Why Alzheimer's Is Becoming Optional Transcript

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Introduction and Guest Background

– Welcome to another episode of reverse inflammation. Summit body and mind longevity medicine. And I'm very pleased in this session to have a fellow professor from U.

C. L. A. Dr. Dale Bredesen who is a pioneer in brain health research and the author of the bestselling the end of Alzheimer's. Dale. Welcome to our program. Thank you for being here.

– Great to be here Steven. Thanks so much for you to Rob and to you for having me on. – So your area in Alzheimer's. I know you've said that it's the third leading cause of death.

And of course our program is about aging and longevity. How did you get into this field in in the first place? What what inspired you? – Yeah that's a great point.

Of course nobody wants to live to 100 and have 20 years of dementia as the last 20 years. Um I got interested in this as a neurology resident because you know as a medical student I thought you know if you don't have your brain functioning what do you really have?

That's the thing that's that's really critical to be a human being. And as I went through my neurology residency, the striking thing to me was we neurologists have so little to offer our patients therapeutically and if you look at it of course the history neurodegenerative diseases from Alzheimer's to Lewy bodies.

Frontotemporal dementia to a L. S and on and on and on represent the area of greatest biomedical therapeutic failure. As everyone says you know there's everyone knows a cancer survivor. No one knows an Alzheimer's survivor.

And so I wanted to understand the fundamental nature. So my laboratory for 30 years was all about what is the fundamental nature of the neurodegenerative process. Can we understand that so that we can begin to fashion effective preventions and treatments. – [s] Beautiful. Beautiful.

That's great. And can you give our audience your perspective and it's something we ask all of our guests your perspective on the process of aging and longevity. – [d] Yeah. You know, this is a fascinating because there are so many things now that were that are coming out from the epigenetic studies, looking at biological aging and actually seeing that you can move biological age backwards as I'm sure you've talked about, but you can also move it forward to more rapidly with things like ongoing inflammation. So I think we're really getting at what is the nature of this.

And you know, and I was at an aging research institute for for years, the Buck Institute's founding president and CEO there. And so we were looking at what are these processes and it really boils down to essentially three things, Number one, you have a genetic program that gives you a lifespan.

How Aging and Longevity Work

So in other words, if you're a mouse, it's very hard to get to 100 If you're a human, it's a lot easier to get to a human, you have a pre programmed length if you're a human, it's very hard to get to 500, but we want to understand why that is and then within that you basically have two processes. One is a process that's just about you suck at living. In other words, the thing that's driving aging in many of us is we eating horrible processed foods, pro inflammatory foods were exposed to all sorts of toxins, we're doing everything wrong and that is part of it.

Then if you do everything right, there's still an underlying change, changes in your telomeres, changes in your methylation patterns, changes in essentially in your stem cell population.

So there is that underlying So I think of aging as being those three things together, a genetically pre programmed lifespan you're gonna live about X plus or minus.

Then on top of that, you have, are you good at living or not? Are are you doing things wrong or? Right. And then on top of that there are these underlying processes that are part of the of the aging of our soma is basically, – [s] so maybe as we um focus more on your area of Alzheimer's, we can sort of tease apart some of these considerations that you're mentioning and to start off, I'm wondering you have a notion that the standard model of Alzheimer's has gotten something's wrong.

Can you explain that to to the audience? – [d] Yes, I think you've been very kind to the community to say that they've gotten something's wrong. I would say I've taken a step further, the entire field is backward because of the fact that this disease has not been understood for what it actually is?

And so we're told this is a disease. And by the way there are dozens of theories it's a disease of misfolded proteins of aggregated proteins that you know that that are now you know that that are now interacting with each other.

It's a disease of prions of amyloid of tao of herpes simplex uh you know on and on and on of reactive oxygen species, you know of D. N. A damage, you know on and on and on.

And the reality is it's none of those simple things. When you look at the epidemiology, you look at the neuropathology, you look at the genetics, you have to come away with an understanding that fits all of these.

You can't just say, well it's just this and then it turns out that the epidemiologists show that you're completely wrong. You have to have an internally consistent model.

And when you look at all the different things and there are of course there are dozens and dozens of things that we know contribute. And of course air pollution being, you know in L. A.

