Introductions and Dale Bredesenu2019s Background

Welcome to the MS and Neuroimmune Summit. I am here with my very good friend, Dale Bredesen. Now, Dale and I have known each other for many, many years.

And we have, a functional medicine approach in his case, to Alzheimer's, in my case to Multiple Sclerosis. But there's considerable overlap with what we do now.

let me read Dale's, wonderful bio here. He graduated from Caltech, received his MD from Duke. He trained with Nobel laureate Professor Stanley Prusiner.

It was the founding president of the Buck Institute for Research on Aging. His research led to over 230 published papers. quite a few more than I have.

in the first reversal of cognitive decline in patients with Alzheimer's, using a precision medicine protocol that really is based on the functional medicine principles that Dale uses and I use in our clinical practices.

He's the author of two New York Times bestsellers. He's the Senior Director of the world's first precision medicine program for neurodegenerative diseases, which is in the Pacific Neuroscience Institute.

Did I miss anything, on your bio, Dale? No. It's perfect. Thank you. Terry. Always great to talk to you. So let's talk. first, I really, like, started off with a recovery story.

you you have many, many, that you've seen people with significant cognitive decline who were able to reverse. Could you give us an example of one of these patients?

Absolutely. And, you know, we have a paper. In fact, it's it's just been posted. So you can actually see the preprint on people who have sustained their improvement, some of them for over a decade.

So let me mention Sally is one of many. and she was someone who was a nursing professor. when in her 60s, she began to have problems. She, she got to the point she forgot to pick up her granddaughters.

she could not she could not put together a gingerbread man anymore. she was struggling just in her everyday activities, so she had pretty significant problems.

she then went, actually got evaluated. she had an amyloid positive Pet scan. So her diagnosis was Alzheimer's. And now Alzheimer's is a pathology. So it was her stage of.

It was the third of four stages, which is called MCI, mild cognitive impairment. So she was fairly impaired at that point but still able to take care of herself.

She went on a drug trial. She found, by the way, that she was ApoE4 positive. There are 75 million Americans who have one copy of ApoE4. 7 million who have two copies.

that's the most common genetic risk factor for Alzheimer's. So by all criteria she had that she scored a 24 out of 30 on her Moca test. Which means for someone who's a nursing professor, that's significant. MCI.

Alzheimeru2019s Recovery Case Study

she started on treatment. That was to remove the amyloid. And with each injection, she actually got worse. Now, to be fair, she didn't. She wasn't told if she was in the control group or the treatment group, but something clearly was

happening.

We've heard this story before. She got worse with each injection. After about eight injections, she said, this is not working. This is making me worse, not better.

she contacted me. we we evaluated her. And actually, it turned out that she had very significant exposure to mycotoxins, something that is not even recognized at this point by mainstream medicine as being a contributor.

But our research over the years showed that anything that decreases energetics, increases inflammation or increases toxin load can contribute to this network insufficiency that we call Alzheimer's disease.

So she went on, and just as you said, a functional medicine sort of approach. And we very much came to this from test tube research. which is why I believe in it so much.

The research drove us here. She went from 24 to 30 perfect scores on her markers. she's able to pick up her her grandkids again. She doesn't forget she can do gingerbread men.

She remembers things. Interestingly, six years of doing great. Then she had some problems again. Okay, she was reevaluated. She had gone down on her testing scores.

She was reevaluated. It turned out she had three new things. Number one, she had some sleep apnea that hadn't been nearly as bad before. Number two, she had Cryptococcus lorenzi in her sinuses, which is relatively uncommon.

And number three, she had a new leak in her roof with new growth of mold, new exposure to mycotoxins. Those three were treated. And interestingly, her scores are now higher than they were even when she was treated the first time.

So she's now seven years doing absolutely great. Okay, so what? Before we start our interview. You and I were talking about the similarities and the differences between Alzheimer's and multiple sclerosis.

And for the listeners, what when I was first diagnosed in 2000, my neurologist said, don't worry. Her cognitive decline is not an issue for people with MS.

