Introduction and why early evaluation matters

Welcome to this exciting episode of the Reverse Alzheimer's Summit. I'm so excited to have Dr. Dale Bredesen here to explain how he came up with this protocol and what he's learned specifically in the past year or so.

There are so many exciting advances in Alzheimer's research, and in fact, I have delayed this particular conversation into our last possible day because it feels like every week there's a new research study in the news or a new trial that is been completed.

Something going on that's advancing our understanding of Alzheimer's. Dr. Bateson, thank you so much for being here to have this conversation with me.

So great to talk to you, Heather. Congratulations on all the great work you're doing. Thank you. Well, you have paved the way. And so I want everyone to understand that I learned from Dr.

Bredeson in 2017 the Bredeson protocol, basically what he was describing in the end of Alzheimer's. So I went to a weekend course where I learned how to apply essentially functional medicine, naturopathy medicine to the really kind of scary and intimidating world of Alzheimer's for me as a provider.

And nearly immediately when I got back, I started seeing patients get better. And this I mean, this still moves me to tears when I see it. It is just so overwhelming how much we can reduce suffering and improve lives.

And so, Dr. Bateson, I want to dove into what can we do? I know you do. You always are so great about giving people takeaways. So let's just start with that.

Like, if there were one thing that you could do or suggest that someone do, if they're concerned about their cognitive function, what would it be? Well, the thing that I always suggest to people is a cognitive copy.

So so get evaluated and see what's happening is we're getting was you know, we started with this kind of very broad, we can't miss anything, but we're now getting a better and better look at what is actually going on in the brain of a patient as he or she is developing Alzheimer's.

And we know that that's going on for 20 years, typically before dementia occurs. So you've got a long run up there. And so getting evaluated and now there are better and better tests.

So you can look earlier and earlier and more specifically. And, you know, a lot of people have said, well, I wouldn't want that test because I wouldn't want to know.

Sticking your head in the sand is not the way to go. We now know that pretty much what Alzheimer's has gotten to be excuse me, has gotten to the point where it's really optional.

There is so much you can do and the earlier you know, the easier it is and the more complete you can really prevent this. So for the vast majority of us, we should never have to worry about Alzheimer's disease.

And, you know, your generation and the young people, your generation and younger, should not fear this disease because you can check it out. You can get evaluated. You know what to do.

You can get on an optimal protocol and everything's great. And these new blood tests are going to tell us early, as I said earlier and earlier, and give us so much of a better picture.

And now we're adding more and more that can actually be done to reverse the cognitive decline. And as you well know, pretty much 100% of people with MCI, that's second of four phases.

Subjective cognitive impairment, pretty much 100% of them will get better. And I always ask the physicians I talk to, have you ever seen a person who actually went on prevention, did the appropriate things, but still develop dementia?

I have not heard about a single patient yet, but when on appropriate prevention, when they had no symptoms and were scoring well and still developed dementia.

So we really can in those especially in those 2/1 times, have a tremendous impact.

New blood biomarkers for Alzheimer's

And I just got another on Instagram yesterday, another guy who said, you know, I'm four for and reverse my MCI and doing really, really great. It's just always wonderful to hear about that.

It's so fun. It's such a privilege to work with people. So these blood test that you're talking about, this is one of those news articles that I was referring to.

There are new blood tests even just of this past year that are now available to test to see if you have a propensity towards or a risk for Alzheimer's or maybe you're even already having those pathophysiological changes in your brain that are associated with Alzheimer's.

And you walk us through that and what they mean. Yeah, it's a great point. And, you know, to be fair, people have developed these based on the pathology of Alzheimer's.

So they really did focus on things that they thought were the causes, but has become very clear. These are not the causes of the disease, but these are mediators of the disease.

These are your body responding. So there's one that's quite early, but not very not very specific. So it's very sensitive, but not very specific. And that's GFP glial, fibril area, acidic protein.

And what that's telling you is your brain's astrocytes have have recognized that there's something wrong and that they are now responding and that is an early change.

But it doesn't tell you it could be has some head trauma, it could be some stress, it could be frontotemporal dementia, anything, basically. But at least tells you, is my brain under assault?

No, no. Excuse me. And then on the other hand, one that is much more specific is fast. Votel now the one that's that's commercially available today is fast volatile 181 but fast photo to 17.

That seems to be a bit more specific and also perhaps a bit more sensitive is coming out soon. So probably June, July, something like that, you'll be able to get hospital to 17, something like that.

And so that will tell you, yes, what's going on in your brain could lead down the road to Alzheimer's disease. And again, it's not the cause, but what is what fast photo is essentially telling you is how is like bullets if you're trying to make a structure stable, you've got all these bolts along the way and on the microtubules that are holding out your nerve writes you both them down with Tao.

Now when you want to pop that off because your body is saying, Oh, I've got to now move my resources into protection and I'm going to have a protective downsizing event.

So it's really synaptic elastic activity. What it does is it phosphorylated the Tao which pops it off the microtubules and allows them to collapse. So it's telling you your brain has ongoing collapse of neuritis.

So please get on treatment. Please get on something to now bring these back up again and move you back from a synaptic elastic state to a synaptic blast extent.