You got to be concerned about air pollution. There's a lot of information over the last few years on increased risk with air pollution but of course insulin resistance and you know on various toxins and various inflammatory genes and and various pathogens and leaky gut. So how the heck does all this fit together?

Well the way it fits together is this is a ultimately all time. What we call Alzheimer's disease is a network insufficiency. You have a beautiful plasticity network within your brain and you can go right down to the molecular species level. Amyloid precursor protein, the parent of a beta of amyloid is a molecular switch that responds both to positive things and to negative things.

And so when it is seeing that you've got ongoing inflammation or you've got toxin exposure or too little energetic support or too little trophic support.

It will now essentially change your brain put your cells into a protective downsizing mode essentially it's switching you from making and keeping synapses to pulling back to protect yourself. And there's a direct analogy to what happened to our country When we entered the pandemic. So early 2020 as we all know, you have a path in this case you have an insult, we respond to it.

Rethinking the Alzheimeru2019s Model

They say shelter in place socially, distance, don't go to work all this stuff and of course the country went into a recession, your brain is doing very much the same thing when things are good and you can trace for example, estradiol one of dozens and dozens of of different molecule and molecular species estradiol binds to its receptor enters the nucleus alters the transcription of hundreds of genes and one of them is the alpha secret taste that cleaves your A P P into the two fragments S. A.

P. P. Alpha and alpha CTF which signal growth and maintenance. At the same time you can look at NF kappa B which is responding to inflammation. So anything that's inflammatory also enters the nucleus affects hundreds of

genes. And two of them are the beta and the gamma secretase which now take your A. P. P. Into this downsizing mode.

So you're literally saying, am I allowed to grow and maintain or do I have to downsize and really put my resources into protection? So the amyloid that we have vilified in this disease is a protective agent. That is antimicrobial as professors Robert Moyer and Rudy Tanzi ship from Harvard showed a number of years ago.

– So I love what you just said because we're learning in a lot of different areas that the brain body have two choices. One is to grow, the other is to protect. – Exactly. – And I'm wondering if if the conclusion from what you just said is it's not a particular process.

It's how it's what determines which of those two directions the brain or body or cells take. Is that correct? – Absolutely. And so so you know, again we we keep hearing the discussion as if this is a pathology.

Oh it's a dysfunction or you're making amyloid for some reason we better get rid of that amyloid. You're making towel? Let's get rid of that towel. No you have to back up and say why is the brain, your body's not trying to kill you? It's trying to heal.

So why is it responding that way? And there's a lot not known about this. Now, when you have pathogens within your brain, you make amyloid because it kills them and surrounds them. So it isolates these pathogens.

And what are they finding? We're finding. P gingivalis, the neuropathologist, have been showing us for years. You find, for example, pathogens from your mouth, P gingivalis within your brain.

Herpes simplex from your lip, within your brain. Of course, various fungi um that can be coming in through sinuses. You can also have things like spiral Keats that can come either from the mouth or can come from systemic circulation. Tick borne illnesses are associated with cognitive decline. But then as you mentioned, you're always you're also making this judgment. So when you have sleep apnea, you don't have enough support for your brain. It's got to go into that protective downsizing mode. And we see this all the time with people who have low oxygenation at night. Beautiful Study published a few years ago showing that just tracking the average spo2 at night correlates beautifully with your hippocampal volume. So all these things you've got to have that support and I should coming back to what you said about the choice.

There are four major components groups of things that will trigger you to go toward the protective side or the growth side and they are enough energetic mitochondrial function, oxygenation, blood flow, huge blood flow is critical here and and ketosis.

The ability to burn a substrate. Second thing is trophic support. You need that BDNF if you're going to be making synapses, things like hormones and nutrients, you need the estradiol, the vitamin D. The you know the androgen, all these things and then the third thing is inflammations or anything that's inflammation is going to be a problem is gonna push you toward the protective side all these different organisms for example. And then the final thing is gonna be uh in the various toxins and those are you know in organics, organics and biotoxins.

So those are the big four. – So this is this is great. Can we take this down to a behavioral level in terms of how we translate what you just said into the optimal behavior and thinking patterns and things of this nature that may influence those four different categories.

– Yeah, that's such a good point. And in fact behavior is extremely important in cognitive decline and in fact most of the Could prevent this. You know, my argument is that Alzheimer's is is really now optional if you if everybody gets on appropriate prevention or earliest treatment we really shouldn't have a lot of Alzheimer's currently 15% of the population dies of Alzheimer's.