That has since been modified, to recognize that cognitive decline can be become an issue. in brain fog can become a problem. Why people end up becoming, losing their employment and that, cognitive decline, certainly is an issue.

So let's talk about what you see as the similarities and the differences between Alzheimer's and, MS. This is such a good point, Terry, because, you know, we tend, as physicians and scientist to be in siloed.

We're going to study MS. Or we're going to study Alzheimer's. I actually think you learn more about MS. And Alzheimer's by comparing and contrasting the two.

So what happens is there are many related pathways. But what's very interesting to me is when you get Alzheimer's, what we see again and again and these these are both what I call defamatory diseases.

There's inflammation. There's degeneration with MS. It's inflammation. Degeneration with Alzheimer's. It's degeneration is more than inflammation. But they both have both.

And why are they different. So what happens is with MS. you actually go through you've got the of course you've got the your innate immune system activation.

But now you have your adaptive system and of course you have autoantibodies. And of course, you know, things like reactions against both, ng gcam and Epstein-Barr virus, for example, as one example.

Whereas with Alzheimer's, these are typically older people. You're not getting the adaptive response. So the problem with Alzheimer's is you have an ongoing mild innate response, usually with very little of the adaptive response.

Whereas with MS. it tends to be younger people. They have a more brisk the ability to make an adaptive response. And the problem is that it is a wide adaptive response.

So in other words, you're not just targeting one thing. You have auto antibodies. So there are two different neuro immune abnormalities, both leading to degeneration and inflammation okay.

So for the listeners, for us, we have a lot more inflammation driven damage, particularly early on, in the disease. But behind all that inflammation is this slow, progressive degeneration that is going on, going on invisible at first because we have so much inflammatory damage when we get older, over the age of 45, there's not as much inflammatory damage now that progressive degenerative damage is very visible and becomes more and more visible over time.

And I think what I heard you, found, going to translate correctly with, Alzheimer's. There's not as much inflammation damage in the brain. There is a slow, progressive, degenerative damage that is accumulating and becomes visible.

I see there are some really young cases in their late 40s and 50s, but most often the Alzheimer's is visible. I would say in the 60s. It's such a good point.

I'm glad you brought that up, because when I was training in a way, way back in the 1980s, we never saw people in their 40s and 50s with Alzheimer's disease.

Now it's the one of the most common things we see is a 52 year old person developing Alzheimer's. So what happens is that in your right, the the disease, Alzheimer's tends to manifest in terms of symptoms in people in the in general, 60s, 70s, 80s, 90s.

Although we as I say, we see a lot of people in their 50s and sometimes even 40s. But what is very clear now from blood tests, from Pet scans, from spinal fluid analysis, this disease begins biochemically about 20 years before a diagnosis.

And by the way, there's a wonderful paper came out recently in the New England Journal of Medicine from a group in China that followed people, thousands of people for years and watched who was going to get dementia, who is not going to get dementia.

And then they showed that on average, when the changes occurred in the blood, in the spinal fluid and the Pet scans and showed that, for example, the A beta changes in the spinal fluid were on average 18 years before a diagnosis of Alzheimer's disease.

Now with these new tests, p tau 217 being the most important one you can actually see in the blood, years ahead of time. And I recommend we also I'm getting my next week.

Alzheimeru2019s and MS: Shared Mechanisms

Actually it's just become available a very there's one group that's doing a highly sensitive form of p tau 217 so that we can all avoid dementia. So yes, you're right, it's it's different ages.

But it does. These things start much earlier in life as you know. And again for the listeners with the M. S. in their immune issues, these, degenerative changes are likely happening to us two. And, that is why we two are at risk for, brain fog, difficulty with our memory, with our recall and some cognitive decline.

Now, what what you're what you're doing in your Alzheimer's. And what I'm doing in my Ms. patients is evaluating all of these, metabolic pathways, these risk factors, and seeing what people have and then addressing them one by

one.