There's also neural filament light, which is telling you it's again, something different. Each of these is complementary. That one's telling you whether there is neuronal damage.

That one's not specific as well. It doesn't tell you that it's Alzheimer's. It goes up and ALS, for example. But you can at least look to see, do I have ongoing neuronal damage?

And then the last one is about a 42 to 40 ratio. And that what is really telling you? Am I in a state where I am pro-inflammatory, where my 42 is going down in the blood and the 40 so the ratio goes down as you have this pro-inflammatory state.

So these are all different bits of information that you can glean that will tell you if you are in this process. And of course, then you can actually look at what begins to what is actually causing it.

And you're looking at things like homocysteine and you're looking at leaky gut and you're looking at all the things that we always talk about that are so crucial.

Now we're able to see that your brain is actually responding to those things. Okay. So if I understand this correctly and of these are new to me too, right?

As a clinician. And so the way I use like an hs-crp, which is a measure of inflammation in the system, it basically something's wrong, but it's very nonspecific.

Now, these are measures of more or less inflammation in the brain, but they're not going to tell me why the brain's inflamed. And that's going to be a really interesting question.

If those basically say, yes, something's up, something's wrong, something's out of balance, then it kind of inspires us to do more detective work to understand why is it the toxins, is it infections, is it stress, is it sleep issues, hypoxia?

You know, any of the list of things that we talk about all the time again? But they're going to be something we can use to maybe even track change over time.

Is that accurate? We can get sort of baseline, say yes or no. This is an issue, put our protocol to work and then tested again and make sure it's going in the right direction. Is that right?

That's exactly right. So so as it's not just inflammation, there are other insults, as you mentioned, that can do this so fast. Photo is a good example.

It's telling you that you are pulling back on things. So if anything that's causing that. So now what you want to do is treat this person and you want to see this hospital go down.

And what they've seen is it takes about six months or so to be in a state. Now, what's interesting, you know, we're always looking for what's better. So MRI takes some time to change.

We saw it. We saw definite changes within nine months in our trial. Cognitive testing can happen fairly quickly, but it's relatively although you can quantitate it, there's some subjectivity and people would like biomarkers as well.

There's electrophysiology. You can look at changes in P300 and things like that. But most clinician, most clinics are not set up to do the electrophysiology.

So here are some simple blood tests that will help you to optimize things. And they can say, okay, Heather, you're really going in the right direction because this phosphatase is just plummeted.

Or they may say, you know, Heather, there's actually a few things you might have missed here or you may have not checked for yet. Maybe you have to push a little harder on this or push a little harder on that.

And because it's come down a little bit, but it hasn't come down too much. And I should mention in the anti amyloid antibodies, what they were showing is with that as a monotherapy, they had to push massively.

They had to remove 75% of the amyloid, which of course, has all sorts of side effects just to see a tiny reduction in the hospital. So my hope is that with the appropriate things being done, we'll really be able to see a much greater reduction in town.

You know, we'll see hospital should it should go down as we do the right things for these people. How exciting and how how fun to be able to measure these things.

You know, as a clinician having these tools and our tool belt is really exciting for me.

Why amyloid drugs have limited benefit

So you just mentioned some of these amyloid antibody treatments would you take us through those? Because I think a year ago when we were having this conversation, Aducanumab was available, but now they're doing so.

Would you just help us think through like when that would be appropriate to use, if ever? Yeah, this is a great point. So the problem here's the problem in the 1990.

So now, 30 years ago, it was suggested that, hey, amyloid may be the major bad actor here. Let's just remove the amyloid. And everyone got excited about that and let's develop things that will remove the amyloid.

Initially, as you know, it was tried with active immunization and that led to some unfortunately, some deaths because you were creating a tremendous amount of inflammation within the brain, just the opposite of what you wanted to do.

And so then people said, well, let's, let's then make antibodies and do this passive. We will administer the antibodies. Now the virtually all of them have failed.

So Papa Newsom have had no one improve. There was no improvement overall in people with rapid news of Mab Solanezumab huge studies, no improvement there.

Cronyism began to narrow mab all of these things unfortunately have failed. Then as you mentioned a year ago along came out a can of Mab. Now it failed in some studies but in one study at one dose there was a 22% slowing.

And so in a but somewhat bizarre FDA decision because all of the all of their consultants had recommended against it, they said, well, since we know that you're removing amyloid, that's probably going to be good for you.

So even though it doesn't seem to help, cognition will assume that it might one day and will will give it accelerated approval. It was really backward thinking.

Unfortunately. And now fortunately, some more sage wisdom came in and groups from the EU and of course Medicare and the Cleveland Clinic and on and on said, we're not going to use this, we're not going to pay for it unless it's part of a clinical trial.

And so I think that's really kind of put a halt to wide scale use, large scale use of aducanumab. And then you've, as you mentioned, looking in, a man came along and of course, Danino Mab is following quickly on its heels.

Danino Mab being the one from Lilly looking into Mab did slow the decline, did not make people better, did not stabilize them, but it slowed the decline by 27%.