So you know, the the nice study by Professor Christine for a few years ago who did, serial autopsies showed that it was the third leading cause of death in the United States. Not to be fair.

That was just pre pre pandemic. So it is a very common cause of death and it actually dwarfs the COVID-19 pandemic, probably 45-1. In terms of the currently living Americans who will die of Alzheimer's disease. So as you indicated behavior is huge stress.

One of the most important factors. If you are undergoing lots and lots of stress, you are first of all going to shrink your brain as you know, second of all you're going to affect your immune system.

And a lot of Alzheimer's interestingly is about a mismatch between the innate immune system and the adaptive immune system. So just again as in covid you die of cytokine storm because you've got this innate system that's just cranked up and the adaptive system has not cleared the virus in Alzheimer's it's slower.

You die of cytokine drizzle, It's years and years of mild activation of the innate system without a clearance because you have continued exposure to these various things.

Brain Protection vs Growth Signals

So no question about it, behavior is critical and of course, behavior on the side of compliance, getting the right things, doing the right things to address these different areas, which is why, you know, health coaches have emerged as such an important part of our health care system? – You mentioned two different kinds of immune responses.

Can you explain that to our audience? – Yeah. This is a great point and it's so important because these are fundamentally important to many illnesses starting with Covid 19 and Alzheimer's disease. So when you first have a response um to to a new pathogen, a new challenge um you have a non specific response to in other words, you just sense that there's danger. These are the so called pants or pathogen associated molecular patterns. Um and so these are typically through the you know the toll like receptors and things like that.

So you're now your body is alerted. Something is wrong but it hasn't you know, it hasn't characterized the you know the bad actor yet. So it's like the police saying, okay everyone just going at your house early at night, you know you want to have a curfew, you want to lock your doors, it's a general signal and that's the innate immune system.

It's the older part of the immune system. And interestingly that turns out to have a very interesting form of memory. So once you've been inflamed, you're at increased risk for future inflammation which is a really critical piece here. But then the in a perfect world what happens and normally what should happen is now your adaptive system, your T cells, your B cells you're now going to recognize and characterize and present the antigens from this now, you know aha, we know who the bad guy is.

You're going to develop a beautiful response with antibodies and t cell activation and part of that development is to turn off the innate response so that you have a beautiful system where you get inflammation, you turn off the inflammation and now you have a very nice adaptive response. And then of course you have a long term memory so that if you ever get exposed to that again now the problem is for reasons that are critical for path a physiology in Alzheimer's disease, you're not completing that loop. And there actually is there are fag acidic defects in Alzheimer's disease things we don't quite understand.

We also know just poor nutrition, poor resilience, poor overall health is increasing your risk for Alzheimer's. And part of that is because you're not able to get rid of it. If you're if you're continuing to be expe opposed to changes in your oral microbiome, you've got this in your brain if you're continuing to be exposed to pathogens to any to air pollution, to things that are inflammatory ins you're never getting to the point of turning that thing off and responding completely. And so that's what happens now in covid it's really interesting because the virus itself prevents you from having an early cytokine response. That's part of its M. O.

And therefore you have suddenly have this burst where you finally recognize oh my God I've been overrun by Covid you have this massive cytokine storm which as you know, can be lethal. – You know Dale.

You mentioned that Alzheimer's is optional. That's that's quite a bold statement. Can you elaborate on that

statement please? – Yeah. And when I say that, I'm not saying for 100% of people I'm saying for the fact that we currently have about six million Americans who have Alzheimer's. And what I'm saying is for the upcoming generations, they don't have to deal with this. They don't have to allow this to happen because we know so much more now about what can increase your risk, how we can prevent decline, how we can reduce risk and how we can prevent you from develop Alzheimer's and then if you just begin to get it we can treat things early on.

So when you get Alzheimer's you go through four phases, everyone is focused on treating the last two. If we simply move to the first two you can basically prevent this in everybody. So first, as you know you go through a phase where you're asymptomatic.

Already your pet scan can be abnormal 20 years earlier than you get a diagnosis of dementia due to Alzheimer's but you have changes in your spinal fluid for example and of course there are multiple new markers that are coming out. Looking at foss photo in the in the blood.