And what we see is mental clarity begins to improve. their Moca scores, improve or the mini mental status scores improve. what I thought might be very helpful, for people is to begin to understand what some of these, metabolic pathways are that, you know, are so important for that risk of, degenerative disease, and neuroinflammation, disease that, that lead to our cognitive decline.

Yeah. Such a good point. And so in the laboratory of 30 years ago, we began by looking at what is the signaling that is involved. And if you look, for example, everyone knows that, you know, amyloid accumulates in the brains of patients with Alzheimer's.

Well, where does this amyloid stuff come from and what is it for? And so what it turns out is this is something that is part of your innate immune system that is actually produced by your brain cells mostly, but other cells as well.

and it is a an anti-microbial peptide. So it is a little bit like when you would put an insect in amber, you are sequestering. So it's a, a sequester and, and an antimicrobial peptide that will now get rid of things that are invading your brain.

So when you have viruses or fungi or things like that, then you actually make this stuff. So, I'm thinking we know it's, increased in people with Alzheimer's.

would this be probably increased in people with, Ms.. It's a great question, and it depends on what kind of insults the brain undergoes. As I mentioned, it's really part of your innate immune system.

So what happens is when your adaptive system now gets turned on, that's the more specific part of your immune system. One of its jobs is to turn down your innate system.

So of course in Ms. you have the adaptive system that's more active, whereas in Alzheimer's it's more the innate system. So for those people who are successfully turning off their innate system, you probably wouldn't see much amyloid.

Now, having said that, this new, p tau test, which is positive in Alzheimer's, they're what they're noticing is something interesting. It's blipping up just a little bit in people with long Covid, as if the people with Covid are actually kind of beginning on a long road that could end up with Alzheimer's.

It's certainly possible that you'll see the same thing. with maybe a factor, you know, thinking about the, people with M. S. who are, having overly active response to EBV herpes, that are, perhaps contributing to their difficulty.

Might those folks have more, more, amyloid? They may. And again, it depends on how much is the innate response, how much is the adaptive response. And it also depends on type of organism.

So here's what's interesting Terry. When you have response to a neuro immune response to Alzheimer's, you're, you know, you're making this amyloid. It has a certain profile of insults that it kills.

Well, when you have a different group of insults, you make alpha synuclein that is a different kind of anti-microbial peptide. And of course, that's what is associated with Parkinson's and associated with Lewy body disease.

And then you have, other times where you make things like tau. And of course, there are tau up at these without the amyloid things like cortical basal degeneration, and progressive super nuclear palsy.

These are diseases where tau, which, by the way, is a third kind of anti-microbial in this case protein. So what we're what we've seen in the 20th century as being here's a pathology that must be causing the disease has really turned out to be.

These are responses to various insults, different viruses that you mentioned. E of course is the classic one for Ms..

do you see more? Ms.. For example, after Covid, because we see exacerbations of all the time after Covid.

We certainly see people after Covid having, pseudo relapses, they have, worsening of their disease. And it takes many more months, from which to recover.

And, I think we're going to see that there has been another sharp increase in the prevalence, of M. S., after, and, you know, 2020, I will have another major, epi studies that will confirm that.

But I expect that we're gonna see many more, cases of Ms.. You right now, we have a million cases of Ms.. Here in the U. S, I expect that number to be sharp.

Sharply up. so we've talked a bit about these antimicrobial peptides. why don't we talk a little bit more about some of those bio toxins that, you know, are a, a big factor for cognitive decline in the Alzheimer's world.

And we can draw how they may be a factor for cognitive decline in the Ms. and neuro immune world. Yeah. And I and I apologize I want to finish answering your question about signaling because that is a critical piece.

So the amyloid comes from its parent which is amyloid precursor protein. It is a molecule that sits in your neuronal membranes, especially its synapses and to a lesser extent in other cells.

And then what's really interesting about it, it's a switch. It will give you one direction, literally connection. When you when things are good, you have enough hormones, you have enough nutrients, you have low inflammation, low toxicity.

