzheimer's is that Ms. involves the adaptive immune system much more.

So it's all about autoimmunity. Whether you believe it's mostly a response and, immune response against, I've seen Barr virus or whether you believe it can be many others as well.

I mean, whether you think, you know, the the cross reaction is with glial cam, whatever you believe, it clearly involves the adaptive immune system. Whereas with the Alzheimer's, Alzheimer's, we're really talking more about the innate immune system.

Yes, some people do get some autoantibodies, but in general, that's the big difference between the two. And of course one's targeted more in the white matter.

One's targeted more in the synapses. Essentially, for Alzheimer's, it's essentially synaptic process. And you know, and one is of course, is tends to be a little bit younger in people.

One tends to be a little older. But of course, as we understand these conditions better and better, they have more and more similarities. They're both what I call defamatory diseases.

They're degenerative and inflammatory. And we now know that the Alzheimer's one starts much earlier, where there are people with changes in their use.

M30s. Whereas we used to think of Alzheimer's as old timers and Ms. as the young person's disease, now it's looking like they both have all ages. Yeah.

You know, and I and I do think there's considerable overlap, but there's certainly recognition that the more toxicities you have a more aggressive a mouse.

You have earlier Ms.. That cigaret smoke air pollution mercury all make asthma symptoms worse.

Glutamate and neurotransmitter decline

And that the chronic drizzle as you beautiful that you put with the cytokines can activate the microglia in the brain and that can accelerate the development of enhancing lesions and accelerate the degeneration that happens with Ms..

That begins around age 45 when we have fewer new enhancing lesions, fewer relapses. But we have this what looks much more like the Alzheimer's picture, the degenerative side.

Yeah. And of course, there's there's some evidence that that involves glutamatergic, signaling as well. There's some glue to make a contribution to the cognitive decline in both cases.

Let's explain what glutamate is for our listeners. Right. So glutamate is a neurotransmitter that is used. We always think about acetylcholine because that's the most important one for memory and the one that is low in Alzheimer's.

But there are a number of other neurotransmitters of course we hear a lot about dopamine. There's norepinephrine. There are various peptides like somatostatin, which has also been shown to be, low in, Alzheimer's disease.

And so these things are, are communicating between the neurons. And the big one for so-called excitatory neurotransmission is glutamate. And so if you look, for example, at your ability to activate your muscles, this is a glutamatergic form of neurotransmission coming down through the spinal cord ultimately, of course, and then out to the muscles to activate them, which then that part is as cholinergic.

But so in Alzheimer's, you do see this enhancement in glutamatergic signaling. And that is also been implicated in cognitive decline, associated with Ms..

Okay. So what do we do about that the decline of these neurotransmitters. Yeah, a great point. So I mentioned earlier that, you know, the three big players in Alzheimer's are inflammatory inflammation and toxicity and

energetics.

Addressing inflammation, energetics, and toxicity

There are three smaller players. And that is neurotransmission, trophic activity from things like nerve growth factor, brain derived neurotrophic factor, hormones and nutrients and then stress.

And that's the other big one. And stress impacts, no question, impacts so many of us when it comes to optimal cognition. So what you really want to do is look at this in the certainly in the case of neurodegenerative conditions.

And you can again look at Ms. as is having a big neurodegenerative component ultimately just as you mentioned. But then what you want to do is bring up the supply and bring down the demand.

So we want to improve the neurotransmission. We like to enhance the trophic support. We'd like to improve the energetics. We'd like to reduce the stress.

We'd like to reduce the toxicity. All of those things are critical. And you need ultimately to get to of course, what's causing these. And this is why we always say, well, at the beginning, go ahead, reduce the inflammation.

But don't forget that there's something there that's causing that. And ultimately get a long term positive outcome. You're going to need to identify those things, and then you're going to be able to you're going to have to do something about them.

You know, one of the things Doctor Christine Burke, who's one of the people who utilizes our protocol, pointed out that she sees about 9 to 12 months of improvement just with addressing the basics.

But to get long term improvement, you have to identify what's actually contributing. So if it's a pathogen, you got to identify the pathogen and get rid of it.

It might be a tick borne infection like a Borrelia, Bartonella, but Baeza Alicea and a plasma, it might be a change in the oral microbiome. It might be a leaky gut.

I mean, there are lots of ways to change this. And doctor walls you might have seen recently. Very interesting article looking at the microbiome in the brain.