Looking at a beta 42 to 40 ratios. Looking at epigenetic changes all of these So we can look at this better and better. Second phase of Alzheimer's is called ci subjective cognitive impairment And unfortunately our doctors always tell us, yeah this is just normal aging.

You know you're just going through normal aging. You're getting a little older. I had a guy contact me the other day who has relatively late stage Alzheimer's and was told by his doctor that it's just normal aging. I mean we we just have to get away from this.

You shouldn't be losing all these things just because you're 45, 50, 55, 60, 65, 70. You know there are sharp 100 year old. So that's S. C. I. Now S. C. I.

The epidemiologists have shown us last about 10 years. So we have a tremendous window of opportunity to prevent people from getting to that final fourth stage which is dementia. – Yeah. Let me interrupt for a moment and ask you because from as a psychologist there are emotional psychological states that can mimic some of the things you're talking about.

Someone with trauma. They can get what we call brain freeze in which they're having cognitive difficulties. People who grow up in childhood environments that are dangerous.

They're on guard and their brains just don't function as well. People with anxiety, they're going to have cognitive some cognitive impairments at times as well. So it's interesting a lot of things can mimic the same kinds of symptoms. – And that's why it's critical again to determine what's actually driving the cognitive change. And in some people it's really easy.

You don't have to do a lot. And some people you do need to go all the way to a pet scan or to some of these newer tests to determine this. And so once you understand what's driving the problem then you have the best chance to deal with it. The problem has been that so many people have tried to treat Alzheimer's as one problem. Let's just give let's just get rid of that amyloid with an antibody and as we know it simply does not work.

So just to finish that previous part, the third phase of four is what we all call mild cognitive impairment. M. C. I.

Immune Responses and Disease Progression

And it really should be called relatively late stage Alzheimer's disease because it's like telling someone don't worry you only have mildly metastatic cancer.

It is a late stage of the problem. It's really too bad that the term M. C. I. Was used because it's really the third of these four stages. And then the final one where you begin to lose activities of daily living of course is what you is dementia.

The dementia phase of Alzheimer's path of physiology. And so if we simply get people in those first two stages get

them on prevention, get them on earliest reversal virtually 100% of these people do very very well.

And that's why I say Alzheimer's is now optional. If people will still simply get in in those first stages this is not gonna be a problem. And so we really need you know global programs just as we had for polio, just as we had for you know for for other situations like smallpox we need the same sort of program to reduce the global burden of dementia.

– Yeah we started the conversation today talking about how others have gotten the model wrong dale. Can you give us the model that that you work by? – Yeah so we again we spent 30 years in the laboratory studying the underlying molecular drivers of the neurodegenerative process. And what we found is that there are literally there are molecular signals that come from different areas.

So it can be you can get the same sort of phenomenon by having anything that is pro inflammatory. And one of my colleagues, by the way doctor alexei karaoke on also at U. C. L. A. And we've published a number of papers together points out that this is really if you look at the changes in Alzheimer's it really focuses you not only on this idea of the innate system over the adaptive system but even further on what's called trained immunity which is essentially the memory part of the innate system. And as his point was this typically affects the endothelial cells and there's you know you have a pro coagulant state, you have activation of micro glia. These are all the things we see in Alzheimer's disease. So his argument is it's really even more focused on that sort of thing. But there are many things that get you there.

And so again, our model which has to be internally consistent and has to be predictive of treatment. I mean you can throw out any model you like, it means nothing until its predictive of treatment you treat someone and say aha, this proves our model and that's what we're showing.

So our model is that also what we call Alzheimer's is a network insufficiency and this network is about neural plasticity. It has the four fundamental things that I mentioned earlier, inflammation affects it, etcetera, trophic activity.

And it's about your system trying to fight these various insults and we know that we get these, it fits beautifully with inflammation as we have some degree of inflammation and some degree of aging. Your brain is dealing with changes in hormones, changes in oxygenation, changes in air pollution, exposure, changes in mycotoxin, exposure, changes in microbiome, oral microbiome, sinus microbiome and even brain microbiome, you're dealing with all these things.

And so it is now having to go into this protective downsizing mode. As long as you don't discover what's driving it into that mode, it's just going to get smaller and smaller and smaller.

