P5P - Podcast - Dr. Dale Bredesen - 11.07.17.mp3

This is David Eigen with the next edition of P5 Protocols. I feel blessed to have Dr. Dale Bredesen on this podcast. He is the first doctor / researcher I have met who has operationalized a program for the masses, delineated in his recent book, The End of Alzheimer and is building a business around it to ensure that the world has access. He has extensive data to back up his findings, so I truly believe it is worth your time. In the transcript, because the data is so dense, I have changed my format a bit to make it more readable – and likely still failed! ***Bold and Italics are background***; underline is treatment and **bold and underline is strictly for emphasis**.

Dr. Bredesen's approach is programmatic. He looks at every possible thing he can find – culled from 30 years of research and doesn't simply look to help you get every edge you can get, but actually look to eliminate the things that are getting in your way. It is very similar to the approach of Sidney Baker and Nancy O'Hara who also seek to remove what is bad and add back what is good. Dr. Bredesen has automated what I am trying to do with P5 Protocols, which is exactly this: look at and analyze your condition as something that is multifactorial and correct for all. Perfect health is the goal. To me, it's the only goal, because if not, you will fall well short of it and then, with age, look out below. And with that, here is my interview with Dr. Dale Bredesen.

[00:01:41] [DE] I'm here with Dr. Dale Bredesen who has a new book out called "The End of Alzheimer's" and who practices what I'm a big believer in, which is a protocol or what I would call an adaptable protocol that he has developed over many years, [and] has now over a thousand patients in it and hundreds of patients with longer term data, that shows that he has been able to stop, prevent and in many cases reverse cognitive decline from basic dementia all the way through to Alzheimer's. And the book is titled "The End of Alzheimer's". And with that, welcome Dr. Bredesen.

[00:02:22] [DB] Thank you very much David.

[00:02:24] [DE] I really appreciate your having taken the time to be here. And as I said to you off line before, in the original intro I wanted to bring you on or talk about you in my intro because you're the first person I've seen has built a comprehensive protocol and put out there in your book the tests and the way to approach and treat so that a doctor could pick it up and practice etc... so I'd love you to talk first about your approach but you know how you evolved and how you got here going all the way back to your traditional training.

[00:03:03] [DB] Sure. So this came straight from the test tube. ***I had very classical training. I graduated from Cal Tech, I worked at MIT with Professor Mark Wrighton and was interested in basic chemistry and then approaches to neurochemistry. And then I was a postdoctoral fellow in the lab of Nobel laureate Stanley Prusiner and we've been interested for years and my lab has been up for now almost 30 years in what actually drives neuro degeneration. Why is it that we get things like Alzheimer's and Parkinson's and Lou Gehrig's disease, where do they come from, why is it that people haven't understood them better. And you could argue that this has been the area of greatest biomedical failure. As they say everybody knows a cancer survivor, but no one knows an Alzheimer's survivor and of course the book describes the first. And so what we started with was the idea that we would get to [you know] a single drug, a single target. And what we found when we looked at the basic chemistry of this illness is that Alzheimer's disease doesn't come from one thing and in fact the surprise has been that what we call Alzheimer's disease is actually a protective***

response to three fundamentally different processes. Number one chronic inflammation, whether it's from various pathogens like Lyme like but really up from Lyme Disease or like herpes simplex type 1 o from your mouth so forth and so on. Number 2, a trophic withdrawal, so you suddenly withdraw things like nerve growth factor or brain derive neurotrophic factor or *[00:03:08]* ***estradiol. You go on and on and on. Vitamin D is one of the things.***

[00:04:53] ***All these things if you withdraw them especially rapidly, the response is essentially a downsizing of the neural network, which involves the pathology that we call Alzheimer's disease. And then the third thing, it turns out that, the amyloid that you make in Alzheimer's is also a very good binder of many different toxins. So for example, if you want a brain to protect itself by making amyloid, then throw in some mercury. That's a good way to do it. Copper is another one, it binds copper, it binds other metals like iron and Professor Ashley Bush has shown this over many years that this is a very good binder. So those are the three major areas where your brain makes this response, and so unfortunately, people say well it's Alzheimer's disease and we don't know what causes it. But the reality is that what we look at is Alzheimer is a response to insults that are upstream, that can be measured, quantitated identified and addressed.***

[00:06:02] [DE] Amazing. And you started as a scientist and became a doctor so you're not just on the back of it, you're not just saying you're doing research putting it out there, you are on the front lines.

[00:06:15] [DB] Absolutely.

[00:06:18] [DE] So how many people have been through and how many people are current, I believe you said there's over a thousand now on the protocol?

[00:06:24] [DB] So **there are now over 1000, and that actually includes many people from a social networking Web site called Abel.inf** that was started several years ago by a woman named Julie. And these are people who are at risk for Alzheimer's or may already have the beginnings and who know that they have the most important and most common genetic risk factor which is Apoe4. And unfortunately, ***about 75 million Americans have a single copy*** of that .... And about ***seven million Americans have two copies, are homozygous for what we call Apoe4. And so these people are at risk. In other words if you have zero copies you're lifetime risk for developing Alzheimer's disease is not zero but it's about 9 percent. If you have a single copy it is about 30 percent through your lifetime. And if you have two copies it is anywhere from 50 to 90 percent***. In other words you are more likely to develop Alzheimer's than to escape from it during your lifetime. And so these people were looking for ways to prevent and reverse cognitive decline. In almost all of those people - they have about ***2000 people on the Web site - are on some version of the protocol. And then of course we've had hundreds of people also come through and we've trained now over 600 practitioners from seven different countries and all over the US***.