And there were two big surprises. One was that the brain microbiome is present in normal. So in other words, there well, what I was always taught was supposed to be a sterile organ.

The brain not supposed to have bacteria in your brain. Well, it turns out that's just not the case. Normal brains have their own microbiome. Okay, let's stop for a moment.

This is really huge. I was certainly taught the brain and the bloodstream are sterile. But you're telling me that there are bacteria? I knew there are viruses in my brain.

There are bacteria. And viruses in my brain all the time. Yes. I will send you a very, very interesting article going through. And what they did was to sequence these various things and find out where in different regions of the brain.

And to me, as I said, two big surprises. The first one was that normal brains have a microbiome. The second window and there are some differences.

Brain microbiome and oral health

And they looked at Alzheimer's and they looked at Parkinson's, and they looked at the changes in the microbiome. The second big surprise was that most of the organisms that are part of the normal microbiome of the brain are oral

micro organisms.

So these are mostly associated with dentition. So no big surprise when you have really poor dentition a lot of period on Titus you have an increased risk for cognitive decline.

Okay. Hang on. For the listeners we need to tell them what periodontitis is. Yeah. Periodontitis. So this is where you get damage in your in your gums where ultimately you can lose teeth.

And this is, you know, around the tooth typically and ultimately these are the things that are invading into here. And you can actually things like p gingival s and T identical.

These are organisms that cause tooth decay and gum damage and ultimately can get into your brain. This is huge. So if I have a healthy microbiome, because I've been recently testing the oral microbiome of my patients because I, I've really gotten tuned in that there's a lot of benefit for checking the oral microbiome and fixing that for the health of my brain.

And by the way, for pregnancy outcomes and not having early premature delivery. So oral microbiome is very helpful. We've known for a long time that the stool microbiome is helpful, but I'm so glad that you're telling us there's a lot of reason to test our oral microbiome.

Absolutely. We test that on all of our patients. We're right in the middle is, you know, of a randomized control. Let's talk about this trial because it is so exciting.

Yeah I'm very excited about it. So this is this will be the first randomized controlled trial of precision medicine that to show if, if it continues as it's currently going to show a statistically significant difference between outcomes when you use the protocol we developed versus a precision medicine type of protocol versus standard of care.

And currently, we're we're now about nine months in. This will finish late next year. But what we're seeing is that the people on standard of care are not improving.

And the people on the precision medicine protocols, as we've seen before in an earlier trial. And we're seeing an improvement once again, which is very, very exciting to see.

And so these are for people with cognitive decline, a cognitive impairment or diagnosed Alzheimer's. So these are people who have cognitive decline. Many of them have Alzheimer's.

Some of them end up with other things. Some of them end up having Lewy body or vascular dementia or things like that. If if they have a known gnosis of something other than Alzheimer's, we exclude them.

Precision medicine trial for cognitive decline

But for those who just have MCI, for example, these are people and we're taking people with Moca scores of 18 and above, which mean you can see an early dementia.

So explain the Moca score. So the Moca score, it's Montreal cognitive assessment. These go from 0 to 30. A normal person will typically score 28, 29 or 30.

When you're getting down below that, you're into the next phase, which is mild, so-called mild cognitive impairment. And that goes down to about 2122, depending on how your activities of daily living are, whether you're able to take care of yourself, etc.

right in that area, you transition from mild cognitive impairment to dementia. So we're taking essentially the early dementia, as the average for all Alzheimer dementia cases is 16.2.

So when you're getting down below that, you're getting down to, you know, things like single digit Moca scores.

These are people who are in late stage dementia from Alzheimer's disease.

So we're taking the ones that are early stage dementia and late MCI. So what's the average Moca score in your group? The average is coming out around 2223.

Those are kind of that's kind of typical. So so we go down to 18 and we go up. You know anything above 18. Very exciting very exciting. And so this will finish at the end of next year here.

And what have you completed all of your enrollment? No, we got we've had about four more. So we've got about four more to enroll. We're down coming down to the very end of the enrollment, but we're definitely looking for a few more.

And there are at six sites. So if you're within an hour of one of those sites, it's Hollywood, Florida, with Doctor Craig. Tonio. It's on, Nashville, Tennessee, with Doctor David Hassey.

It's Nate Bergman at Rocky River, Ohio, and then Doctor Christine Burke out here in Sacramento, California. Doctor Anne Hathaway right here in Marin at San Rafael, and then doctor Cat Toups, who's out in the East Bay.