Your brain is essentially saying I can't exist with 500 trillion synapses. But I can put out all this amyloid, how these things are antimicrobial I can exist with 400 trillion synapses or something like that and unfortunately you just keep going and unfortunately get dementia and die if you can identify those things and address them appropriately create resilience and then regenerate things like stem cells or around things like inter nasal trophic factors and things are available.

So the armamentarium, which we have been told over the years is zero. There's nothing that will prevent delay or reverse cognitive decline is actually huge.

There's a tremendous amount and the earlier you do it, the easier it is to do. – Well I know you put your money where your mouth is because you just had a study published in the Journal of Alzheimer's Disease on your clinical trials.

Can you share with the audience something about that study and what you believe were the key ingredients to the success. – This is such a good point. Thank you for for asking that.

Why Alzheimeru2019s Is Potentially Preventable

So we were very fortunate to have support from the Four winds foundation to do a trial. Now we tried initially to do our first trial in 2011. It was turned down by multiple I. R. B. S because it's multifactorial multi modal trial and they kept saying, well you just have to have one change but that's not the way the brain works. This is not a linear system that's clear to pretty much everyone.

So we continued and we published some anecdotes just to show that yes there's enough reason to do a trial. Um we've actually published in 3800 people with documented improvements were finally given the green light in 2019 and the idea is to do two trials. We've done the first one and now we're just starting the second one. The first one is a proof of concept trial that uses historical controls and it flips the script.

So with all other previous trials you tell ahead of time, here's what we're gonna do. We're gonna give this drug or this change in behavior, whatever you're gonna do this instrumentation, whatever in this one we're saying for each person, we will identify the factors that are contributing to the decline and typically we find between 10 and 20 so people have insulin resistance or they have, you know, changes in their oral microbiome or sleep apnea or what have you were going to address each thing in each person. So this is a personalized precision medicine approach and we then finish this, published this, as you said in Journal of Alzheimer's disease just a couple of months ago and 84% of the people showed improvement in their cognition. Now, the important thing is here when you look at the successes with anti amyloid antibodies, what they they don't make you better.

They don't even keep you the same what they do at the best is when you're going downhill, they have a modest effect to slow the rapidity of the decline.

We're talking about something completely different. These people actually improved their cognition. And by the way, we have people who have been on this approach for 10.5 years and continued their improvement.

So we have people who sustained their improvement. So this was a fundamentally different approach, a precision medicine type of approach to people who have cognitive decline. And we did take people with both Ci and early dementia I'd like to do is do this now for the people with ci, because I think we can do even better with those.

But these people had a personalized approach which included things like brain training, which has been shown by Professor Mike Merzenich and his group to be so helpful and healing leaky gut and de optimizing their oral microbiome and reducing their stress.

Just what you were talking about early earlier and then fighting the specific pathogens that were identified. Some of them had specific pathogens that were undiagnosed, which we see so often.

And so those were treated and these people did very well. And by the way, you know, this this has worked for people who clearly have Alzheimer's. One of the questions is what about the people who had more of a vascular dementia pattern?

And so far this looks like the same approach may be useful for vascular dementia and Lewy body dementia as well. – So your approach is you're showing some nice results. Why do you think there's any push back to what you to what your approaches. – Yeah. So, you know, it pains me to say this, but we went through a period where big tobacco companies actually had an entire institute funded by them to show that there was no link between smoking and lung cancer. So as you know, I mean millions and millions and millions of dollars were spent on research to show of course, for greater profits.

Then we had the claim that sugar is not particularly bad for you. And again, money spent, Harvard Professor years ago was paid to say that it's really about fat, It's not about sugar and that has led to untold numbers of deaths. Unfortunately, now we have pharma that has spent now $40 billion on various failed trials trying to hang on to this idea that amyloid is the cause of Alzheimer's batman is a mob failed Chronos, a mob failed Gan Tenero mob failed on and on Selena's a mob failed.

Just go right down the list. None of these things has succeeded at a condom a fail in one trial, the other trial they're claiming, well, we think it had a modest effect. The best has ever that has ever been showed again, as I mentioned

earlier, is just a slight slowing of decline.

Precision Medicine Trial Results

Anyone who looks at this honestly for a few minutes, we'll see that amyloid is a mediator except for those few people with mutations in a P P itself where it really is, they're making amyloid inappropriately?