[00:07:52] And there's actually ***another training session coming up through the Institute for functional medicine, December 2nd and 3rd, in Miami***.

[00:08:02] [DE] I was going to, I was going to put that up and mention that for you later which I'll put up on the website as well along with the show notes. ***It's sold out in person by the way. So it's just a webcast at this point. As of any and I guess it's so popular that the***

even the wait list is full, so people are listening.

[00:08:27] [DB] And for you know, for the first time, so many people, both patients and physicians have written. ***We've had a close to 10000 emails now written and said you know we didn't know that there was something that could be done for this. So many people have written amazing stories about what's actually happened to them and improvements that their doctor told them had not been possible***. And so again you know we didn't come at this with a preconceived notion where we if anything we thought that we would get a drug that would have an impact on this illness and what we found is that in fact it is more complicated than that. In fact this is a complex chronic illness and you know you think about it. It even took three drugs to have an impact on HIV which is a simple little retrovirus and Alzheimer's is much more complicated. These complex chronic illnesses are not like the infectious illnesses that we were dying from a hundred years ago where you could target with a single antibiotic. So we've identified it, we initially identified 36 different potential contributors and of course each person has a different profile. So the approach is a problematic and personalized one. But you have to determine you know who's got which pathogen. Do you have a decrease in specific trophic support. One of the most common contributors, as you know, is insulin resistance and we call this type 1.5 because it has both the inflammatory component from things like the advanced glycation and products and it has the atrophic component, which is the type 2.

[00:09:02] And that's from the change in signaling that you get when you expose the neurons to insulin so you now actually have decreased your response to insulin, which is such an important trophic factor for your neurons. And then you need to know, have you been exposed to specific toxins and as you know most of us don't realize that we have these various exposures until we actually look for them. So what happens you go into your doctor and your doctor says oh you have Alzheimer's disease and you say well what caused that. And the doctor says we don't know, it's Alzheimer's disease and this is a little bit like taking your car in to a mechanic and saying you know what's wrong it's not working well. And they say oh this were recognized this very well this is called "car not working syndrome". And you say, well wait a minute, I mean aren't you going to check the gas, the oil, the transmission fluid, the brake pads. No, no, we've seen this many times. We don't check these things because they're not reimbursed. So we're just going to tell you it's "car not working syndrome" if your car's going to die. And this is the approach that we're actually being told by physicians with respect to Alzheimer's disease. You know Alzheimer's disease, the name means nothing. It doesn't tell you what's actually causing it. So you need to look, you need to look at these larger datasets. And we often say that you know there's a huge complexity gap. Think about this. We've developed computer based flying of airplanes, driving up driverless cars. Of course computer algorithms help with improved advertising.

[00:11:32] Why are we not doing the same sort of thing for improving the diagnostics and therapeutics for complex chronic illnesses. That is the basis of 21st century and unfortunately most places you go today are practicing 20th century medicine, where you're looking for a monotherapy for these complex chronic illnesses and as you know it just doesn't work very well.

[00:11:47] [DE] What I always say in pretty much every podcast, is as a patient myself, I am always looking for every edge I can get. And as someone who's lost my father, my sister now and a lot of other people I care about, you know when you're a patient you want every edge you can get. And that's what I think you're looking at. And it's you know sometimes I say if you don't change certain things like nutrition or stress, you know you can take all the drugs you want but it's basically peeing in the wind or Sisyphean maybe is a better way of

putting it. What I'd love to do is, ***maybe I just work better with stories, but is go through two patient scenarios. The first one being less of a patient but a potential patient and the second one being an actual patient and use that as the framework for understanding how your book flows, how you practice medicine and what you're teaching practitioners to then go out and practice on their own***.

[00:12:59] [DB] Absolutely.

[00:13:01] [DE] So let's start with someone on the threshold of a cognoscopy, you know, a 45 year old male comes to you, is just generally concerned, family history and is concerned and so could you walk us through, you know maybe two versions of that. Right so the intro and then one is you get kind of a slightly negative one and the other one is you know generally fine, how would you follow up with that person. So start with kind of the testing you do and then what you would and he's generally fine what you would do. And then he can go into no he's not generally fine, there are some symptoms, how you would approach that.

[00:13:44] [DB] That's a good point. So you know what you're describing is the future as I said in the book. Everybody who is 45 years or older should consider having a prognoscopy and I coined that term because it's easy to remember that when you turn 50 you get a colonoscopy. And so you know when you turn 45 or if you're over 45 you should certainly consider getting your brain checked out.

[00:14:10] And the good news is there are many tests now that can be done fairly easily. Simple blood tests to determine are you at risk. Are you already going down the pathway and again this goes on for many years before there's any diagnosis. So you want to look at number one you want to look: do I have anything that is causing the chronic inflammation that would make my brain respond with an amyloid genetic response. So for example look at your HS-CRP. Is it over 0.9. You want to know. Do you and you may want to know about your interleukin 6 or TNF Alpha but certainly you absolutely want to know about your HS-CRP, also helpful to know your albumin to globulin ratio, so do you have an ongoing inflammation. If so is it because you have a chronic pathogen. Do you have various viruses? Do you have spirokete's? Do you have fungi? Your body actually fights these things for years and years and years and if that's what you're in the middle of doing, it's helpful to know so that you can improve your immune status and reduce the overall inflammation and there's actually as you know some resolving mediators that you can now take to help resolve for the first piece of this, and then we tend to think in the long run more of anti-inflammatories. But again take a step back. It's not just about is it an anti-inflammatory. It's knowing what's actually causing the inflammation that is getting, that is causing the problem. It's not just about suppressing the inflammation.

