Why Dementia Isn’t Inevitable—And What You Can Do Today Transcript

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Introduction and dementia epidemic overview

Hello everyone. My name is Doctor Richard Horowitz and I am the co-host of the Doctors Hawk Healing Lyme Summit 2.0. It's my great pleasure today to introduce to you, Doctor Dale Breslin.

Dale is one of the leading experts in the world on Alzheimer's and dementia. We are very lucky to have Dale with us today discussing updates. Since the last time Dale and I talked about the dementia epidemic we have going on.

We're live in tick borne disease. May be playing a part of it. So Dale, thank you for joining us today. Thanks so much, Richard. Always great to talk to you. Yeah.

So tell me where. Let's get some updates on kind of what's happening right now. With the US in the world with dementia, what kind of statistics are we looking at now at this point?

Really horrible ones. Unfortunately. So about 15% of our population dies from Alzheimer's, so it actually dwarfs the pandemic. So the projections currently are about 45, 46 million of the currently living Americans will die of Alzheimer's disease unless we do something about it.

And the great news is there are there's tremendous progress. There are new, more sensitive biomarkers. So you can literally find if you have, quote, pre old timers, just as you find if you have prediabetes, it's really fantastic.

So we can look years ahead even before symptoms and say okay you're on your way now. Of course everybody is so afraid of the A word nobody wants to say.

This is what you have. So fine, say that it's pre Alzheimer's, but that you're headed for this. Nope. Virtually nobody needs to get this. If they simply get checked, get it on early, either pre-symptomatic or early symptomatic.

As you know, you go through these four stages when you get Alzheimer's disease you can pick it up stage one or stage two. You don't need to wait for stage four, which is dementia, to get that.

Furthermore, there are protocols where we're, you know, of course we're looking and identifying all of the contributors. This disease is essentially a network insufficiency.

You're losing synapses, and you don't have the synapses that are functional, on board to do what you want to do, to remember, etc.. And so, of course, Lyme and chronic Lyme, other tick borne infections absolutely play a role.

The three big drivers are reduced energetic. So blood flow, io, vascular disease, sleep apnea, mitochondrial dysfunction, all of those things. Second one is anything that create a chronic innate immune system activation.

So it's an inflammatory situation over years just as Covid, kills you, with cytokine storm, Alzheimer's kills you with cytokine drizzle. It's long, long and over time.

And of course, chronic Lyme and other tick borne illnesses play a huge role in this.

Causes of Alzheimer's and brain dysbiosis

And we see it all the time. And then the third one, of course, is toxicity. Whether it's inorganic toxins like air pollution, well shown well-proven, whether it's, organics, things like, glyphosate and formaldehyde and toluene and benzene and all these sorts of things, or whether it is bio toxins, okra toxin A and Leo toxin and trachea theses and all these sorts of things.

These are the three big groups of contributors. So we have to ferret them out, identify them, treat them. And of course, this is where you the protocols you have developed have been so helpful for those people who have tick borne illnesses and other chronic infections.

Richard, you may have seen this recent paper, showing that if you look at the microbiome in the brain instead of the gut, we were always taught that it was supposed to be sterile.

You're not supposed to have organisms in your brain. In fact, your brain is full of them, surprisingly. And guess what? They are a most of them turn out to be oral.

Micro. But your oral microbiome, organisms, and it does include, things like, fungi as well. But but all these oral bacteria, are in your brain, and things like p gingival less and previously intermedia and F nuclei and all these sorts of things.

And this is in a normal brain, and then of course, you end up with these changes. It literally it is a dysbiosis of your brain that is associated. And there are specific nuclei like the locust psoriasis that actually have changes.

And specifically that one has, kutti bacterium Agnes, which is from the sinuses. So, you know, absolutely, as you said, these things are coming together.

And the problem has been we're always told there's nothing you can do that we don't know what causes it. I mean, that has changed dramatically. So do we do we know at this point, Bruce, because the thing about poor for most gingival is I mean, it was identified years ago with gum disease as a potential risk factor.