And of course, it was hailed by groups who were being paid. It was hailed as a major breakthrough. So my argument is, look, if if Elon Musk told us that Space X, everybody in those rockets was exploding and dying every time.

But in a major breakthrough, they die 27% later. You know, we probably wouldn't hail that as a major breakthrough from Elon, but that's what, you know, that's what the argument has been.

So they're still pushing for coverage with Medicare. They're pushing for full FDA approval. Again, it's just accelerated approval that's happened so far.

And, you know, time will tell. There's a lot of lobbying, there's a lot of politics going on. So one of the things I'm interested in is, okay, based on what they published, which is 27% slowing in people who had MCI.

So these were actually not even in people with dementia. It was MCI and early stage dementia. What has followed, what has actually published better results than that extra virgin olive oil alone has published a better results than lacking a MAB.

Now, to be fair, the study wasn't big, you know, it wasn't as large. But the fact that something like that can do better than it tells you a lot, right thing combined metabolic activators and these are this is just four different things that have basically improved energetics.

And then I should have mentioned along those lines what came out of our lab. We could ultimately boil down to two things a d equals ie a over E, in other words, Alzheimer's disease equals immune activation over energetics.

So what we want to do is determine why you're a need system is active and I should say Dr. Aleksei Karak, and with whom I've collaborated for years, is pointed out this is really mostly about innate memory.

So you store your memory in these three locations, you store that in your bone marrow, you store it in your endothelial cells and you store it in your tissue.

Macrophages, which of course are your microglia. So energetics, right? That's half the equation. But you also want to bring down the innate immune activation.

The next thing that's done better than the keto MAB is ketones and of course Professor Stephen Kenny and very nice results in patients with MCI on ketones.

Then of course, the other thing has been the RICO protocol, which has done very well. We have a published trial on that and I'm very excited about the trial that you've submitted that is undergoing review currently for your revisions, very excited to see that published and also hopefully to come out in the upcoming book.

So I think that, you know, all of these things have led to very clearly improved results over these antibodies. And it's not surprising these antibodies are removing it's essentially like removing one cytokine, a long acting cytokine.

You're not getting at what's causing the problem. And also, you're you're creating some negative effects because you're now tearing these things out there.

The amyloid is sitting in the blood vessels and is also sitting in the extracellular spaces and is also in intracellular. So you're ripping this stuff.

It's a little bit like taking a patch off a tire and you're now, you know, no, no surprise. You get leakage, you get bleeding into the brain, which has been documented repeatedly.

Now, where I think these would be great, but no one has proposed using them this way yet. Is do the other things first, remove the insults, get the energetics up, get the inflammation down, improve people overall, and then use very low doses to gently remove the amyloid that is left.

Because to be fair, it is a long half lived molecule, so you want to remove it slowly. The good news is there is a dynamic relationship between the soluble and the plaque associated amyloid.

So as long as you're removing it slowly over time, you could do a great job. But you know, in contrast, this idea of doing nothing else and just ripping out all this stuff that has been laid down because you were under assault without changing any of the insults, it is really barbaric.

And it's just it doesn't fit the biology of the disease. It's kind of crazy. We would expect great results from that. Right. So I want to restate slightly differently and just to drive home this message for everyone, those medications, although they've been approved by the FDA and this kind of weird, funky way, they don't reverse the disease process.

What they do is they slow the very painful decline. So from my perspective, this is almost cruel, right? You're asking families and people to go through this torturous process longer, to experience the decline more slowly and yes, I can understand that there is some benefit in having someone be a little better for a little bit longer.

And yet there are alternatives that are available and publish. There's published literature supporting them. And one of those is the RICO protocol, a doctor reticence protocol.

And it shows not only does it slow decline, that's not what we're after, but what we want is an improved quality of life, improved cognition, ability to perform activities of daily living, the maintenance of our dignity.

Right. That is what we really want. And what we see with the Bredeson protocol is that we get improvements in cognition. We would consider it a miracle if we could just maintain the cognition, not slow the process, but maintain cognition, stop the process, right.

What we see is an even bigger miracle is that most of the time people in the same category, MCI or early Alzheimer's, no improvements in their cognition.

And so this is really, really, really exciting stuff. And it's available to you right now, that is. And you don't have to wait for a drug or approval from the FDA.

You can get started today. Exactly. And you know, I should mention one of the most important things is that the improvement is sustained. When you go after the right things, then you actually are turning off the problem.

Now, let's compare that to the drugs they're in. There are two major classes of drugs. It's the antibodies that are removing the amyloid, and then it's the chemical approach, which is Aricept and Namenda.

So Aricept and Amanda, when they looked five years after starting it, were actually doing worse than the control group. So it's a short term solution, not a long term solution.

The antibodies. And Heather, I'm sure you saw this paper that just recently came out when they looked at the brain atrophy. The atrophy was worse in people who had been on the antibodies.

So unfortunately, the this the damage from kind of going about this the wrong way and ignoring biology is that we have the long term effects not being so great.

And again, I think that the future is definitely going to be combining these protocols and optimize these personalized precision medicine protocols. And then you use a targeted drug here and there.