It will signal growth and maintenance. You make new synapses. On the other hand, that same molecule, when things are low, low hormones, low nutrients, low support, low blood flow, low oxygenation, and high inflammation and toxicity, it now switches you from connection to protection.

So now you're making amyloid. You're literally trying to fight with this stuff that is that is coming. And so we see it with insulin resistance. We see it with ongoing inflammation, many chronic pathogens.

All these things that change, that balance are what give you all timers.

Amyloid, Antimicrobial Peptides, and Disease Signaling

And it allows you to study it, figure out for each person what's causing the problem and then actually do something about it. Now you mentioned the toxins. Great point.

They are unrecognized in most cases as being critical contributors to Alzheimer's disease. And they come in three types. So number one is in organics.

There's a lot of work now on air pollution as being one of the most common risk factors for Alzheimer's disease. A recent study that identified the three top risk factors for Alzheimer's.

One of them was air pollution. and so that's key. Also things like mercury, these are all the inorganic. Then they're organics. So these are things like glyphosate and Tal Ewing and benzene and formaldehyde and these things again, all, assaulting the body.

You've got to get rid of them. You metabolize and you sequester them, you excrete them, etc., and you can get overwhelmed by them. And then the third group, and perhaps the most concerning, because it's so common and it also engendered that inflammatory response in addition to the toxicity, these are bio toxins.

And so for people who are living in houses full of mold, you know, some people say, gee, I never knew that mold was an issue. Well, unfortunately, the molds are trying to survive, so they are making toxins.

You know, we we obviously we use penicillin. That was an original one that that doesn't really hurt you, but they make things that are gaseous and it's mainly the, you know, just five types of mold.

It's the Starkey, Beatrice, Penicillium, Aspergillus ketone, me and Wally Mia. Those are the big five bad guys. And they are making these toxins again to try to survive.

They're trying to kill the local bacteria. That would otherwise outgrow them. And when they do this of course it's damaging us. It's damaging our mitochondria.

It's increasing our risk for cognitive decline. It's increasing our risk for inflammation. so many times we hear a whole family. One person will have cognitive changes, one person will have skin allergies, one person will have pulmonary problems, one person will have ADHD.

And they're all related to this exposure. This ongoing exposure. okay. So how would a family and everyone who's listening, you know, the same, list of toxins, you know, the air quality, the air pollution, tobacco, nuts.

Now recognize for Ms. to be a huge factor. Mercury, lead, arsenic, cadmium. Those are factors for people with Ms. and their immune issues. And, you know, the same list of, organic solvents.

Tal Ewing, benzene, glyphosate. These are, problems for the Ms. in your immune patient as well. And mold. yes. can be a problem. You know, let's let's sort of walk through why mold might be in your environment and you're not aware that there's mold in your environment.

Yeah. Great point. You know, and Doctor Richie Shoemaker, I have to give him a lot of credit for his work over the years and again, often against the establishment claims, he's turned out to be absolutely right about many things.

And one of his points is that when you have water damage in buildings and this can be in your home, this can be in your place of work, this can be where you know where you're going to work out.

Whatever, water damage to buildings allows these mold species to thrive. And again, they're trying to thrive in a tough environment where they're being outcompeted by bacteria.

as he points out, they also respond to the fact that we've treated the wood, to, to, you know, to prevent things like termites. And these things therefore, are recognizing, oh, there's toxins here.

I'm going to respond by putting out my own offensive. And so unfortunately, they make these things and they do, share plasmids and things like that. So they end up being able to make these very toxic things, trichomes, these scenes, glo toxin, okra toxin A, things like that.

The good news is we can all get tested for these. We can find out if we have these exposures. We can also find out if we have inflammatory responses to these exposures.

And we can get remediation for our homes and we can actually get, the detoxification over time. And certainly it takes some time. You got to remove these things slowly.

Your body has sequestered them in multiple organs. Yeah. Including bones. One of the common things we see is, as people get to a point of starting to just the beginning of what will ultimately become osteoporosis.