Uh, and by the way, we're working with some people there that are actually getting some some some good effects. But for the vast majority of us this is a sporadic problem, increased risk with a Po E four of course.

But this is a problem in which the amyloid is responding to something it is. Again it's an antimicrobial peptide. And so unfortunately when you have $40 billion here, there the Alzheimer's association was given $1.4 million Oh uh the the the company that that just paid us, they make a drug which we think is a good drug.

Well you know that that's kind of transparently inappropriate. So the bottom line here is that when you have huge numbers of dollars and you have a medical system that is resistant to change and that's the history of the medical system.

So I think of tobacco, sugar and and amyloid really in the same sentence, We have situations where there's a lot of dollars going into preventing change.

And so we've had no surprise a lot of pushback. Um And of course more should be looking at, can we do larger trials, Can we get other people involved. That's the way things are headed – Dale.

You touched on inflammation in the process, can you go a little bit more deeply into the role of inflammation please. – Yeah, this is a really good point.

And I and I think again people have gotten this wrong because we keep hearing, oh you just have to get rid of the inflammation. This is just an inflammatory problem.

Well that's a short term solution. What you want to do in the long run is get rid of what's causing the inflammation. And so there are multiple pieces to this.

So you want to know what's the organism or organisms? Um you know, are do you have a leaky gut um do you have chronic sinusitis? Again, a great place where you know, imaging is so critical to being you know, picking up things like chronic sinusitis. We do in the trial cone beams to look for undiagnosed abscesses that people have as well uh in their oral cavities.

So these things are all critical. And then looking at it's typically the chronic pathogens that are the ones and of course herpes simplex family and the herpes family as a whole. E B V, C M V, H S V, H H B 6A all of these things are critical. And then it's things as I mentioned earlier like P gingivalis and T dente cola that are coming from changes in your oral microbiome.

And then of course things coming from leaky gut, all of these things and you could argue that one of the most common reasons to have systemic inflammation is metabolic syndrome.

I just was reviewing these, we have a computer based algorithm looks at all these different pieces. And when I was doing this actually from a native American group where the diabetes type two diabetes is very high.

And you can see why when you look at the metabolic profiles, all of the ones that I reviewed had metabolic syndrome, relatively advanced metabolic syndrome and so this is another common reason.

You see that high Hscrp along with the abnormally of the Dislepidemia to the insulin resistance, the obesity. These things all go together and by the way very, very addressable clinically.

So all of these things are about in information and of course lots of beautiful work from Professor Charles Syrian at Harvard showing it's not just about an anti inflammatory.

You also have to look at resolution and he coined the term resolve ins So for these these resolving that he discovered that help you to resolve the ongoing inflammation.

So we do you know, we as a country have a horrible omega six to omega three ratio. Typically about 15 to 1. We should be closer to 2 to 1 or 1 to 1 or 4 to 1 of that. That range where our omega three's and omega sixes are more in a good ratio. But so many of us because of our diets because of the way we live in the exposures and all these sorts of things have these very poor ratios and we have low omega three indices as well.

So inflammation critical to resolve it. Critical to prevent it. Critical to understand where it's coming from and critical to remove it. – Great thank you for that answer. I'm curious what other diseases do you think uh would be amenable to this precision medicine approach of yours?

– Great point. And virtually all complex chronic illnesses should be amenable. So what we are now doing is something called the Ark project. So the arc of course Noah's Ark was two by two by two. So what we want to do then is take small numbers of patients with much larger data sets. So we want a few people who have Parkinson's Lewy body frontotemporal dementia A. L. S.

P. S. P. C. B. D. And the one we started with was dry macular degeneration. And we have some fantastic initial data showing improvements in people. And as you know what happens when you get, you know what happens when you get macular degeneration and it goes back to Dr. Lufkin's excellent book about. You know the lies that he taught his medical students. What do we teach people wait for macular degeneration and then inject your eye. What a barbaric thing to do to people who are developing macular degeneration. What we want to do is get into the earliest stages that's typically dry macular degeneration.

You're picking up the druze in your saying aha this person has an abnormality often at the beginning they don't even notice it. But already the first thing that's being that's changing typically is dark adaptation where normal dark adaptation is less than 6.5 minutes most of us are going to be 2.5, 3, 3.5, 4 minutes.