And so if everybody's got these, do we know why certain people at this point, that particular microbiome in their brain is actually causing neuroinflammation with microglial activation.

Why some get it. Is it the other infections, the other toxins, the energetic and immune system. Is it a combination of these factors? Why if we all have it.

And then also the question is should be working more in the oral microbiome, not just the microbiome of the gut, is that maybe one of the new frontiers we should be looking at here?

No question. Oral microbiome dentistry is playing an increasingly important role in cognitive decline from airway, from sleep apnea, from mercury, from gingivitis, from periodontitis.

Just what you were saying. No question. And as far as do we know why we don't? The speculation is that you're looking at a dysbiosis. And in fact, things like gingival actually makes the ginger pains.

These are specific proteases. And in fact inhibiting those has lead in in a trial to some slowing of decline. Didn't make people better, but it did slow in the people who had gingival in their mouths.

It slowed the decline. So there's no question that oral health is a very important part. You know, when I was training many, many years ago, we knew that the people who showed up with brain abscesses often had major oral pathology, but we had no idea that that oral health and oral microbiome was going to be important in the brain's microbiome, the brain's dysbiosis, and ultimately the brain's degeneration.

The other thing I just saw on it, I just read it this morning. I thought it was fascinating. Is there now finding apart from the microbiome, as you're talking about, that's now showing up in the brain that for long Covid, they're now finding the spike proteins in the in the brain bone marrow and in the meninges.

Right. Yep. You probably saw this also that just showed up that it's driving an inflammatory response. And then the question becomes well what do we do about this.

How do I mean this is of course is an unanswered question at this point. But if they've gotten up into the brain, they

used to think that none of the active virus got up.

And the article I just read was it was actually spike protein. So the question then becomes, you know, should we be even using regular, you know, messenger RNA where we get any spike proteins at this point, whether it's coming from the virus or not or using Novavax, one of these newer ones, that's mostly antibody, because the spike proteins themselves may be a problem.

And I don't think we have any good ways. I mean, they're looking at how to get rid of it, but nobody's figured out is it micro clots? Is it narrow kinase.

You know, everyone's looking at different ways of kind of addressing this. It's a great point. And you know Patrick anyway. Well Patrick has been working with a whole team to look at what's the best we can do about long Covid.

And I think he's done a really nice job. And of course they include natto kinase. As you mentioned. They include things like arteries LHP and other things.

So yes, you've got to address the blood vessels. And by the way, a lot of the people that we are seeing with cognitive decline, we will improve their decline.

They'll plateau and then they get Covid and they go right down again, no surprise. It's because you anything that gives you this chronic inflammatory state, be it Borrelia or Bartonella or Covid, will increase your risk and increase this ongoing inflammatory state.

And the problem, of course, is many people say, well, we just want an anti-inflammatory. You've got to go beyond that because that'll buy you some time.

But ultimately you've got to you've got to remove what is causing that inflammation, be it spike protein or Borrelia or what have you. So yeah, this is a tough one because there's no perfect answer when you say, well, if people are vaccinated, they won't have this problem till after Covid.

So it really, you know, I think in many ways the best is to keep your immune system, sharp, keep your immune system, optimize. And and that may be the best way to go.

As you indicated, you can have this spike protein hanging around for a long time. And, of course, there have been previous studies showing Covid in the gut hanging around for long periods of time.

Long COVID, spike protein, and overlapping inflammation

So one way or another, you really do have to address those three things. The micro clots, as you mentioned, which can damage heart and brain and can be an ongoing problem.

You've got to address the immunological changes and you've got to address, you know, the autoimmunity and all that, and you've got to also address, the virus itself. So, yeah.

You know, and and the problem we're seeing in our patients who have all of this and I want to from the studies you're doing, I want people to know kind of the you're doing some great studies on Alzheimer's.

The people that we're seeing at this point, I don't think people realize that in the American population and worldwide, when people get Borelli and Bartonella, it's affecting their B-cell.

So it's knocking out their immune system. Not everybody, but a large percentage. About 20% of our patients have low IgG with chronic variable immune deficiency, 85% have low subclass deficiencies.