You want to remove a little bit of amyloid. Fine. You want to improve the cholinergic transmission, fine. You want to go after fast photo.

How the Bredesen protocol improves cognition

And as a simple example, lithium is a very good way to inhibit GSK three beta, which is the major kinase. One of several p38 is another one and CDK five and things like this.

But the major one that is phosphorylated tau in Alzheimer's is GSK three beta and you can inhibit that with lithium, for example. I'm curious about the dosing rate because some people think of lithium for bipolar disorder rate, it's 100 milligrams, three grams.

But I remember when I was in training actually Jonathan Wright and Alan Gabe had published some research around or they had maybe synthesized research around using just like five milligrams or even 99 milligrams is another dose that you see regularly used for memory and sleep and even antiviral support.

So is that more the dosing that do we get brain benefits with that? Yeah. And a lot of people will use, you know, 10 to 20 milligrams of lithium irritate is kind of typical and I would you know, I recommend talk to Dr.

Katz who is like a functional psychiatrist and part of both of our trials. And, you know, she she argues that you can go up to higher doses without any problem.

So, yeah, you want to you know, you want to be careful. You don't want to be going up above 300 milligrams and you want to be careful about renal function, of course, but I think it's unlikely you're going to have any trouble with 50 or 100, that sort of thing.

Using it as a supplement because lithium, it's it's a mineral can be very, very safe and very effective as. Well. Exactly. So I think know what's happening.

We're getting this larger and larger armaments. And another good example is commentary where it was it was actually tried as a monotherapy years ago and it failed.

But then they noticed in a small group of people who were Abawi for fours, and that just represents 10% of Alzheimer's patients and only 2% of the population there actually did seem to be some benefit.

Now, what that does is interesting. Instead of trying to rip out the amyloid and adding antibodies where you now have to worry about all the all the things that are happening with antibodies in your brain, this is now preventing the oligomerization of the beta.

So you have the less damaging monomers, but you don't have the more damaging oligomers, which is a nice idea. Now they're in actually in trials with a related drug, which is a precursor for homo taurine.

But I think it's an interesting one and this is again available over the counter and people are to be aware typically in their trials they were using 300 milligrams a day in divided doses, either twice a day or three times a day.

And, you know, again, this is not such a bad idea. You're as you're removing these things and you're trying to help yourself by also tweaking some of the mediators, especially in people who have large amyloid burdens or who have reasons to be all of them are rising.

Their amyloid, which is basically things related to inflammation. So again, the armamentarium is huge. And knowing how to use it, when to use it and what's in there is where I think people like you are getting such great results.

You have this analogy of thinking of your brain like a country. I use this every single day. I repeat it is that if your brain is and you say My brain is done right, if you think of your country and your brain is in fight and defend mode.

Yes, this amyloid plaques and these tao, these misfolded proteins, they're essentially part of that defense. They're part of that attack and defend mode.

They're telling us that there's something there to attack and defend and if we rip them out, that we've kind of gotten rid of part of our attack and defense.

And it's too early for us to be switching towards regeneration rate. If we're fighting a war, we don't need to be

building schools and roads or synapses and new neurons, but we want to really be doing is resolving those things that led us to war.

Right. And so that's what I hear you describe it is there's lots of things we can measure that will tell us if we're or not. And then let's allow that to trigger this this move in the direction of identifying and treating those really causal level pieces of which this entire summit is about that.

So if you have any questions about that, they will all be answered this week. Yeah, the point now. Switching to that that regenerative mode, but let's lay down those tracks.

Let's build new. But we don't usually have the resources to do both at the same time. And although we want to be of course supporting with nutrients and there's some nuance to this and it's really important to work with the states and train provider.

It there's there's there's so much we can do from a lifestyle perspective. And certainly my work has been about making this pragmatic and practical and easy to implement as possible.

Yeah, and you've done a fantastic job with that. So yes, if you're in my brain stand and you know, the World Trade Center is going down and you're saying, what the heck is going on?

Then, as you indicated, you've got to start by asking where is this coming from? And it just kills me that in clinics today, people are not asking that enough.

And if you go to a neurology clinic and you say, yeah, I'm having trouble with my memory, and they say to you, Oh, well, you have Alzheimer's disease. Well, yeah.

What caused it? Well, no one knows what causes it. Well, actually, we have a lot of information, what causes it. And it it it is literally a network insufficiency.

So what happens? You get that idea over, you get the innate activation and you get the energetic reduction and that then is putting you into a mode in which you your network is insufficient.

So you're literally downsizing this. And as you mentioned, you're now using your resources not to build and maintain synapses, but rather to fight with the various inflammatory ends and pathogens and toxins and things like that, so that you're now read, distributed and your resources.

And unfortunately, you're living with a smaller brain. And I think one of the things that, again, that's just come out recently was some wonderful work from University of Colorado and from Rick Johnson, showing that when you're when you're essentially triggering fructose metabolism, either because your glucose is very high or because you've got a lot of high fructose corn sirup, and it's typically not so much just from eating fruit because there's plenty of fiber in fruit and things like that.

But because of inflammation stress, it's one of these stress responses goes along with high uric acid things, all these sorts of things. Then one of the things that that does is reduce your ATP.