Very exciting. And what's the total? And you're trying to get to 72. So that's where a good number. Yeah. You know and yes it would be great to do a thousand.

But the reason that people have typically done so many in these drug trials for Alzheimer's is because the effect is so small, so smaller the effect. But as you well know, having done the trials yourself, the more you have to do so, the effect that we've seen is fairly large, and therefore we only need to do 72 to show that there's clear statistical significance.

So the what is the control arm get so the intervention arm gets you know basically the breadth. So we're looking at all the things we talked about. We're looking at inflammatory things at pathogens, at toxins at oral DNA.

And we're actually looking at cone beam analysis to make sure that people don't have hidden abscesses. We're looking at sleep apnea. We're looking at stress levels in their RVs.

We're looking at all these different things of course methylation homocysteine gut. So we're looking at all the things that actually drive the decline.

And then we're the we're directing the treatment against those. So it's a personalized precision medicine approach. Now the standard of care gets standard of care.

Now the standard of care is that you don't look at all those different things. You assume that we don't know what causes Alzheimer's and you give them aricept, which is typical if you have dementia.

Now, interestingly, aricept is not recommended for those with MCI until they have dementia. However, of course many people do use it off label, but we're going strictly by the standard of care criteria.

So if you have a Moca of 20 or less, you get the Aricept. And then. And how long is the intervention period? Is it a year? It's nine months, nine months.

So we have just the fact that we know it's been a rolling admission. So as soon as we get our last one, it'll be nine months beyond that. That will be finished.

And then are you anticipating that the people in the control arm, do they then get access to the intervention arm? They do. As soon as they sit their nine months in the control group, they get six months of free care with all the same sort of things.

Yeah. So there's a tremendous reason to get into the study so that you either get the intervention or you wait six

months and you get the intervention.

Now, for the losers, that's called a waitlist control. But we do that in our studies as well, that people can get the intervention. And then at the end they get some access to the intervention.

New blood biomarkers for early detection

No, I know that you had been talking about a new blood test that is super helpful for people who are wanting to avoid the risk of Alzheimer's and cognitive decline.

Could you tell us about that? Yeah, thanks for mentioning that. I actually think this is one of the most important advances in years, if you think about it.

You know, again, way back when I was training, we were told, oh, you have diabetes or you don't have diabetes. And of course, then people came up with hemoglobin A1, C and then fasting insulin.

Now we can look very early. So you don't have to wait until you have diabetes. In fact, very few people need to get it. You can look early, or at least for type two diabetes, you can treat it.

You can make sure you never get all these horrible consequences of long term diabetes. Well, now the same thing has happened with Alzheimer's disease.

So we can all get these tests early. They will show you where you're headed if you're headed to Alzheimer's even before you have symptoms. And it really is allowing for the first time for all of us to say we don't have to get Alzheimer's.

This is now becoming optional, as hard as that is to believe, we can actually now see it coming and headed off so that nobody needs to get this. And there there are three blood tests that are using these.

We've looked at, you know, the group that's doing the most sensitive ones of all is a group called Neuro code. So we've worked with them to produce something called brain scan.

So you can actually get this directly. I had it actually, I had it done myself just a couple weeks ago. And they actually came to my house, drew my blood.

It was very simple. So you go to get a brain scan.com and you sign up, they they can come and check it for you. The three things are very complimentary because they tell you three different pieces of information.

So for the first time you can really get a beautiful look at the biochemistry going on inside your brain and make sure that you don't have future cognitive decline.

So the first one is called p tau 217 and p 217 is specific for Alzheimer's and pre Alzheimer's. So you can pick this up early. You never have to go on to get dementia which is wonderful.

The second one which is complementary to that is called GFP. That's for glial fabulous acidic protein. What happens there is that when you have problems in the brain, you are astrocytes.

The supportive cells basically puff up, get big and start to work. They're they're there to be part of the inflammatory response, and they're there to be part of the rebuilding response.

So they are helping you. And these things, when they puff up, they make more of this GFP because that's actually part of their intermediate filaments.

So you can pick that up in the blood and see if you've got ongoing inflammation from any cause in your brain. And then the third one is called Nflx neuro filament light.

And what that one does is it's part of the neurons, not the astrocytes. But what it tells you is whether your neurons are compromised for whatever reason.