Bartonella. That makes it even worse. And then they get long-covid on top of it where they get T-cell exhaustion. So now your B cells making antibodies aren't working, your T cells going after intracellular infections and cancer

isn't working.

And then they all come in with mycotoxins on top of it, as you mentioned, with glial toxins and the rest, which is also suppressing their immune system.

So how can we expect people if they're getting all these infections and in fact, in dementia, right. They're showing bacteria, viruses, parasites and fungi.

All of them are showing up in the brains of these people. And now we've got an, in a sense, an immunosuppressed population where the average doc is not even looking at this right.

Going forward. It's like, here's your vaccine. You'll be good with Covid. But we found that glutathione deficiency was one of the major reasons people were getting sick.

We published the first article on it in April 2020. I didn't know at the time the virus needed to replicate by lowering glutathione. And of course, you need glutathione is one of the major antioxidants to protect against all this free radical oxidative stress.

So we're in a particularly vulnerable situation now for people getting dementia. As we mentioned, with over 46,000,015%. When you can't fight these things that are now associated with dementia.

Exactly. So and I'm so glad you brought this up because I wanted to ask you about this. Here's a profile of a patient that I've been seeing again and again.

Number one, they have low immunoglobulins for no apparent reason. Why do they have low immunoglobulins. Number two. And then so that if someone says to you, you know what's what's going on here, we need to give you some IVIg.

Well why did you have that. The secondly they have low glutathione. And thirdly, they will often have a low white blood count. So they are immune suppressed and antioxidants suppressed.

And so a what's what is your recommendation in terms of evaluating these people who come in with low gamma globules? Because most of the docs really don't know.

Where do we go from here other than just giving you some? I mean, there's clearly primary immunodeficiency where there's genetic factors. But we published this last year that initially they were talking about Lyme causing immune deficiency years ago.

And we had found it because when Borrelia invades the lymph nodes, it knocks out the part of the lymph nodes that's making the IgG antibodies, which is why in a lot of the chronic Lyme patients, we had up to 45% of our chronic Lyme patients.

And the numbers from Johns Hopkins were exactly the same. It was 45% had CDC positive IgG m Western blot immuno blots positive, and it was an early disease.

It was because the IgG antibodies were knocked out and the body was only making IDM that most people think of as early. In this case, it's it was early in late.

So from what I've seen, it's a combination of the lime and Bartonella causing this chronic variable immune deficiency with subclass deficiencies. And then we're seeing people with T-cell exhaustion from Covid.

And and again, the more toxins getting in. The thing about the white cell count, that's interesting. And I only learned this a little while ago because I go searching whenever any of my patients have it.

You know, it's associated with mercury toxicity. We just found a patient who had chronically low white cell counts,

and we couldn't figure out why. And I tested it for mercury toxicity by serum.

And then six hour urine gMSA. It turned out that's what it was. And we started chelating the mercury. And the white cell count started coming up. So this goes back to what you were saying about the environmental toxins, the small particle pollution, less than, you know, 2.5 microns, getting in through the nasal passages, causing neuroinflammation.

It's this combination of some infections and toxins that's for us at the top of the list driving inflammation in this case neuroinflammation. And unless you get to the root causes of it, you're right.

I mean, you can try and block inflammation or give drugs for end stage disease, but giving things like aricept and acetylcholine, Ace inhibitors or namenda or some of the newer drugs that are causing brain shrinkage, even if you're removing amyloid burden, you're not stopping the main reason it's there.

It's like, I don't know how the health care system got to the point. We name a disease, we throw drugs at it, but we don't get to the root causes of why people are sick and you're seeing it, not seeing it.

And or, you know, various fields are overlapping with the same causes. Yeah. You know, the sad part is it has been a business solution, not a medical solution to throw these things like these, you know, $56,000 a year, antibody these, at this, when it doesn't help, so it it doesn't make people better and it does make their brain bleed, brains bleed and all that sort of stuff.

So, yeah, it's a such a good point because very many of these things are, are overlapping. And one of the things that I wanted to mention, one of the things that's come out of our research that I'm really excited about is that you can see, you know, the famous physicist.