The first patient we dealt with with macular degeneration, 13.7 minutes to dark adaptation. He didn't even he was picked up with Drew's in by an optometrist and didn't know that. Yeah, you're on your way to having some significant problem here.

After one year on we adapted the profile to especially for the path of physiology of each of these things is different. He went he was at 5.38 for his dark adaptation completely normal. Now we'll see.

Can we keep that going for the next 20 years? We'll see.

Inflammation, Toxins, and Chronic Disease

But this is the way things are going to take the genetics and the biochemistry of each of these chronic conditions and target those with a personalized precision medicine sort of approach. And I think, you know, this has obviously been very popular in very effective in tumor biology and in treating oncology, treating people with cancer, adapting this. Now for each of these neurodegenerative and other complex chronic conditions I think will be very successful.

– That's great that you're looking at all these other illnesses with your approach. And it seems to me that one of the key takeaways is checking early enough diagnosing early enough noticing early enough because the earlier you do, the more that the intervention can benefit the individual.

– No question. You know what's interesting to me when we all, you know, went to medical school and learned about diseases We we saw and we were exposed to the success of 20th century medicine with acute illnesses.

You know, we're really good at pneumococcal pneumonia. Simple illnesses like that in the 21st century. It's not that

simple. These are now network diseases, it's multiple, it's systems biology now.

And the big problem, as you know, with complex chronic illness is that you don't develop the symptoms until a relatively late stage of the disease because your body is responding with things like atherosclerosis and amyloid plaques and tau and Drew's in and all these sorts of things. So, I remember being depressed when I was a medical student when I was told, Yeah, you get your first symptoms of renal failure when about 80% of your glamour, regular glamorous filtration rate as declined and you get your first symptoms of Parkinson's when about 80% of your dopamine ergic input to your striatum is lost. This is horrible.

So we need to develop approaches to look earlier, which is why I like the dark adaptation in macular degeneration. These are early changes that we can look at and these, all of these diseases should be treated earlier.

But of course our health care system has said we don't want to do prevention. We don't want to pay for it, We don't want to get ahead of the curve. We want to wait until you get really sick. It's just, it's again, eh, everything is backward because of the old fashioned notion that we just can't do things about these illnesses.

– And the same thing is true in the area of stress and dealing with stress. People, people, the stress processes a cumulative process. People really don't really deal with it, really motivated to deal with it until they have some serious problems, even though they noticed symptoms a lot, a lot earlier in the process. So we can say the same thing there.

– And I was gonna say, you know, this is where wearables are going to change for the generation after us. The wearables are going to change everything. So, you know, I was surprised when I got my Apple Watch a few years ago to look at my heart rate variability.

Great way to look at stress and wow, the difference between when I'm doing, you know, deep breathing and getting it to bump up to 1 21 30 and when I'm in the middle of some stressful Event and it's 17, you know, it's just a huge difference.

And I think that I actually think wearables are going to have a big impact on complex chronic illness. Look what we can do now, we can look at your heart rate variability, your sleep. I check my sleep parameters this morning, I check them every night. How much rem did I have?

How much deep sleep? How much superficial sleep? You can now look at this what's happened with the continuous glucose monitoring the C. G. M. S. Are changing the way we think about this this is the future. And so people will be looking saying you know what I don't have to wait until I get Alzheimer's.

I'm already seeing that something is wrong with my physiology. You know we can look at telomere length now we can look at your your microbiome. We can look at your oral microbiome before you get a lot of period on titus and get the p ginger palace into your brain. You can find that it's there and use some dental side and get rid of it. So there is so much that can be done.

And these early warning systems are going to be helpful. – Yes. Yes. So in my language we refer to that as greater awareness from a psychological perspective. Yes. – Absolutely.

So dale other than Alzheimer's what is going on in the research in the areas of longevity that excite you right now. – Yeah You know I'm very excited about the epigenetic ability to look at biological age. Now again its associations and of course I should I should point out a lot of this came from Professor Horvitz lab at U.

C. L. A. So another plug for an excellent professor at U.

Other Diseases and Early Detection

C. L. A. and Professor Horvath has done just brilliant work over the years looking at epigenetic association with age and now what's happening is there are there are different epigenetic associations with things like rapidity of brain

aging. There's a really interesting group out of Kentucky called true diagnostic Ryan Smith and that whole group they're doing really exciting work looking at different associations, different programs and I should say doing all sorts of collaborations with a number of medical schools.