It could be that you had a car wreck. It could be that you have frontotemporal dementia, it could be ALS, it could be it could be Alzheimer's. So, for example, when I look at people, if I see that their their tau is high, but the other two are normal, they have a relatively quiescent Alzheimer's.

And we want to then follow. You can then use that to follow over time as you bring it down, which is wonderful. So we can see, yes, we're on the right track or no, we're not on the right track.

We have to add more things. If I see that they're also they're GFP and NFL are high. That tells me they have a more active form of Alzheimer's, and we really have to get in and pull out all the stops just to do everything possible to get these all to come back to normal.

If your p tau is normal, on the other hand, but the other ones are high, that tells you something else other than Alzheimer's is causing the problem. It could be frontotemporal dementia, could be Lewy body.

There are lots of things, but we need to look further. But we know it's not all timer's, so we certainly know that the neuro filaments and g fab are two really big markers in a mouse, but tau is not is very specific towards Alzheimer's.

Exactly. So again, if people want to get this, you go to get a brain scan. Yes. Get a brain Nasscom and you can do it. There's there are local draw stations all over the country.

There are all there also are mobile phlebotomy that can come to your home or your place of work, whatever you like. I think this is like knowing your blood pressure, like knowing your lipid panel.

It's something we all should know because it tells us, it warns us ahead of time, helps us to avoid the horrible years of nursing homes, etc.. Yeah, avoiding Alzheimer's.

Now I'm thinking that this may be age related, that as you're older, the neuro filaments and you Febs are anticipated to go up. Yes. No. Oh it's interesting the so the normals do change a little bit.

On the NFL's just as you said. So yes. Your NFL goes up slightly. Interestingly, the G fab doesn't change much and the p tau doesn't change much. So it really gives you a pretty good marker to know at any age where you stand.

So you know, anywhere from your 20s to your 90s. I always argue that, you know, your most important asset is a functional brain. If you don't have that, then then nothing much else matters.

So this is why you such an important thing to to have it for everybody, to make sure that they don't, don't suffer cognitive decline. And how often would one I would recommend people getting this particular test.

Lifestyle factors that protect brain health

Yeah, such a good point. So every five years between 35 and 60. So 35, 40, 45 etc. up to 60 after 60 you want to get it every two years. And of course if you start noticing cognitive decline then you want to get it at that time.

But if you're asymptomatic, then every five years up to 60 and every two years after 60. Now what what I've been reading is that people are being diagnosed with mild cognitive impairment much, much earlier in life.

We're seeing some of that, and there is occasionally in the 40s. Is that what you're seeing as well, though? You know, you hit the nail on the head. It's so true.

So when I was training, you know, way back 40 years ago, we never saw people in their 50s with Alzheimer's. This was late 60s, 70s, 80s, 90s, so-called old timers disease.

Now, not only do we see people in their 30s and 40s, one of the most common things we see is people in their early 50s with full on, you know, Alzheimer related dementia.

As you mentioned, MCI, we see that in the 30s and 40s fairly frequently. And of course, there's been some enhancement of that with the pandemic. As you can imagine, anything that gives you this chronic inflammation is increasing your risk.

So Covid and not just Covid infections, sometimes we see people declining after Covid vaccines. Again, it's inducing this chronic state of inflammation.

So this is again why it's so important. And doctor Walls, you may have seen this surprising paper recently where the group was trying to study the effect of exercise on memory.

What they found was there was a difference in the ability to learn and to build a store memory in people who were ApoE e4 positive. That's the common Alzheimer's risk genes in their late teens.

Already you could see a difference, which is shocking, but tells you the biochemistry is different even then. So it again, it's very important for everyone 35 or over to get evaluated, see where you stand because there's so much you can do about it.

Wow. Well, well, let's begin to talk about what are the things that, you know, our listeners. Because again, because there's so much in common between mild cognitive impairment, Alzheimer's and M. S., what are the things that under my control that will either accelerate my risk towards mild cognitive impairment or can may help protect me from the mild cognitive impairment right.

So as I mentioned earlier, the big players are inflammation, energetics and toxicity. The lesser players are neurotransmission, trophic activity and stress.

And so addressing those. So step one is finding out. And now you know this is why we recommend everyone please get a Cognos copy. Just like you know, you get a colonoscopy when you turn 50.

You want to get a Cognos copy if you're over 35. And so including, you know, your brain scan to know where you stand. And then the Cognos copy tells you why you're there, what are your risk factors.