[00:15:44] And then secondly you would want to know what is my status with respect to insulin resistance. You want to have a fasting insulin done and you'd like to see your fasting insulin be 4.5 or lower. A lot of people are walking around with fasting insulins of 10, 15, 20. We've even seen up into the 30s and 40s. And what this means is that although you might feel OK, your body is working very, very hard to keep your glucose under control. You have insulin resistance. And before it contributes to cognitive decline, you want to address that and restore your insulin sensitivity. And that's absolutely possible and something that's happening every day and again and **we go through all the ways to do this in the book** and others of course have described this sort of thing previously. People who are working on things other than cognitive decline. So very important to know that. Helpful also to compliment that with hemoglobin A1C of course and fasting blood sugar. All of those are helpful. And as you probably know neural exosomes [testing] are just appearing and they

should actually be available this year. This is going to change everything because this gives you a way to look at your ongoing brain chemistry with a blood sample. ***You have about 1.2 billion of these tiny exosomes. They are about 1 seventieth the diameter of one red blood cell. So tiny little fragments. And there is actually a bidirectional transfer. They come out of your brain, they go back into your brain and you have them from multiple organs. But it turns out about 10 percent of these come from your brain***.

[00:17:32] And so you ***can actually isolate those away and then - and Professor Ed Goetzel at UCSF and the NIH has done this successfully*** - and you can look at whether there is insulin resistance in your brain, whether there is enough trophic support in your brain, whether you have increased amyloid in your brain. Same for tau and things like that. So this will be very, very helpful. And again should be available this year. And then you want to know what is my status with respect to trophic support for my brain and especially have I had any rapid changes recently. So that's where you want to understand what is your vitamin D level, what is your estradiol, what is your pregnenolone, testosterone, progesterone. All of these things are free T-3, 3T-4 reverse T-3, these things all contribute to the support of your neural network.

[00:18:31] And if you look when we were looking at the molecular biology underlying Alzheimer's disease over the years the interesting surprise to us was that amyloid precursor protein, the APP and it all comes back to the basic science here. This actually serves a little bit like a CFO. So it actually looks at all the different things coming in and all the different things going out. And if you are in the red, if you do not have the support for your neural network, it participates in the downsizing of the network. So you want to know that you have enough support. And again many people walking around with suboptimal thyroid function, suboptimal vitamin D levels, suboptimal testosterone function, suboptimal estradiol, progesterone, just go right down the list there. And again supporting these, optimizing these and as you know from your background with functional medicine physicians, in the end some of the other interviews that you've done, you know very well it's **not good enough to be at the low end of "normal"**. ***What we call within normal limits is really misleading, simply as two standard deviations from an average that you've taken from various patients and is in no way related to what is optimal. So that gives you then the idea of you know, I am suboptimal in things that could contribute to giving me type 2 cognitive decline and ultimately Alzheimer's***. And then you want to know what is my exposure to toxins. And there are many of us who are exposed as you know, just living in our society, we're exposed to hundreds and hundreds of toxins.

[00:20:13] I have to say that ***when we started this, you know as a scientist, I was very skeptical that toxins would have anything to do with Alzheimer's disease. I thought we were going to identify you know one specific cause for everybody. And that turned out to be completely wrong. There are multiple contributors, dozens and dozens of them and you need to look at them. So if you have a high copper to zinc ratio, then in fact your amyloid binds up that copper. So it's a protectant. If you have high exposure to mercury, whether it's inorganic mercury or organic mercury, you want to know that. And again until you start having some cognitive decline, you won't know that you are being exposed to this so it's good to find out ahead of time. Then, what about mycotoxins. One of the big changes has been that we that there are mycotoxins and of course Dr. Ritchie Shoemaker has spent much of his career evaluating these mycotoxins and looking at mycotoxin related illness, what he has dubbed chronic inflammatory response syndrome or servers, and I have been very surprised to see how many people in fact have exposure to these biotoxins. So we want to look at all these things. Currently, we have a computer based algorithm where we look at 150***

different things and generally what we call recode report, for reversals cognitive decline that tells you what subtypes am I at risk for. Because many people will have a little bit of type 1 and some of type 2 for example, they're often not pure and then it will also generate an initial program and of course the final decisions are up to you and your physician.