Feynman said that nature uses only her longest threads to weave her patterns so that each, piece of the, of the, of the quilt reveals the organization of the entire tapestry.

And so you see this at every level. So we studied the molecular biology for years of AP amyloid precursor protein. You can drive it one way and make amyloid, and then it turns out that that's the protective Rob signaling pathway.

And you can drive it the opposite way. And that is the connective. So there's connection and protection. And this thing is it's not there to give you Alzheimer's.

It's there because in good times it supports you making synapses sap alpha which comes from this. And alpha ktph these things are there for synaptic plasticity.

And they help us to make new memories, to have neuroplasticity, the same system. When things are bad, you've got microbe changes, you've got mercury, you've got things like this now switches, it's cleaved at three different sites and now gives you four peptides that are all about protection.

And A-Beta is one of them. It's surrounds the various particles. It surrounds these microbes and kills them. And so it is an antimicrobial peptide. And so you go back and forth and then you see that at every level.

So what happens is you have a literally a system that is linked throughout your body that goes from connection signaling to protection signaling. And when you switch over, you have, as you know, more thrombosis because you get the micro clots, you're changing the endothelial cells.

You have less reload. Reload is what's allowing you to put out these fingers and make connections. And they say, oh, reelin somehow seems to be good for Alzheimer's.

Yes, real. And is part of the connection side. And these things are all linked. And you have what you have change in what's called hypo signaling, which is a lysosomal, a degradation of a-beta.

Immune suppression, toxins, and root causes

So now you don't degrade your a-beta you have a change in your in your app signaling, in your ApoE interaction with DNA, on and on and on. So you can just go right down the list.

You are literally just like you go from sleep to wakefulness. You go from connection to protection. And of course, part of that is limbic reprograming, vagal, a sympathetic overdrive.

You're in this mode of I'm under assault and I need to change the entire system to deal with that. It's just beautiful to see. And it tells us why all of these things like Borrelia and Bartonella and Cobit are, are giving you more Alzheimer's disease, right?

Yeah. So, you know, when when it was interesting when I was doing I'm in the process of writing a third science book for Simon and Schuster about why we're staying ill and the first chapters I did, just to take a look, to look at all these chronic disease was Alzheimer's.

I wanted to know whether all 16 factors on the emergence model I was looking at in Lyme apply to Alzheimer's. So I did, you know, an eye search, and we went through it.

Every one of them was there. And now I started looking at autism. Every one of them's there ALS every one of them is there. It's like Crohn's. It's like, hold on.

It's like it seems like we're missing the boat on these chronic diseases because as you're saying, that switch over from making synapses to kind of making beta amyloid to protect, if we could lower the load of all the things driving inflammation, infections, toxins, microbiome issues, we're seeing Marcel disorder from people with leaky gut.

Right. Which also is getting into the bloodstream, driving inflammation, sleep disorders, insulin resistance with glucose intolerance. All of these things are the factors driving inflammation.

But nobody goes back and says, I mean, you're looking at it and I'm looking at it. What happens if we start decreasing these factors one by one, lowering the load of all these primary factors driving inflammation with the secondary effects of inflammation, mitochondrial dysfunction, hormone dysregulation, Potts, dysautonomia affecting the autonomic.

No one's looking at the whole picture. You and I are looking at it because we see these people who are horribly sick. But I mean, I think it's a paradigm shift that we need to be looking at for Alzheimer's, for chronic Lyme, and for most of these chronic diseases, if we're ever going to stem the health care costs, and get people better and not be dealing with such a large percentage of people who are sick with these chronic, fatiguing musculoskeletal neuropsychiatric illnesses.

Is such a good point. And you know what is really interesting? This is where prions come in. And so, of course, I did my postdoctoral fellowship many years ago, with the discoverer of prions, Professor Stanley Prusa, who won the Nobel Prize in 1997 for his discovery of prions.

And these things seem to be biological signal amplifiers. So you're going one way. It's a switch, so it helps you to switch. You're going from the connection.