So we can now actually say to someone and Dr. Kara Fitzgerald published about a year ago that here's some people where she can actually make them about three and three and all about 3.5 years younger biologically essentially just by doing the right sorts of things.

And so we're going to be able to see that what speeds this up, what slows this down. And then again association with other parameters such as brain aging. So I think that epi genetics and along with this being able to look at things like detox. One of the things I'm really excited about it when we see Alzheimer patients we lump them. So you've got P.

C. A. you know which is essentially post your cortical atrophy and by the way described by another U. C. L. A. Professor Dr. Frank Benson. So thank you the late great Dr. Benson and P. P.

A primary progressive aphasia. And then of course the classic kind of amnesty presentations. These are very different and yet we love them as Alzheimer's disease.

And so in fact the epigenetics may teach us a lot we've seen just as a clinical observation. The non am nested presentations tend to be associated with toxin exposure.

So we recommend anyone who sees someone with a non am nested presentation. Please dig deeply into the possibility of two toxin exposure or potentially some of these chronic pathogen exposure. But especially looking at mycotoxins and organics and in organics. So I'm hoping that the epigenetics are really going to tell us aha these are, you know, these have different profiles and that will be able to help guide us. Another thing that's coming up is the hyper coagulation states, partly because of this activation.

Abeyta binds complement, It is a pro coagulant and by the way, it also invests vessels not to cause them to leak, but to patch the vessels, it's a flock violent.

So I think we're being able to look at this disease in a very different way now. And looking at these things like epigenetics age is gonna tell us a lot about aging and about disease. So we're gonna have a just a raft of things.

Looking at aging. Now, being able to measure biological age. – You mentioned Ryan Smith and true diagnostics. They're actually one of our interviews in this program. I'm I'm currently in discussion with Ryan about doing some research to see if there's a correlation between biological age and my model of resilience, psychological resilience. So I'll let you know when we move along on on that as well.

This has been a wonderful conversation Dale. I really appreciate the time you're taking to to meet with me and to share your amazing information with our audience.

Wearables, Epigenetics, and Prevention

We've touched on a lot of things that people can do during the course of our conversation. Is there anything else you want to add to that about? You know what people can do to reduce their chances of getting Alzheimer's?

– Yes. So we've developed actually programs pre what one's called pre code prevention of cognitive decline and the other is called recode reversal of cognitive decline.

And I've published books on these. So you can look at the details as I mentioned as you mentioned earlier, we published an article recently but it's you know, it's detailed. So it's it's helpful to have someone to work with you like a health coach. We've trained over 2000 physicians now in 10 different countries and all over the US to do this.

And there are people you know all over the country. So you can certainly get that if you look just look at my

cognoscopy. We recommend everyone to get a cognoscopy If you're 45 years or older.

We all know to get a colonoscopy if you turn 50 but get a cognoscopy. As well. Simple to do And these are important things. The bottom line is we really can reduce the global burden of dementia if we all work together. So I encourage people please get a cognoscopy.

Please work with someone who knows what they're doing and let's make Alzheimer's a much less common disease. – Thank you for that. And finally how can people reach you? How can people learn more about what you're doing? What's your website or other ways that people can connect with this amazing uh source of information coming from you and from your group. – Yeah, I mean you can look me up on DrBredesen.com There's a facebook, we have instagram as well uh and and we're on twitter as well. So Dr. Bredesen and Dr.

Dale Bredesen for some of these so easy to do have books out as I mentioned, one called the end of Alzheimer's now available in 33 different languages.

So you can you know, the usual amazon and Barnes and noble and all that sort of stuff. Or look at our papers and the papers I should say are freely available online.

So again the tremendous amount to be done. Unfortunately in the United States, the average person with Alzheimer's spends $350,000 by the time he or she dies horrible to think that you can do much much, much better by getting started getting on prevention or early treatment so that you can save yourself, you know, vast amounts of heartache of dementia and and and not allow your family to go destitute. We can, you know, again, as a country we could do far better. – Thank you again.

I loved having our conversation. I look forward to the next time and thanks again and have a good day. – Thank you, Stephen. Take care. Bye bye bye.