[00:21:57] But this will generate that initial one place to start the discussion. So as you said let's take two different examples. First example, everything's perfect. First of all, that's very rare. Most people we find have, if they're asymptomatic, between three and five suboptimal contributors and if they are symptomatic between 10 and 25 different suboptimal numbers so, we want to know where you stand. If everything really is perfect, then you want to check again in a year or two. But of course if you get symptoms, you want to check as early as possible in that time. So we usually then say okay you're doing the right thing. Check back in a year or two that's fine. Let's take this situation where there are several abnormalities this is the much more common one. So for example we often find that people have insulin resistance so someone comes back with a fasting insulin of 10. They come back with a homocysteine of 12 for example. Again, you wouldn't know this walking around, but these increased, for example, the increased homocysteine is associated with a more rapid brain atrophy as you age. So we want to bring these back into being in an optimal range from the sub optimal range and there are straightforward programs which is why this overall is a personalized programmatic approach, **21st century medicine is not mono therapeutic it is programmatic**. [**DE Comment**: *This is the impetus for starting P5 Protocols. In fact, we looked at P5 Programs, P5 Frameworks and other similar names but felt Protocols sounded best and in fact if you throw in the word Adaptable (Protocols) and then it works for customization. For our investment firm, in a few years, we expect to be the leading investment fund in this field*.] And as you know the fundamental change is from the diagnosis which is "what is it", doesn't tell you why, to why. **We're going from the what of the 20th century to the why of the 21st century. We want to know what contributes**. **So in this scenario when there are some abnormalities, then we set up a program that is optimal for that person. And then you want to check back in six months, get rechecked because as the metabolism goes, so goes the cognition**.

[00:23:59] [DE] Wow. All right. So, if someone is asymptomatic, is going to get retested you're saying every year, even if you're 45 and everything's perfect?

[00:24:03] [DB] If everything's perfect, you want to go one to two years. So yeah there's no problem with waiting a couple of years. Again, as long as you don't have further symptoms. You want to look at that. If you have abnormalities, you want to fix those and get rechecked in about six months.

[00:24:31] [DE] And how does someone go about getting all of the relevant tests without a lot of brain damage. Or you know trying to find them all and put it all together.

[00:24:41] [DB] It's actually simple, you don't have to suffer any brain damage at all. As I say, ***we've trained now over 600 practitioners, by the end of next year we will have trained over 2,000 practitioners from all around the world and all over the US. And so you can simply you can go on the website, DrBredesen.com and that will link you up with a person who's actually doing this and who's been trained***. [This is still done by email] **You can also of course come to the training if you're interested in doing that. We're also now starting, we just actually, a week ago, started a town hall meetings for people to ask questions and to make sure that people are kind of up to date** on you know what are the things what are the optimal things to check to be able to have the optimal

outcome to prevent cognitive decline, or if you've already started down and of course, to reverse the cognitive decline.

[00:25:35] [DE] And from what I've read and listened to your other interviews. Someone who is 45, just before we move on to the other person who is a more serious case, someone who is 45, who may even have three to five things, you're telling him you know start watching these things and then come back and get re-tested. And, you know my dad used to joke, without using the actual curse words, because he was not a guy that cursed a lot or anything, but he started joking, this got to be 25 years ago. He said you know for years my problem was CRS, which is "Can't Remember Shit". And then he said, I think I've now moved on to CRAFT disease which is "Can't Remember An F'ing thing". It was a joke, it was like you know today, it's concussion here, concussion there in sports. Back then it was you got your bell wrong. And you know because no one had any answers or no know knew any better. But now here we are with something that people can now actively pursue to make sure they never deal with it. It's a total change in mindset.

[00:26:46] [DB] Absolutely.

[00:26:47] [DE] But I'm all in favor personally and ***I'm going to do it. I'm going to go through the whole thing sometime in the next couple of weeks***, I need to find someone local. But I'm going to so I'll put those results up when I get them. [Maybe!] And then so I'd love to go on to, so the first was a 45 year old may, so to be democratic, we will go to a 60 year old female and showing material signs of cognitive decline. Not yet, you know you tell me what's a better example that flexes your program but either further along, less further along. But definitely showing material signs of cognitive decline and that person comes to you as their doctor. What do you, what would you do if they were your peers.

[00:27:38] [DB] Very good point and you know just to finish up the previous discussion one of the exciting things that I've been hearing from the various physicians is that they're now seeing because of what we've published and because of the ability now to reverse that decline we're seeing more people come in and say you know I just want to be on prevention. My, you know, my mother or father or both died with dementia. And I'd like to get on prevention now. And you know this is the way of the future, this is the way that we will reduce the global burden of dementia, which is a huge trillion dollar global problem. Now you brought up the ***60 something year old woman*** ***and this is a very, very common presentation. And interestingly as I mentioned in the book when the symptoms have started in the 40s or 50s and as you said if it's 60 and it's been going for a few years, this person would certainly fall into that group. It is more likely, much more likely, to be Type 3 toxin related. We see this a lot and it looks as if we don't know the final answer to this but the current suggestion is, that when you are young, you are actually storing these toxins, be they in mercury or organic toxins or bio toxins. You are storing these, you are sequestering these in your bones so as you hit menopause, you begin now to have more osteoclastic related to osteoblastic activity. In other words, more of the loss of bone and that's of course what ultimately can lead to osteoporosis***.