Oh, we gotta go over to protection. Well, so what that means then is when you address enough of these factors, just as you were saying. Now you flip back into the switch, now finally flips.

And we do see many people where you need to go through limbic retraining. You need to go through vagal stimulation to get you back into that mode of not being under assault.

Because these things are a real problem. And I should mention, you know, I'm, I'm a book coming out in March called The Ageless Brain. It's all about how do we all keep our brains sharp for our lifetimes?

I often start by asking people who wants to live to 140 because it's such a big issue now, I want to live longer, so a bunch of hands will go up and I'll say, and spend 70 of those years in a nursing home with dementia, you know, and go back down.

So what we really want to make sure that all of us has a brain span that is equal to our lifespan. And I mentioned your work, and I mentioned either the, the wonderful a documentary about chronic Lyme, and hear people just basically saying, well, we can just claim it doesn't exist so that we don't have to pay insurance.

I mean, it's just it's unconscionable. And yet it goes on. And then here comes a long, long Covid. And then it's like, well, wait a minute, maybe we kind of missed the boat on chronic Lyme here.

So there's no question that these things all play a role and that you're you're the organism is making this determination. Do I have to put a lot of my resources into protecting myself and not neuroplasticity and things like that.

And that's what it's doing, unfortunately. So if we can allow it, the luxury of putting resources into health and neuroplasticity and good function and good mood and good sleep, I mean, it makes all the difference in the world.

So, so the biggest factors, and by the way, great title for the book, The Ageless Brain, I love it. And and you're right about the aging, because my grandmother, who was 104, she was in a nursing home and she had dementia by the end of her life.

So she lived a long life. My grandfather was 99.5 and completely well, although he developed colon cancer and, and that was it. But I do have longevity. But it's true.

I mean, every one of my family who's lived long by the time they get to close to 100 years old, their cognition, you know, is shot at that point in time, except for my grandfather.

So the prevention stuff I know a lot of people know about Mediterranean diet and some of the good things. Why don't you share with people some of the most important factors?

We talked a little bit about it, about, you know, diet, making sure there's no insulin resistance, low carb, Mediterranean style exercise, getting sleep.

Can you go through that a little bit? The basics of good health and good brain health and also these biomarkers that you talked about just when we were beginning.

What might we want to do to look at to kind of evaluate our risk going forward? And saying, hey, I really need to do 180 degree turn around of how I'm kind of dealing with my health right now.

Great point. And so everyone who's 35 or over, should know their biomarkers, just like you would want to know your blood pressure or your hemoglobin A1.

C you should know your p tau 217 so there are three critical biomarkers. And there's a very, very sensitive way to get these. Now it's P 217 GFP and NFL.

All these give you three complementary pieces of information. P Tau 217 tells you whether you have that signaling that's pulling back the Alzheimer related signaling.

So it's a very much an Alzheimer specific signaling. What the tau is doing is the tau normally is is stabilizing your microtubules to allow you to make connections.

So again, when things are good, you're in connection mode. The tau is being used to stabilize the connections. When things are bad, you phosphorylate that tau which pops it off, allows you to collapse these.

And then this thing now turns into an anti-microbial protein. So this goes after these various microbes. And again you're switching from connection to protection.

So you want to know if you've got a bunch of that around that you're doing that you're doing as you can see you start to see this coming up. It tells you before you ever have symptoms so that you can do something about it.

The GFP tells you whether you have ongoing neuro inflammation, just what you were talking about earlier with neuro inflammation. And so from anything.

So it's complicated. But I'm sorry to spell that out for people. Yeah. So it GFP would be light. It's glial fiber Larry acidic protein. And so it's G like golf.

F like Frank a like apple, p like protein, GFP, and that actually is a it comes from the intermediate, tubules, in the glial cells. So what happens is when these astrocytes in your brain are happy, they're not making a lot of GFP when they're called out because, oh, the brain's under assault again.

They get larger and they start making a lot of this GFP. And you can actually pick this up in the bloodstream. And then final one is NFL neuro filament light.

And that is coming from neurons. So that that tells you whether you have neuronal damage from any cause.