And then and as you kind of alluded to, you can address these things. And we we think in terms of two stages. Stage one is the seven basics. And stage two is the two groups of of specifics.

So the for the basics everybody can do a plant rich mildly ketogenic diet very similar to what you recommend. And with know you know, a high group of, you know a plant dominant diet, high phytonutrients, high fiber, great at detox, great at improving your lipid profile, great at improving your glycemic profile, all those things.

And which doesn't mean that you, at least in our case for Alzheimer's. Does it mean you avoid meat and fish that you want to have grass fed beef? If you're going to have some, you want to have some pastured chicken, pastured eggs, wild caught low mercury fish.

Those are key. But it's mainly is a plant rich, mildly ketogenic diet. And then exercise, sleep, stress. I check my sleep every night and see how much every morning when I wake up.

You know, what's the total sleep? You want to have? At least seven hours. What's the REM sleep? You want to have at least 1.5 hours. What's the deep sleep you want to have?

At least one hour. And what's the oxygen saturation? You'd like to be at 94 and above, if possible. So all those things tell you a lot about where things stand.

Then you want to check your, you know, HRV during the day, see how much stress you're under and things like

that. So it's just to go back over them. So there's so it's going to be diet, exercise, sleep stress brain training is number five detox and break brain stimulation in general.

Whether you do it with brain training photo bio modulation with some light therapy, all of these things are stimulating your brain. Now to stimulate it.

Remember you want to support it too. So you'll want to do the other things. Correct. Have appropriate nutrients. The key for this diet is to get you into a state where you are metabolically flexible.

You can go back and forth between making ketones and making glucose, utilizing ketones and glucose. What happens? For most of us, our neurons are only going to work with ketones or glucose.

But as we get a little older, even over 40, we're starting now to lose both of those. The insulin resistance that's so common is losing the ability to utilize insulin appropriately, and it prevents you from making ketones.

The Ageless Brain and closing remarks

So it's really a double whammy on your brain function. And we want to bring both of those back by making you insulin sensitive, which now allows you to make some ketones.

And I usually recommend just take some ketones at the beginning because it's, it's giving your your brain the energy it needs. So those are the seven basics.

Then this two groups of specifics are pathogens and toxins. We want to know in all these chronic pathogens do you have herpes simplex. Do you have EBV.

Do you have you know, p gingival. Do you have one of the tick borne illnesses? A lot of these chronic pathogens are a cause of chronic inflammation and therefore leading to some degeneration in the brain.

So we want to get want to identify those, get rid of them. Want to optimize your immune function of course. And then the final one is looking at the toxins that I mentioned earlier and then detoxing from those.

If you do those things, virtually everyone does very well. Now the later you wait, of course, the harder it is to get things all the way back. Now, one more thing I want to catch before we wrap this up.

You have a new book. Yeah, that we'll have that will have come up by the time this airs about. Tell us about the book because I encourage people to go pick up this wonderful book.

Yeah. Thanks so much, Terry. So the new book is called The Ageless Brain. And the idea was everything that we learned over the last 30 years in the lab and then with the trials with Alzheimer's disease, we want to be able to translate that for everybody.

So how can we all make sure that our brain span is just as long as our life span? As, you know, a big problem in Western medicine is that the life span is fairly long and the health span is fairly short.

So we have a long sick span. After our health span, we want to lengthen that health span. And the same thing with our brains. We want to lengthen our brain span so that we are we are doing well with our cognition throughout our lives and with what we now know.

Virtually everyone should be able to do that. Checking the right things, getting on active prevention, optimizing things, having access to blood tests, having access to follow up to genetics.

All these things are wonderful now. So this book is really about how to keep sharp for your entire lifetime. It's a wonderful book. And so we're going to encourage everyone to pick up that book, because a lot of the things that you're going to learn in that book are really applicable for the community as well.

Yeah, absolutely. Okay. Dale, where to? People find you if they want to learn more. Yeah, you can go to Doctor Brothers Incom, or you can go to, look at on Facebook on x, on, Instagram.

Any of those, at, doctor Dale Bredesen. And look at any of those you can follow there and you can look in the books on the we have three, three different books out of is that Terry mentioned a fourth one coming out.

This is the end of Alzheimer's, the end of Alzheimer's program, and then the first survivors of Alzheimer's. So all of those are good ways to see what we're up to.

They're all wonderful, wonderful books. So get them yourself. Give them to the people that you love and care about. Thanks very much.