[00:29:01] But long before you have osteoporosis you are still releasing things from your bones and so that gives you the exposure and you now proceed down this cognitive decline which turns out to be mostly type 3. So, ***on the other hand, the people who are in their late 60s when they start are more likely to be the inflammatory non type fuses than the type 1. And on the other hand, the ones that are starting in their 70s and 80s are more likely to be the atrophic, the type 2. Now again it's not a 100 / 0***, but that's a general

rule so if the person comes in and they are 60 and they've already had some problems over the last few years, and I'll give you an example of one person. Actually, this was a person that's been on a **CNN interview** and has done extremely well. ***This is a person who did start in her 50s and came in her 60s, and clearly had difficulty with memory, clearly had difficulty also with so-called non-amnestic things, so so-called cortical symptoms, difficulty with calculation sometimes, difficulty with organization, so-called executive dysfunction, one of the most common non-amnestic problems, difficulty with visual perception some of these people have. Some of these people have difficulty with word finding so an aphasia. And there are well-recognized presentations for Alzheimer's disease. One so-called primary progressive aphasia, someone presenting with aphasia, posterial cortical atrophy, presenting with problems, with things like perceptual visual perception and things like that. So we would want to look at this particular person again and look at all the different potential contributors***.

[00:31:00] The most important thing about all this is **not the program, it's the evaluation, it's the determining for the first time why people are actually getting Alzheimer's. And when we do, it's just shocking to see how much of this is out there. How much exposure to these bio toxins, how much exposure to chronic microbes and chronic pathogens, how much insulin resistance is out there, how much chronic inflammatory response is out there**. And people you know end up going on, for you're, the woman that you mentioned here, Let's talk about Sally for a minute. She came in and had MOCA of 24. ***She had an amyloid scan and her brain showing that she already had amyloid in her brain, so she actually went on one of the drug trials for Alzheimer's and interestingly each time she would get the injection she would get much worse. And what that turned out to be do to, and we've seen this a number of times now, is because, again, you are making this as she was as a response to being exposed to, in this case biotoxins, and so you when you reduce the amyloid, you're reducing your protection. And she would get much worse, so after three injections, she said this is making me much worse not better. I've got to get off this. She did get off it. And then over the next year and a half, she went on our protocol. She increased her score from 24 to 30. 30 is a perfect score*. She noticed huge improvements. Her husband noticed a huge improvement and she remains on the protocol, and one of the most important things about everything here, is that people who improve stay improved. As long as you stay on the protocol, you sustain the improvement. So you really do address the underlying pathophysiology. We've had a number of people who will go off and on and off and on when they go off they get worse. Typically within 10 days to two weeks, when they go back on they get better again. And so there is a there is a temporal link to improvement**.

Now **this particular woman turned out to have a very high C4a. And that is as you know, it tells you that you have an ongoing activation of your innate immune system. One of the simple ways to think about what Alzheimer's actually is, is to understand that the amyloid that you make is in addition to being a protective response, is part of the innate immune system**. So you're simply saying I have ***activated the innate immune system which as you know is the older part, evolutionarily older part of the immune system. This thing should then be deactivated when you deal with the pathogens appropriately. You've got your adaptive immune system which is the newer part, which then kills the various pathogens you dispose of them. You know you resolve the inflammation, you're back to where you should be***. The problem with **Alzheimer's is that you have a chronic activation of this innate immune system, just as you do with SIRS that Dr. Shoemaker has written so eloquently about, over the**

years. So, it is actually a cousin, it's related to what he has described. And so in this particular person that was shut down. Her C4a went from thousands, and it shouldn't be more than 23-80. She came down to below 1,000. **So a marked improvement in her biomarkers, along with a marked improvement in her cognition both on the memory side and on the non-amnestic side. She's able to plan things better. She's able to do word finding better. She's able to do calculations better. And again she scored perfectly on her follow up MOCA.**

[00:35:04] [DE] And so when she, so she comes, so I assume you get a lot of people that are kind of giving up, as opposed to being I'm sure it's tipping now. But historically people kind of giving up and looking for help. I'm guessing that it's tipping now towards as your name gets out and the book gets out and as you're you know as you're as things get out there about your work that you won't get the second or third shot at them. But you know so she comes to you, she's going to get the set of tests that you delineate in your book. And you know maybe I can get you to create some kind of PDF that people can use to make it a little easier. But they come to you and they're going to get this battery of tests that you mentioned, it looks her and you put it to through your software. And then you are going to come up with a first plan.

[00:36:06] [DB] First thing is it tells you what subtype. So she actually ended up having mostly type 3, with a small component of type 2 and a smaller component of type 1 and 1.5. So she was a mostly toxic that as we call type three or toxic or "vile Alzheimer disease" as opposed to inflammatory or hot or atrophic or cold. And you mentioned about a PDF. So a number of people have said, you know you need to get something out that is a simpler version of this, because the book goes into detail about you know how we developed this etc.. **So I am working on a follow up book that will basically be more of a manual, a handbook. However if you want something quick now, if you go to the end of Chapter 7 and the end of Chapter 8, we have some summaries of the evaluation and then the personalized approach**.

[00:37:01] [DE] Yep I'm staring at it right now. It's great. So **that's on page 167** and I'm not going to spend any more time with the other one. Here it is a **203**, anyway. So then she goes on a protocol for, in a case of someone who is showing decline, how often are they checking in?

[00:37:25] [DB] So if you are showing decline, what we typically see, is we tell people get on the program, it's going to take a few months to optimize the various things and then you know, don't expect results before three to six months after you've gotten on the program, and really done the majority of it, because it takes some time you know, get the nutritional piece correct, to get the exercise piece correct, to optimize the various so-called nutraceuticals that are actually being used, so the various things, you know you are literally changing the course of something that's been going on often for a decade or more by the time you're having these symptoms. So you know it takes a few months in and you want to check in, the first couple of times, you want to check in monthly and then you can, as you get better, you could make it quarterly and you can make it you know semiannually, you can go out to annually and then even a couple of years out, once things are going very, very well.