Amyloid, prions, and the connection-to-protection switch

So by looking at the three together you can say, is this likely Alzheimer's? Is this likely frontotemporal or something else. Is this likely. So a non Alzheimer's cause of problems.

Some people will also add a fourth which is about a 42 to 40 ratio. That is another inflammatory marker. So those are those can really tell you and I recommend people start at 35 and get it every five years.

You only need it every five years. You can. It's plenty of time. When you hit 60. You can switch to two every two years just to make sure. I just checked my own about two weeks ago.

Happy to see that they were all in good shape, so I'm going to check them again in two years and we'll see how things go. Then. The other thing to know is what is my risk?

So that one tells you what is your status. The other one tells you what is your risk and what are the drivers. And we call that a Cognos copy the for the first one, we we actually have teamed up with the group that's doing the most sensitive one, which is neuro code, and we have something called brain scan.

So you can do get a brain scan. You can get actually get it directly. It's very easy to do that. With the the second piece we call this a Cognos copy.

Just like everyone knows. You get a call, ask me, what do I love? So I love the word I'm getting a colonoscopy. I love it. Maria Shriver told me don't ever use that term.

It's a terrible term. Cognos copy. But, I mean, it's easy for people to remember, you know, you need a colonoscopy. If you're over 35, you should get a Cognos copy.

And again, every five years. Fine. And that will tell you all these different drivers. So it'll tell you what about your methylation status. What about your inflammatory status?

What about your toxicity status? All the things that could hurt you in the future. You want to know. And that gives you a nice background. Unfortunately, as you mentioned earlier, most doctors, the standard of care in medicine is not to check any of this stuff and just to say, oh, you got Alzheimer's.

You know, nothing we can do. And, you know, I want to mention that while we're here, we've got some. Just when you actually do look at these things, just as you have these amazing cases of chronic Lyme reversals, we're seeing the same thing, with a reversal of cognitive decline.

Doctor Christine Burke has a remarkable patient, who came in with a Moca of seven out of 30. This is, you know, end stage Alzheimer's. And this guy is turned around and is doing better.

Great to see, a wonderful health coach, in New York City. Kerry Rutland, who's got a, an example of a person with a posterior cortical atrophy, who, which never there's no history of PCA being improved.

She's got dramatic and very, very well documented improvement. We've actually written that up for publication. Doctor? Doctor. Craig. Tonio. Down in Florida, who's getting fantastic results?

Has a patient. He's shown with a cortical basal degeneration. Again, I'm not aware of a single person who's turned around with that illness. He's got a beautiful result with that.

You know, we're seeing things that just haven't been seen before. So you mentioned, you know, what to do about it. So there's seven basics. And then there are two groups of specifics.

The seven basics are just what you said. You know plant rich mildly ketogenic diet exercise, sleep stress, brain training, detox and some targeted supplements.

And there we could you could spend an hour, ten hours talking about all sorts of supplements. And obviously, you know, many of them that you use with great efficacy.

Those are the basics. And in targeting the things that are actually causing the problem, that's the most important part, finding out what's causing. And then the two groups of specifics that you have to go after, are, pathogens and toxins, just as we were talking about earlier.

And the anything that gives you chronic infection and chronic inflammation, whether it's metabolic syndrome, whether it's leaky gut, chronic sinusitis, we had someone recently, with, Cryptococcus lorenzi.

I want a surprise as a sinusitis and treating. That definitely helped her as part of the overall protocol. So looking at these things and no question, tick borne illnesses are among the very top.

And as you mentioned, the problem is these things open the door for each other. It's amazing. It's like they were gentlemen opening the door for a lady.

You've got the tick borne illnesses, as the gentleman saying, come on, mycotoxins, just come right on in here and opening the door for these things to come in.

So now you and we see it all the time. People can no longer detox. So now they have high mercury. They have high mycotoxins and they have chronic Lyme or other tick borne illnesses.

Bartonella bees, er, like all the ones that you treat. This is a real problem. And so you kind of you're living in this suboptimal state and ultimately it's called a disease.