So you literally are going into a lower energetic state physiologically. It's because you think winter's coming. So you're like, Okay, I'm going to now go into my cave and I'm going to have a few months here and I'm going to conserve resources.

That's not a good thing. When you're trying to keep your brain functional, you're again, it's you're pulling back. Your ATP levels are actually declining so that all of these things lead to this protective pullback that now decreases your synaptic count.

And you can look and you can it tells you a lot about why people get this when they get it. You have to lose a certain number of synapses. So no matter how bad things are when you're younger.

The second thing to know is you can lose synapses anatomically or chemically. When you're when you're losing them chemically, you can bring them back more quickly.

When you're losing them anatomically, it's more difficult. So you kind of have to keep count on both methods. Are you losing more anatomically or chemically when you hit that threshold, then you start having symptoms.

So we want to get there before the threshold, which is where these new tests are going to be very helpful. And as you indicated, we remove the problems, we optimize what's there, and then we get into a regenerative mode.

And that's where things like intranasal trophic factors and stem cells and hormone optimization, all these things are so, so helpful. And again, you know, we should be able to do better and better as we apply these things appropriately to people.

And there are more and more things in the armamentarium that are so helpful. Though, as you well know, in 2017. And then again in 2020, The Lancet published the kind of comprehensive report on Alzheimer's and dementia.

And they list the the factors that are basically that we can manage, that we can do something about. So there are risk factors like age and gender that we can't do anything about, right?

The whole list of now 1517 modifiable risk factors that we can change. And they suggest that 40% of dementia could be prevented or even completely don't have to happen at all.

Right. And this is from The Lancet, which is a highly reputable journal in the U. K. Now, why is it that knowing that if that that that literature is out there, that it's in a highly respected journal, why do people still go into a neurologist?

And are they still told there's not much we can do to prevent or reverse this disease? Yeah. You know, one of the points I made in the paper we just submitted was that we must retire.

The phrase, the assertion that it's been used year in, year out, which is that there is nothing that will prevent, reverse or delay Alzheimer's disease.

It's just it's been proven incorrect. It's been proven incorrect by the finger study, by our clinical trial, by your clinical trial and by others. So it's kind of at this point, it's kind of silly to say that.

And yet you hear it all the time now as. You state it heartbreaking because people are suffering. That is people that's the refrain. And people are still hearing it, even though it's so outdated. Now.

I agree with you, this is a real problem. And then the other one that I did always kills me is they say we don't know what causes this disease. Well, actually, we know a lot about what causes the disease.

Clinical trial results and study design

Now, you mentioned The Lancet, and I think that's great. You know, what they tell you is don't don't smoke. You know, get your blood pressure taken care of, you know, some basics.

This idea that you could prevent 40%, I think that's a huge underestimate. We should be able to prevent the vast majority. You know, as I mentioned, when you and I were talking earlier, I have talked to so many doctors to say, have you seen anyone who's gone out, has gone on active prevention, done the right things, being compliant and yet still develop dementia?

I've never heard about a single case, so in fact we're quite good at preventing dementia and reversing, especially in the SCA phase and especially in the MCI phase, 84% people of people improved in the in our trial and in even in some cases, as you've indicated from Miramar, you have people with very low MOCA scores who have end stage dementia and are still improving.

Now they're not improving all the way back to normal. That's the sign. That's our goal. As scientists, we need to understand what will take someone from a moca of zero to a moca of 30.

And along those lines, as you know, we're also now extending what we're doing to other degenerative diseases. So I'm very excited about that possibility.

And I think that, you know, we should have a place where people from all over the world can come and have treatment, especially early on, in any neurodegenerative condition.

So I wanted to share a little bit more about the research because you and I are both very involved in that. So we've talked about some of these other trials that have been going on.

And then you referred to this 84% number. So which is just describe the clinical trial design of that and the follow up to that. Yeah, great point. So this was a trial that we published in just last year, 2022, and in that it was a proof of concept trial with historical controls.

So we took 25 people who all had to meet criteria for mild cognitive impairment, MCI, which is really the third of four phases, or early dementia, which is the fourth of of the four major phases.

And these people had MOCA scores of 19 or above. They had to have abnormal CNS, vital signs, scores, and they had to have complaints. So they're a huge 21, which is where their partners will say, are there problems?

They had to score over five and anything over four is considered MCI and over 14 is considered dementia typically or correlates with dementia. So they all had to have over five to get into the study.

Then they were treated for nine months with a precision medicine protocol. So we looked very extensively at we looked at their genetics, looked at whether they had no hyper coaxial ability who actually worked with Intelex DNA.

And Sharon Houseman Cohen, who did a great job at looking at the genetics for these people. Then we also looked at their biochemistry, looked at what they were doing.

We then also looked at their MRI with volume metrics to see whether they had atrophy. And then over time we would also look back at their MOCA scores and their CNS vital science scores.

They were treated with the protocol we developed. So we looked, we subtype them, and then we looked at what are their inflammatory things going on? If so, what is it?