Potentially, they rerelease these toxins back into the bloodstream. And so now this is why we believe this is why it's so common to see people in their early 50s, right around the time of menopause, that they are now, getting a diagnosis of Alzheimer's disease.

Wow. And that thought about, that issue. So, I may have sought it out that I had a mold exposure. Yeah. by seeing a functional medicine, practitioner who put me through my detox protocol, I remediated my living environment.

Great. I'm doing well. I now am in menopause, so I'm in my, 50s. 60s? I'm beginning to get osteopenia. I've not done a great job of protecting my bones.

My bones are getting more and more, thinned. And during that osteoporosis process. Osteopenia. You're telling me that, that's what it. And I suppose I could be releasing the lead that get stored in my bones, the mercury that had gotten stored in my bones so I could be, so worth probably storing, not just bio toxins, but the, heavy metals.

would you say the organic compounds, the glyphosate tailings, benzene? Would that be getting stored in bone as well? I don't know, obviously, those are much more hydrophobic.

So my my guess is they would be in more hydrophobic environments like liver and things like that. But it's a good point. And actually, you know, Doctor Joe Pisano was pointing this out.

This you go through this about seven year Osteo classic burst where you're now rereleasing these things. so you really have to be concerned at that time of life.

and I would I would love to ask Joe whether these things are in the bone as well. Again, my guess is these much more hydrophobic compounds are going to be in more lipid rich environment, as opposed to the bone.

So probably the big thing, I'm thinking would be the, heavy metals, in, those bio toxins, in the bones.

Toxins, Mold, and Environmental Exposures

and so again, to the listener, perhaps, the suggests that we could give them as, if you're menopausal and you're going through a cognitive decline, be sure to ask for a toxin evaluation that includes both, mold exposures and those heavy metal exposures.

Yeah. And, Terry, if I could ask you a question. So one of the things years and years ago, it was pointed out, that one of the Ms.. Experts came to to visit the Buck Institute and pointed out that, when you prevent those early days of to say things like that, when you prevent this migration, you're preventing the production of the Ms., plaques and you're preventing, the, the relapsing and remitting attacks that you're really not changing the continued slow march into degenerative aspects.

What's your take on that? How is that shaking out? what? It looks like we've been able to do is delay the conversion to secondary progressive M. S. by five years.

Okay. And so at five years, really wonderful. And I and I do tell my patients it's really important to stop the enhancing lesions to stop the relapses.

because we want that, that, we don't want you to have the enhanced lesions, or the relapses, but unfortunately, you know, my neurology colleagues were not talking about the degeneration.

and that was because they didn't have any drugs they missed. They weren't doing what you and I knew to do is like, this is all about the modifiable lifestyle factors.

This is about diet, exercise, stress and sleep. And then we can also look for the toxic exposures and, deal with those so that more comprehensive, integrative, approach that you take to Alzheimer's and I take to Ms.

can slow that degenerative, decline. Fantastic. sorry. Go ahead. You know, the good news in the Ms. world. You know, when I first started talking about all this stuff in 2009, it was it was, you know, very threatening message.

I got banned and people really attacked me now. But we can do your research, and kept publishing, you know, the this surprisingly positive, results that we would find.

And now the idea that diet matters, there's a lot more agreement that diet matters, that we can have a debate as to

which is the best diet. But there are many diets that can consider, that sleep matters.

Stress management matters. and, we've been saying for a long time that exercise is really very important. So when I go to the Ms. meetings, there is more discussion that that that. Yep.

People are still super excited about the drugs. And I think it's fine to be excited about the drugs, but they're now beginning to say it's just as important that we talk to people about, their wellness behaviors.

Are you seeing any of that in the, Alzheimer's world? Yeah. there's so it's interesting, there's a little bit in terms of people talking about, well, you know, yes, we should be doing more exercise and some, some basic things.

But there there's really been a schism. there's been most of the PR, most of the articles written, most of the op eds written are all about get an anti amyloid antibody.

Now this is despite the fact that they don't make you better. That's been well proven. They slow the decline by 27%. So at least a little bit of slowing a decline that that's the effect they cause micro hemorrhages in the brain.