[00:38:22] [DE] And do you, so you look for 150 things and obviously that's more expensive. Once you're nailing these things or eliminating certain risk factors, does this set of tests start shrinking?

[00:38:35] [DB] Yeah and I should say ***when I say 150 things, some of these are historical things. You want to know critical things, like you know did you have difficulty with organizing. If you've had difficulty with non-amnestic pieces, that tips you toward looking harder or looking more deeply for whether there is toxic exposure because that is a common concomitant of the exposure. So not all of these are blood tests. And we're looking at more and more of these things to be able to be reimbursable and make this easier and easier and less and less expensive. And yes of course, you say once you know that absolutely, if your main problem turns out to be a couple of simple things, as an example we had a guy who who came through a few years ago - who by the way has done extremely well - and he actually had 23 different sub-optimally. But as you know as we started to optimize these things, you know, he could get a blood test for three or four or five things that were still suboptimal and he's really come into line and done extremely well***.

[00:39:44] [DE] So and you know, when I first saw you present back in February, you had videos of people as they went downhill or near the bottom and then as they improved. It is amazing to me. And seeing the patients marveling at their own improvement because they were given you know kind of a death sentence or very certainly won't even remember parts of their life, let alone the death sentence.

[00:40:19] [DB] Yeah that's absolutely true and then I describe some of these people in the book and actually there's in one of them, ***actually wrote a beautiful you know a couple of pages about what it feels like to be descending into dementia. This is the woman I called Eleanor in the book and she's doing by the way extremely well currently. And it's now been a year almost two years now on the protocol and you know she was very sharp about her own observations and she could see what she was losing and noticed that she, you know she couldn't do certain things, although interestingly she said you begin to lose insight. So she said she didn't realize how much she had lost until she got it back.***

[00:40:53] **And actually she went to a major university on the East Coast, both before and after, to document her own clear improvement on her testing scores that go along very well. A lot of people have said you know is this just all placebo effect and the documentation shows that this is not just a placebo effect. These are people who have documented improvements in things like their hippocampal volumes on MRI and on their own quantitative neuro-psych testing. So this is not just "in their mind". This is you know, this is truly anatomically and chemically in their brains**.

[00:41:35] [DE] There is some value either way, even to some of that. Because if people believe they relax, if they relax, they probably are breathing better and getting more oxygen in their parasympathetic, so, what are the main things that you would recommend that you talk about in your book. So, but, from sleep to stress reduction, the things that you think can have a profound impact, that are relatively easy, well relatively simple, won't say easy because doing anything on a regular basis isn't easy.

[00:42:19] [DB] Yeah you're right. Behavioral change is not easy. And so, and again I really **recommend not to treat blindly**. The ***whole key here is you want to know what is causing your cognitive decline or your risk for cognitive decline. So you want to address those things and yes if you have insulin resistance, which so many people do have, then you want to include a whole program to address that. You do want to make sure to optimize your exercise and you want to include both strength training and cardiovascular. You want to include stress reduction and as you know it's been***

surprising. Again **as a scientist, I never thought that meditation things like T.M. were going to have a major impact on health. But the data are irrefutable. You know it improves plasticity and improves blood pressure, it improves cardiovascular status, it improves cognition. You just can't deny the data that are coming out as you know, now even things appearing on improving telomere length. So you shouldn't leave that out. You know that is an important piece you want to have stress reduction and no question, sleep is one of the most common problems**. It's a real badge of honor. When I was an intern in residence in medicine and then neurology, you know it was a badge of honor. You know **I stayed up all night to take care of this or take care of that. And we stayed up all night. You know all the time. This is the worst thing you can do. It increases your amyloid burden, it decreases your ability to clear it. It decreases your ability of your brain to get rid of damaged proteins and things like that. It increases things like your norepinephrine. So you know that is one of the worst things you can do for yourself. And one of the best things you can do to improve cognition. And then of course the whole approach nutrition has turned out to be extremely important.**

[00:43:17] And in the book, you'll see the so-called keto-flex and **actually my wife Dr. Aida Lasheen Bredesen, did a tremendous amount of beautiful work along with Julie G. who started the Apoe4 website on developing the Ketoflex 12-3, what they called the nutritional guide**. This is a largely plant based but flexitarian way to drive yourself into mild ketosis which turns out to be best for cognition and many people as they drive themselves into mild ketosis and when I say mild ketosis, we're talking about 0.4 millimolar beta hydroxybutyrate up to about 4 millimolar and this turns out to be very good for cognition and people notice the difference when you are insulin resistant and you are not driving yourself into mild ketosis. You don't think as clearly, your cognition is not as good. So that's an important one and certainly **MCT oil as you know, many, many people have been using MCT oil, which again helps you to get into ketosis and helps to provide fuel**. [DE: I use MCT oil almost every day.]

[00:45:15] And **then of course healing a leaky gut**. So incredibly common for people to be walking around with leaky gut, not knowing it until they find out that they have a chronic inflammatory state. So this is again damaging and another thing to heal, so all of these things are critical pieces you know. Is your magnesium optimized? Magnesium,, absolutely critical for cognition. And in fact magnesium 3 and 8 have been used as a monotherapy with some modest but significant effects on cognition. So again looking at all these different variables and then optimizing them gives you the best outcome.