We addressed the inflammation we addressed the glycol toxicity. We addressed the low hormones, the energetic failure. We addressed the vascular changes, we addressed the toxicity, and we addressed any sort of thing that we could do in terms of preventing trauma or healing previous trauma with things like BDNF and that.

And they were treated that way for. So this was a, you know, was different for each person. We had appropriate diet, exercise, sleep, stress, brain training.

The usual basic seven things. And they they did very, very well. 84% of them improved their scores on CNS vital signs 76% improved their MOCA scores. And then interestingly, their MRI's also improved their gray matter, which declines a little over 2% per year in people with MCI and Alzheimer's actually went up instead of down.

And their hippocampal volume, which did shrink slightly, shrank less than normal aging and far less than someone with MCI or Alzheimer's. So with all these different parameters, they did better.

And by the way, they also did better with their insulin resistance. They did better with their vitamin D, they did better with their homocysteine, they did better with their hs-crp.

So all of these things improved in accord with their cognitive improvement. And so exciting. And so this was over

nine months, right? And independently I was running a very similar trial.

We looked at slightly different things, but had 23 participants. Although our participants were a little more advanced in their cognitive decline. So we took participants with MOCA scores of 12 to 23, and instead of seeing as vital signs, we looked at Cambridge Brain Sciences, which is another battery of testing that test cognition a little more, more specifically and and with basically a more scientific way than our clinical MOCA score.

Right. And we did this over six months, so it was a little bit more compressed, it was a little quicker. And we got almost identical results to what you guys got.

We saw that 74.9% of the time or maybe it was excuse me, 73.9% of the time we had our participants improving their MOCA scores, and the Cambridge Brain Sciences had the same participants improving their cognition in that way of looking at it as well.

And so very similar study design and very similar results. And so other years was published a year ago and July of 2022 in the Journal of Alzheimer's Disease right now, minus in peer review and hope.

We're hoping for publication this summer. Yeah. So, yeah. So these are things that are advancing the research so that more and more people can have faith that this is something that will work for them.

Now, one of the very one of the criticisms that's totally normal and makes plenty of sense is that these are not controlled trials. And so that's what's next, that's on the horizon.

And you are currently recruiting for a follow up trial. So both of our trials where feasibility trials, right. And we were just looking to see, you know, this is a complex approach.

This is a very complex protocol. It's a lot to ask someone with cognitive decline. So we wanted to know, is it feasible? Can we do this? And sure enough, not only did we show that we can do it, we showed that we get really phenomenal outcomes, miraculous outcomes.

And so now the next step to do is to have these controlled trials. And I want to just make sure everyone understands that when you hear that there's not enough research around this. That's true.

It's true. We get it. Yes. And we are working as fast as we can to get the research out there. I have personally had funding for a trial since 2019, maybe the end of 2018.

And here we are starting in May of 2023 and it hasn't been published yet. And part of this is covered, but part of it is just the snail's pace of research.

And Dr. Reticent, if you would, maybe share about your follow up trial, the controlled trial, and then also share a little bit about why it was so hard and inserted this paradigm of research that we exist in, that it was so hard to do a multifactorial intervention.

And even though you had funding, you weren't able to start those trials immediately. It's such a good point. And you know, when you are changing the standard of care, it is very difficult, as they say.

You know, and this goes back to Machiavelli, who said this is the hardest thing to change is is the status quo, because the people who are doing well with status quo are not going to budge.

And the people who would do better with the new are kind of waiting to see what happens. So it's going to be tough to change it. And as you indicated, and we tried to do this trial back in 2011 and were turned down in 2011 because it's not your typical trial.

They wanted something with a single variable. And I think we all understand it's easier to do the math. You know, it is a it is a simpler and more obvious approach when you change a single variable.

But the problem is to say, you know, we're going to do this in a more scientific way, but poorer outcomes, that is a horrible tradeoff. You know, we want to start with what are the best outcomes and then and then form the trial around that.

And the research is showing the laboratory research is showing this is a multiple contributor disease. This is not a simple disease like pneumococcal pneumonia, where you can just target one pathogen and everything's great.

You're now targeting a network, a system. And so to do that, you've got to tweak it at multiple points. It's a different way to do medicine. And I hope that five or ten years from now it will be the norm.

But it takes a while to convince people. As you indicated, I think there's a moral imperative that every neurologist is aware of this and has some basic understanding of how to deliver this to patients or refer them to somebody who does get it.

Yeah. These aren't. Suffering tissue. Tell us about your new trial. And I'm sure there are people who are listening who are interested in understanding if they could potentially be involved.

Yeah. Thanks so much for mentioning that. And so, yes, as you said, the next step now is a randomized controlled trial and this will be the control group.

You have to be fair to them. They're still looking for something. So what we do is we simply delay their treatment for nine months so everybody will get the treatment.

And if you're delaying, you're just doing it. You're doing standard of care. You're still going to get what's the standard of care for nine months, and then you're going to go on the protocol, if you want, at the end of that.

And this is going to be done with Dr.

Future trials, prevention, and broader neurodegeneration

Craig Tannehill, who is down in Hollywood, Florida, Dr. Nate Bergman up in Cleveland. Dr. David Horsey, who's in Nashville, Dr. Christine Burke, who is in Sacramento or actually Folsom just outside of Sacramento with Dr.