Unfortunately they cause brain swelling. They've caused a few deaths. they are massively expensive. By the time you pay for all the infusions of the drug and the follow ups, it's about $50,000 per year.

Every year. they mostly haven't been covered by Medicare. Even the ones that are are covered are partially covered. So you still have a lot of out-of-pocket.

So it's really, as they said, we've been looking for the drug. This is not it. this is not a great situation, but there is a huge pharma effort because it's a big, you know, business side.

It's quite a big win for the pharmaceutical companies. So we really haven't gotten to the point where people have said, look, these other things need to go together.

We have stressed over the years the long term is going to be targeted drugs with a an overall precision medicine protocol. I look to the for the day and that's kind of where we're headed.

but so far it's really been, you know, a very much of a, of a polarizing, situation in neurology. Well, yeah, I think the Ms. world may be a little ahead of you guys.

Then there's still a huge, excitement for the drugs and the Miss World. About 20 different drugs that, have a varying level of effectiveness for turning off the inflammatory component.

Right. but none of them do anything for the degenerative component. Zero. It's nothing. But that was the question. Okay. and so, now I'm going to go on to the next question because there's the point.

Lots of money into trying to find, compounds that will improve re myelination. and, if you follow any of that research, do. You know, I really haven't, but we have the same situation in, in the case of Alzheimer's, not re myelination.

It's really, synaptic, rebuilding and synaptic reestablishment. And it's about, you know, intranasal trophic factors and stem cells and things like that.

but I'd love to hear more about what's going on with re myelination. So in the mouse world, we have a bunch of compounds that are super exciting, for, mice and rats and, very abysmal, for humans.

and I expect that will probably be the same for the synaptic, compounds that we develop for Alzheimer's that will define compounds that work really well for rodents.

and, mice and rats, but don't do work very well for people.

MS Progression, Remyelination, and Mitochondrial Health

And your, part of that is, when we get around to doing human studies, we eat what we want, go to bed when we want to exercise or not. have a fight with our spouse or not.

and so there's all these environmental factors that you can control in your mouse study that we cannot control in human studies. and so, those compounds may someday be helpful, and they may someday find compounds that are helpful in the Alzheimer's world and in the mouse world.

and if they ever get them, and you and I get to use them with our precision medicine, we'll have far more success than all of our neurology colleagues who would use just the drug and not address all the environmental factors.

Yeah, I think that this is what, to me, this is the big change that medicine must undertake. when I was trained in medicine, and I'm sure it was similar when you trained in medicine as well.

it was about figure out what the disease is and then write a prescription that's appropriate for that disease. That was it. And the problem was, is, you know, as you've pointed it out, with functional medicine, you're actually looking at what's causing the problem, with where the drugs, you're, you're trying to go around the problem and you're trying essentially to trick nature.

And so I think we need to be much better at understanding when we perturb any system. The system is out of whack, which is why the person has a disease.

We need to bring that system back to normalcy, not try to trick the system into, you know, messing up and into changing. So, you know, like, just do as much as you want, and, you know, trigger your Ms., but you're going to take over because we're not going to allow you to have the B cells to respond to it.

Okay. Well, that's that works, but it doesn't necessarily work for the long run. And we had the exact same thing in Alzheimer's. You can give, for example, something like Aricept to Napa XL, which now will boost up your you're still calling a little bit.

Well, okay. But you again, you're ignoring what's actually causing the problem. And your body is now going to respond by making more of the enzyme that breaks down the acetyl choline, because this has been a response to something going on in your brain.

So again, you're trying to trick Mother Nature. And in the long run, that doesn't work very well. Yeah. I mean, this is where, again, anyone who's listening when you come to see Dale, work with one of his trained practitioners or your working with me or one of my trained practitioners, you'll come in.

You have a very detailed history, to really understand all these environmental factors. and then you'll probably have a fairly detailed, set of diagnostics that you would go through, will be guided, in part based on this detailed history.