[00:45:54] [DE] I have to ask this one this leaky gut question. Other than taking out the gluten and probably a lot of the different lectins, what are the other things you can do, you know, just kind of a baseline, to help leaky gut.

[00:46:13] [DB] Oh there is actually a tremendous amount that can be done actually. So again first thing you want to know is do you have one and we, ***some people use a Cyrex array test to determine that. But then you want to know what is causing it. And for that you can Cyrex array 3 and 4 as an example. Do you have gluten sensitivity? As you know there are many things that contribute to leak gut, from alcohol, to sugar, to stress, to you know soft drinks, to you know to gluten sensitivity, to non gluten sensitivity things like other grains and things like that. So you first want to know what is causing you to leaky gut. And then of course you want to stay away from those things that in your case are contributing***. And then of course you want to heal it up and whether you use things like, slippery elm, or things like colostrum, or things like bone broth, and they each have their own positives and negatives. As you know one of the complaints

recently about bone broth is some of these you're leaching things like metals and things out of the bones. So you want to know that it's good bone broth. But there are different ways to do this and different people like to use different approaches. However you do it, you want to **get to a state of a healed gut and you want to get to a state of avoiding the things that were causing that leaking**. And then again, **resolving the ongoing systemic inflammation and then preventing future inflammation and as you know this is now being implicated in Parkinson's disease, in multiple sclerosis, in of course IBS and things like that in Alzheimer's disease and a host of other inflammatory conditions**. I think of these, and **I've talked to Terry Wahls about this sort of thing. You know M.S. and Alzheimer are essentially cousins. And in fact Dr. Alexei Kurakin who is a connectomics expert and has written papers with me on this area, pointed out to me a number of years ago you know, when you look at the connectomics of Alzheimer's, it's actually quite similar to the connectomics of multiple sclerosis**. ***I said no, these things can't be related, one's young, ones older. No, in fact they are related and so I think of these as "de-flammatory" diseases. They both have a degenerative component, which we think of more in Alzheimer's but it's also there in M.S. And then they both have an inflammatory component which of course we think of more in MS and less in Alzheimer. But they both have both components and you need to identify the causes and of course Dr. Wahls has had tremendous success with her approach to multiple sclerosis***.

[00:48:54] **So we think of these things as diseases that have both an inflammatory and a degenerative component**.

[00:49:03] [DE] And on the patients you get, in terms of, is there anyone where even far along where you can't stop the decline. Have you ever seen it fail, to stop the decline.

[00:49:28] [DB] So you brought up a really good point, which is at what point is this not helpful anymore and ***of course patients are told all the time it's hopeless there's nothing you can do. You know we know this disease is called Alzheimer's and you know you're going to die. And you know in fact that's turning out to be incorrect. There are, you know, many, many people now who are getting better. Having said that there are several, and I mentioned it in the book, there are several points to make here. The earlier the better. Preferably as you said earlier get on prevention. But if you don't get on prevention, as soon as you have symptoms, please do not wait. Get in there, get it checked out and get on a reversal program. The later the harder and the less common. Not every single person responds. The most common reason for non-response is not doing the program. And this is no different than what Dr. Dean Ornish described years ago with cardiovascular disease. If you don't do the program, you don't get better. The more of the program you know, you do for his program, the more cardiovascular disease gets better. We see the exact same thing. Having said that we have seen people. There is a woman who had a MOCA's score of zero and the average for all Alzheimer's patients is 16.2. So in fact, this person had very, very severe and late stage Alzheimer, couldn't dress herself, couldn't really speak other than an occasional yes and no, you could not interact with her husband much, had lost the ability to ride her bicycle, things like that, she just was really not able to do much of anything. She has come back beautifully. I have to say, I did not think we would see improvement in her and we recommended against going on a program but her husband said No I want to give this a try and they've done remarkably well. She dresses herself. She speaks, she speaks in full sentences, interacts with her husband very well. She dances with her husband again, she rides her bike again. However she hasn't come back to normal. So the farther along you are, the harder it is to get back***

and one of the things that we're interested in now from the research perspective how can we make it, how can we continue to look for, what it will be to take people who are late stage and make them better and better and better. We can improve them somewhat. But can we get them even farther back. Will things like stem cells be added. Will that be the critical piece to restore lost neurons, lost synapses and things like that. We don't know yet but clearly what we want to do is all these other therapeutics on the background, whether you're testing a new drug or what have you. You want to do it on the backbone of an optimized overall program.

[00:52:01] [DE] You actually just took my next question, which is, you know, where are the next areas of research that you're focused on which I guess is stem cells or what else. I know, I've talked to several stem cell scientists who say, you know, a lot of it is still years away. So, what do you see, I mean, when I think of a platform like what you are creating, which is how to approach a disease, all the things to look at. And then you still are a researcher and scientists and go to conferences, I assume you're still talking to other people who may have pieces to add, almost like an open software platform, an open API as we call it, that you can plug into. Other than stem cells, is there anything that you're particularly focused on because it sounds like it's not a lot of the drugs now?

[00:52:49] [DB] You know so there's a tremendous amount of. And so there's actually too much to be focused on. Yes, ***stem cells have the potential at least, to bring back some of the lost cells, to bring back some of the lost synapses and also as you know, they provide their own trophic support for other cells locally***.