But for a long time you're living in a suboptimal state. So the majority of people that you're seeing here actually have this dementia illness. You're pretty much seeing exactly the same overlap that I'm saying in my chronic Lyme packages.

As far as the causes. But I guess the question I would have for you, because it's we were talking about infections and toxins leading the list. If it's the bacteria like Lyman Bard, but it's also viruses HSV one and two and herpes virus six seven, eight and the rest, parasites.

Like we see a lot of BBC of course. And fungi. How do you determine in someone I mean, I know how to determine active Lyme, but we don't always get pcrs that are positive for the virus.

We do find it especially more after long Covid where they have six reactivation than EBV. In fact, years ago they just had a study done on, you know, the Shingrix vaccine that if you can stop shingles reactivation, it's lowering dementia, right?

I mean, that was a really interesting one they had. But same thing with flu and flu vaccine. And by the way same thing with BCG. So oh so it's interesting.

One of the so that's a question for you. Why is it shingrix and flu vaccine and BCG all seem to lower dementia risk. On the other hand giving someone Covid boosters seems to be enhancing the risk.

So there's something we need to understand. There I and again we don't have the answer. My my question now that they found the spike proteins actually in the bone marrow of the skull and in the meninges.

Of course. My question is whether we should go back to Novavax, some type of antibody. Because vaccines save lives. There's not a question about it. It's just a question of is the spike protein a problem with no matter where you're getting it from?

Prevention basics and brain health biomarkers

Right from actually Covid itself, that we just don't have the answers, at this point. So what? And you're doing a workup for these people and you're, you're looking at all of this.

Are you essentially, it sounds like you're running through the same 16 MPs. It's fact who's pretty much I'm running through. So you're still finding at the top of your list of dementia causing factors.

It is in fact infections and toxins following by all these other inflammatory factors leading to mitochondrial dysfunction posits it's pretty much the same things we're finding. Correct.

So it's interesting. You could just call this chronic illness of Western society. And it would do it would encompass what you're doing, what we're doing and what others are doing.

Cardiovascular disease, cancers, all these things are chronic illnesses of Western societies related to what we do now. I think what's happening, of course, in our case, we're seeing changes in the brain.

You're seeing changes in you with arthritis and with other things and with brain fog as well. So for some reason, the ones that we're seeing are affecting cognition out of proportion to other things.

We do see a lot that will include arthritis. But it's surprising to me how many we see that have dementia with little else. They're actually doing pretty well in many other ways.

So I would say if you look at the factors that are driving the patients, we see the number one factor is metabolic disease. We see insulin resistance, glycol toxicity.

Pre diabetes is a common one. You know, the standard high CRP. Right. Write down, you know, right down the list here. They often have damage to the gut.

They they typically have insulin resistance. So they have a high homa er they have dyslipidemia. They may have some extra weight. They have often have hypertension, I mean, classic American, metabolic disease.

That is the number one contributor. And of course, this contributes to things like sleep apnea. Many of them end up having sleep apnea. And so when you start looking at these things, dissecting these pieces say, well, wait a minute.

You know, you've had sleep apnea for a while. No one's ever figured it out. No one's ever done anything about it. Oh, by the way, you also have this infection.

So that's the number one. But yes, on top of that then it is it's infections and toxins. So as I mentioned, the three big things. Anything that reduces the energetics to the brain, your brain is very is a glutton when it comes to energy.

It takes about 20% of the energy that your body is is producing about 20% of blood flow. You need that blood flow. You need that mitochondrial function.

You need that oxygenation. If you're dropping down into the 80s and 70s at night, you know you're not getting it. And then the second one is the inflammation we talked about.

And the third one is the toxicity that we talked about. And it is amazing because people will go along and we're dealing with someone, for example, right now who has Parkinson's.

And you, it turned out this person had massive, massive exposure to a micro toxin that is known to damage mitochondria. Well, he was already told, well, nobody knows what causes Parkinson's.