Anne Hathaway here in Marin in San Rafael, and then Dr. Kattouf, who's out in the East Bay. What an all star team. I just love it. Fantastic. And all six of these people have seen this with their own hands who have produced that have had tremendous, tremendous results over time.

I never tired of hearing of great results from physicians who are doing this well, as all six of these are. And so I'm really honored to work with them.

We're also honored to have Diana Merriam and the Four Wins Foundation that's a fund that funded the first trial. And they are actually now funding this second trial again.

So we're very, very excited about that. And the great thing is there are a lot of new tests, as we talked about. So now we'll be able to look at epigenetics for brain aging, for biological age.

We'll be able to look in some, some cases we'll have some electrophysiology as well. Not all of the sites, but some of the sites will be able to look at FOS.

FLATOW And and will be able to look at GFP and things like that. So we'll really be able to get a great look at whether what we're doing is driving this in the right way.

And because we can look at it along the way, we can actually make some tweaks to see if, okay, it's coming down partly, but it hasn't come down as much as we'd like.

Let's now make changes or let's look at what we might have been missing, or we could optimize and see if we can get even better results at nine months.

So we're very, very excited about that. That is so exciting. So I'm just waiting for my paper to be published and then we're going to go on our follow up trial on our randomized controlled trial.

And I will be picking your brain and Kat's brain and anybody who will talk to me about how to design that based on what you guys are learning. Because, yeah, it's time to move all of this forward and make sure that we're answering those questions about getting enough research out there that shows that this really is something that will benefit so many families who are faced with the potential of going down the scary, dark path of Alzheimer's.

You know, if there's one thing that we could change, it would be to move people earlier. So I just today got the data that if you look at the peak of when people are going on prevention, on the prevention that we develop versus when they're going on the reversal we developed, the peak for the prevention is in the sixties and the peak for the reversal is in the seventies.

If we could move those two decades earlier and even if we could move them one decade earlier, but ultimately I'd like to see them forties and fifties.

There would be very little dementia. You really could make a huge impact. So if you're the child of someone, but. Yeah, yeah, get everybody in the family evaluated and get on active prevention.

That's the best thing you can do for your family. I think in the future. Everyone's 40th birthday should include a present of prevention of cognitive decline, and as you've indicated before, it also improves the cognition you have.

Yeah, so there is just so much more that can be done and it's just a matter of changing the zeitgeist. Everyone's so used to hearing, Well, there's nothing you can do, so just wait, wait, wait.

And we hear about this all the time. I just it kills me. I hear about celebrities who have waited until it's very, very late. We need to get people to come in earlier, recognizing there's so much that can be done.

It's so much so, you know, this isn't just about Alzheimer's for you. You're a neurologist and you are I mean, some people may be obsessed with the pathophysiology and the science of how this is all happening.

And what you found with your research is that this doesn't just apply to Alzheimer's, that there's actually a more general application for these concepts in these interventions.

Can you talk about some of the other diagnoses that have where there's demonstrated benefit. Is such a good point. So what I'm doing right now is actually writing a paper on the Unified Theory of Neurodegeneration so that what our research suggested is very different than what has been in the literature over the years.

The literature over the years has said it's a you know, it's about reactive oxygen species, it's about metal toxicity, it's about misfolded proteins, it's about aggregated proteins, it's about prions.

And again, yes, there are prions. There's no question about that, that tremendous work to show that these things do they do have this prion like effect, but they don't seem necessarily to be causative.

This is, again, a response to these various insults. It's one of the mediators. So all these different ideas, what our research suggested is that all of these are network insufficiencies.

So you have a supply and you have a demand for every network. So you have a network that some serves motor control. That is what goes awry in Parkinson's.

You have a network that is neuro plastic, which is what goes awry in Alzheimer's, you have a network that supports your macula, which is in macular degeneration, what is not supported enough.

So what we can see is that in all these diseases, the supply is too low and the demand is too high in a chronic or repeated way. And it can be because of pathogens or toxins or what have you.

The cool thing is each of these has its own network with its own Achilles heel. So we know that in Parkinson's, for example, the Achilles heel is complex.

One of the mitochondria. Anything you do to inhibit that, whether it's Paraquat or whether it's MP P or whether it's TCE, try fluoro ethylene, which unfortunately people are getting exposed to all the time and they don't realize it until they get Parkinson's.

It's horrible. Any of these things, you're going to change that network support. You're going to start dropping in. Of course, your supply is dropping because of all sorts of different things in macular degeneration.

The biggest one is actually smoking. And in Alzheimer's, you could argue that the biggest one really is insulin resistance and metabolic abnormalities that are so common in our country.

But of course, there's a huge role for things like Mycotoxins as well, because. Sleep apnea and. Proinflammatory and sleep apnea and so on. The good news is with each one of these now, we can start listing all the things that can that can contribute.

We can check them, we can now change this. And we've seen improvements for example, in people with Lewy Body Disease, which is really exciting to see.