[00:53:19] So they will be very important and yes some of the stuff is years away but as you know **there are already ongoing stem cell trials for Alzheimer's disease. So certainly some it is here already. The second thing we're focused on is how do we optimize the data sets, you know look looking at 150 different things in the long run is a tiny, tiny, tiny number. So you know whole genomes, metabolomes, lipidomes, proteomes, microbiomes, these are all things that need to be optimized. Having larger data sets, that is 21st century medicine that's the way things are going. Then of course, adaptiveness to Parkinson's to Lou Gehrig's. So we've already had the first few cases for Parkinson's, for Lewy body, for ALS. And the question is how do we adapt this approach to look at the critical variables for other neurodegenerative illnesses. And it will, programmatic be the best way to go after these other illnesses as well. And then as you said looking at how do you test drugs. Part of the problem, and I mentioned this in the book, as you know that when you're asking a drug to help someone with Alzheimer's you're asking that drug to do about 100 different things. You're asking it to optimize your chronic inflammatory state and to deal with toxins. You know a drug, a single drug is just not set up optimally to do that. So a drug, we always tell the patients, you know, they want to cover 36 holes in your roof covering one hole is not going to help you that much**.

[00:54:44] So the drug is a fantastic approach for one hole. So you want to then add the program for the other 35 or so holes so that you can get a better effect of the drug. **So my hope is that in the future, we will be testing all of these drugs on the backbones of the optimized programs. And again much more interaction with Silicon Valley so that we look at much larger datasets and make this, you know, in fact, again we take it for granted that we are using complex algorithms for things like flying our airplanes. Why are we not doing that every day and having an optimal expert programs to work with our practitioners. Clearly that's the way of the future**.

[00:55:34] [DE] This has been amazing. I actually could go on with about 50 other questions but in respect for your time and hoping to keep listeners what I'm going to do is put all the names you mentioned down in the notes, linked to your website which is www.Drbredesen.com. A link to your book on Amazon. And I should probably do Barnes and Noble though to be fair. And I'm going to put up links to some of your other YouTube or other presentations as well as podcasts you and several others, that I thought were very good, that got and do more of the fundamentals of the disease. And I just, you know, I chose to, what I'm doing right now which is referring to those instead of repeating them, since you already did that, and then you know you have as you mentioned in the middle, started a company so that you can really roll this out in a professional manner which I think is just fantastic. And you know, as I said to you, as I said to my own G.I., you know, I said your problem is that you acutely treat a chronic disease instead of chronically treating an acute disease and you can't take for granted the fact that just because the body is adaptable, that it's not acute. We call it, you know chronic because we somehow adapt, that doesn't mean it's not acute. And so you're actually one of the few people out there that's really attacking this and doing that. So I am thankful, I'm in awe, and hope what you're doing very soon becomes the norm as opposed to the exception.

[00:57:20] [DB] I look forward to the day when we're all practicing 21st century medicine and my great hope is that we will reduce the global burden of dementia. This is a major major problem as you know.

[00:57:33] [DE] I think you're going to be you know, certainly a big part of the foundation that has to happen. So and I think it started and the great thing about the Internet is that it can happen. It can start moving quickly at least to the major medical centers. And what I'm hoping this podcast is that this reaches both those doctors and those other people on the boards who can have influence or people who are strong willed patients, who can get their doctors to see, because a lot of doctors I think would love to adopt this, love to trust it and simply just don't have the time. It's no fault of their own and it's absurdly busy schedules and families and lives. And it doesn't take away from anyone else and that's why I think what's great about your book and everything you're doing out there to promote it is giving out this information, so there's not a big energy you know headwind where it just takes so much energy to try to learn it. You've simplified it in this book, I think is perfectly laid out, brilliantly laid out, to walk someone through step by step so I thank you. I look forward to being in touch and again to be of any other things that you want me to link to or and I'll forward you just before we post notes.

[00:58:49] [DB] That's fantastic, thank you so much. Thanks Dave for having me. I really appreciate it. Take care.

[00:58:53] [DE] Thank you.

Resources

Website
www.Drbredesen.com
Lecture: https://www.youtube.com/watch?v=6D5aA_-3Ip8
Mark Hyman: https://www.youtube.com/watch?v=IPDGG_EnxRc
Steven Gundry: https://www.youtube.com/watch?v=p6lkRXaQKwk
Dr. Mercola: https://www.youtube.com/watch?v=qrQyxWP-S2s

Dr. Perlmutter: https://www.youtube.com/watch?v=1VpYOwH3hS8
STEM Talk: https://www.youtube.com/watch?v=HS7VZydS8HI
Dr. Robert Lustig: https://www.youtube.com/watch?v=b0hlrQQN1dU
The Brain Warrior's Way: https://www.youtube.com/watch?v=b0hlrQQN1dU

Book
The End of Alzheimer's
https://www.amazon.com/End-Alzheimers-Program-Prevent-Cognitive/dp/0735216207
https://www.barnesandnoble.com/w/the-end-of-alzheimers-dale-bredesen/1124998286?ean=9780735216204#/

Mark Wrighton at Washington University
https://wustl.edu/about/leadership/chancellor/

Dr. Ritchie Shoemaker
https://wustl.edu/about/leadership/chancellor/

Stanley Prusiner
https://ind.ucsf.edu/ind/aboutus/faculty/prusiners

Other

The institute for Functional Medicine
https://www.ifm.org/

Apoe4
www.ApoE4.info