And then we can create a plan for how we're going to address. What were your unique environmental factors, contributing factors that led to your cognitive decline due to Alzheimer's or or your M. S.

symptoms and cognitive decline? so we can sort out what's going on or or, again, any of the other neuro immune problems that are attacking your cranial nerves, your brain, your spinal cord, or your peripheral nerves.

You know. So I wanted to ask you so one of the other points that's been made is that when you have a degenerative component in MS. this can be a glutamatergic phenomenon, a cytotoxic phenomenon, with Alzheimer's, we see some of that as well, where there is a glutamatergic component, there's a cytotoxic component to the degenerative problem.

But again a lot of this is about you've literally changed your signaling. You're switching your resources from connection to to protection. You're making these things that are responding to insults.

What's your sense about the underlying important biochemistry that is driving the degenerative component as opposed to the inflammatory component is this cytokines is what is it that's driving this?

yeah, I think it comes down to mitochondrial, inefficiency that the brain doesn't have enough energy, to maintain, the myelin doesn't have enough energy to maintain the synapses.

And so we see this progressive, and more rapid loss, brain volume of spinal cords, and, so, you know, that was my my first really big idea was it's the mitochondria that are driving disability.

and so I first focused on supplements from about a hundred. That helped a little bit. And I was super grateful. And then when I got into a more, a much deeper understanding, with functional medicine, I was still focused on supplements.

And then I, you know, I see a little bit more, how to look into the issues more, comprehensively, and address more at the root cause, but might have kind of this function is certainly a big, big driver.

It's not the only driver. Yeah. And what is damaging those mitochondria? I mean, certainly I hear all the time and people with MS. will say I just have no energy.

which makes no sense. But what's so. What's driving that? Yeah. Poisons. You know, the, heavy metals, the bio toxins, the, the, chronic infections, that, and then, terrible nutrition.

if you don't have, the, appropriate minerals, nutrient minerals, magnesium lipoic acid, creatine, carnitine, that can be a huge problem. and so resuscitating your mitochondria, again, is a multi-faceted approach.

And, you know, when I investigate, what are the root causes? we often see mitochondrial dysfunction, as part of the picture. And as we address all of those factors, mitochondrial dysfunction, dysfunction diminishes, oxidative stress diminishes.

but in the usually it's not just one thing. And I'm sure this is what you see. It's usually rarely is it one thing. It's usually multiple hits to that person's biochemical pathways that we have to address.

And do ketones help in terms of their energy support? It does help lot. You know, particularly for people who have cognitive decline. It's very helpful if people are willing to do a quadratic diet, it can be very helpful.

Precision Medicine, Ketones, and Where to Learn More

There are so many folks who have, metabolic dysfunction, insulin resistance, type 2 diabetes, obesity, cognitive decline, all of those people, generally do much better on a diet.

Now, for some, ketogenic diet is too hard. And then we're going to, do a paleo diet and, perhaps a lower carb version of the paleo diet. if if they could do the, the ketogenic diet and they have metabolic dysfunction, it can be very, very, very, very effective.

Fantastic. All right. Thank you. Well, Dale, this, of course, is amazing. You and I could chat for hours, you know, and I look forward to the next time we'll be able to be at the same conference together.

And, can, in fact, sit in chat for a couple hours. I look forward to it as well. Thank you. Terry, always great to talk to you. Well hang on. So where do people find you so they could learn more about you and your approach for cognitive decline?

Yeah, absolutely. you can either look at DrBredesen.com, that you can read one of the books, The End of Alzheimer’s. or The End of Alzheimer’s Program, or The First Survivors of Alzheimer’s, three different books, that you can look at there.

We actually have another one that's going to come out next year, which is called the Ageless Brain. really looking at how to make our brains do better in terms of their overall aging.

you can also go to, Facebook, Dr. Dale Bredesen, or X, formerly Twitter, any of those things, Instagram, all of those, to see what we're doing. that is marvelous.

Thank you so much, Dale. Thank you again, Terry.