We've seen improvements in people with vascular dementia and we've seen some improve, some initial improvements in people with dry macular degeneration and again with dry macular degeneration.

If anything, that's a simpler disease than Alzheimer's disease. So there are 11 million people with macular degeneration more than Alzheimer's in the United States.

And the current approach is essentially to wait for it to become wet, which is where you're now actively bleeding into your eye and then give injections into the eye.

This is barbaric. And what do you do with these injections? Again, it goes just the opposite of physiology. What it's doing, it's preventing you from allowing your blood vessels to grow with your macula, are crying out for support.

And this is saying, no, we're not going to let you do that. So what do you do? It increases your risk for geographic atrophy, which is an end stage form of dry macular degeneration.

Let's get in. At the beginning, the optometrists and ophthalmologists are seeing this all the time in these early dry stages and typically using an approach called a Reds two, which doesn't actually decrease your long term risk.

So it so unfortunately, it really does not work very well at all. What we want to do is actually improve where you stand and we want to prevent you ever getting too wet.

Macular degeneration by you know, by again, just by doing appropriate physiological things, getting the oxygenation, getting the supply, getting the blood flow, reducing the toxin load, improving the inflammation.

All these things are critical. So there is overlap as you, but each one has its own sub network that has its own Achilles heel. This is such a hopeful message and so exciting for those who are suffering or know someone who has suffered.

You have in your work with Apollo have made this thing accessible and now there's a new partnership with nutrition for Longevity where there's even meal delivery services.

I want to make sure that our audience understands what support is out there, because as you know, we have been

talking about this can feel overwhelming for people.

It's a lot to do and it does require work and some resources and yet it's so worth it. And there are tools out there to help you to implement this at home.

So would you share a little bit about Apollo? Yeah. So glad you brought that up. So Apollo is a software company that has basically created a community so that we can begin to get people to, first of all, to understand what's driving the problem and then ultimately to do the right things about it and the idea here was that as physicians, you know, there's only so much we can do.

We can't look at a piece of paper and say, here's your 3.33 billion base pair genome and here are the changes. So you need computers to read these things.

You need to have them. And ultimately, there's a lot that people do today. But for the future, you'll you'll want that little helper, which is a software that says, okay, don't forget, these things are those things and here are things. Here are the things that can be helpful.

And as you mentioned, probably the most important recent change is through nutrition for longevity. And I'm really thrilled because people have said to me over the years, look, starting with diet is the thing to start with.

Everyone should get on a plant rich, mildly ketogenic diet. But you're telling me I got to go to the store? I got to think about this. I got to go home and cook things.

I got to think about, okay? Everyone said, Look, can't you just send me some meals? So finally, yes, the answer is yes. And they're actually relatively inexpensive.

I really like what they've done them. I sampled them several months ago and I was actually shocked at how good they were, so I was really impressed. Nutrition for longevity really did a fantastic job and we're grateful to them.

These now can be shipped anywhere in the US and soon, hopefully outside the US, but certainly at the moment anywhere in the U. S. you can get these very easily.

And the, of course the the for the future, the goal is that these would ultimately be covered by Medicare because they will be part of what's improving cognition.

But as I say, they're not terribly expensive. You're going to be paying for something somewhere to eat anyway. So they've done a really great job with this.

And this is a plant rich, mildly ketogenic diet. When I when I did it myself, I checked in. Yes, my ketones went up on these things. So I really appreciated and they're delicious.

They've got all sorts of things and there's a pescatarian one that you can choose or in other ones you can choose as well. So this was on I should mention that Julie G and my wife, I eat a lashing by this doctor.

Dr. Aida LESHIN did a great job with putting these together and working with nutrition for longevity. Oh, fantastic. I know there's going to be people listening to it a little happy dance at home right now that there's finally an option that is read.

It's an approved and working through Apollo and this is just going to make it so much easier to get those outcomes that people got in the trial. And there was another trial.

We have just a couple of minutes left, but I just want to mention Rob Rouse. He was a great author on a trial that showed this was it. 55% of people who were enrolled in Apollo had improved cognition.

And I bet that that number is going to jump a ton when there is access to this meal delivery. You know, I'm glad you

mention that, because this is very complimentary.

So the idea was, instead of these trials where we're you know, we're controlling certain things, we're bringing people in and we're saying we're working with them one by one.

The complimentary piece that you really want to know is what's going on out in the community. If you have trained physicians who are seeing people and treating them, but they're doing their own thing, sometimes they may be changing things.

They may not have the same follow up. It's not really a trial. It's a pragmatic, essentially observation what's going on. And as you said, right around 50%, just a little over half the people.

So it wasn't as good outcomes as it was in the trial, and yet it was far better than any of the drug approaches. So again, we are continuing to optimize this.

We're continuing to get better and better ways. And you've done a great job with simplifying, helping to simplify things. We want to make this as simple as feasible as possible for everyone, one, to avoid cognitive decline and to reverse cognitive decline.

Dr. Bateson I could not be more grateful to you for your mentorship, for your kindness, for your support and for your time, especially today, sharing with our audience.

Thank you. Thank you so much, Heather. Here's to great outcomes for all of us. Cheers.