Will just give you some Parkinson's treatment. We'll just focus on the end stage here, the symptoms, without looking at what's driving this problem. So one of the things I wanted to ask you is for some of these people, it ends up that the micro toxin exposure is exogenous, but for many of them, it ends up that they've already got some in their gut or in their sinuses.

So the problem is you remove, you know, they can even move out of their home. It doesn't matter. They've got endogenous problems. So it's funny. You it's funny you say that because I just had a patient with this and in most of the cases.

So we're starting now more regularly to actually check the sinuses for biofilms and, and mold spores. The majority of the patients when I give them MHC alpha lipoic acid, glutathione, 500mg twice a day for mold, a little bit deeper, eight, 500 twice a day.

Give them, essentially, I use GI detox, but you could use many different clay charcoal. And if they have tri co thickens. I use, defibrillation with glucagon.

And we give this to the majority of people, and we see the toxins come down beautifully. I have this one guy, his, And it was coming down very nicely.

We checked him and we checked his home. Home was negative. And all of a sudden it shot back up and we checked his sinuses, and he was loaded with biofilms and mold and a spores.

And we actually had to give him a troconis, all with biofilm agents to lower it down. So you're bringing up a really important point. You it's not like you're living in a moldy house.

You are the moldy house. You you actually have the spores themselves. And again, it's not standard of care. Even I have been doing this for a while. I'm still learning, you know, from people like Neil, Nathan and Joe Krista, I'm still learning over time because even though it's not the majority, I am finding it now.

And now I'm starting to check everyone for nasal passages because we are seeing it. And of course, the closer it gets the nasal patches is the faster.

Obviously you're getting this up into right into the brain. But yes, it's an excellent point that that's exactly what we're seeing. Now for mitochondrial, you know, I take things like my toe are with nicotine high right beside and the rest goes, I saw the studies on it.

I know there's a lot on your list and an A and other things out there. Have you found a cocktail apart from things

like, you know, ATP 360, CoQ10, acetyl l-carnitine.

Might. And are these things. Do you have a cocktail you specifically like for mitochondrial regeneration that you've tested? In your patient population?

We don't I mean, we have so different people, we're using different things depending on what they're low. We have you know, it definitely. You know, your lithium a has been interesting because of its, Milo phage mechanism to turn these things over.

And interestingly, the person I just mentioned with it, with the, with the Parkinson's, your lithium eight, no question. He noticed an improvement. Of course we like PCU because you end up with more mitochondria.

That's another issue. There was a very interesting study of what goes wrong with mitochondria in Parkinson's, and the finding was surprising that you actually have a complex one leading the complex.

One is the one that's typically abnormal in Parkinson's. And you can just inhibit complex one with rotenone or paraquat or MTB. You get Parkinson's. So it's very clear that there is an intimate relationship between your complex one function of your mitochondria and the Parkinson's syndrome itself.

Because of the pesticide exposure that's been associated with it, then yes. Various pesticides. Yes. And so many. And degreasing agents like TCE is a big one.

Tri trichloroethylene, and and there's there's actually a nice book, out there. And that is, that goes through, all these things by Doctor Dorsey and his colleagues, prescription for ending Parkinson's, which I think is and then goes through all these different things.

So many people have been exposed. It's mostly with interestingly, with that, it's mostly these organic toxins that seem to come up again and again and again, things like TCE and and DDT and deal and Agent Orange.

All of these things have increased risk. So but what this study showed is that if you look at complex one, you see a couple things. Number one, somehow it is mis assembled so that there is, an evanescent functional species.

You don't have a stable, complex one for some reason. And then secondly, if you look at the members, the actual proteins within complex one, they have a very high degree of carbonyl modification indicating damage to these various, constituents so that you don't you, you lose that significant complex one activity.

And so therefore we want to have some we first of all, we want to remove what's causing that things like okra toxin A and TCE and all that stuff. But secondly we want to turn over the mitochondria, get the new complex one things like neuro lithium a but other than that the standards of nicotinamide ribose side B6, you know, all the cocu, all these things to try to rebuild these.

And I think there's going to be more and more, there's a nice the epi catechin is coming out. This is called Metal Catalyst. You may have seen this. This is a new one that's just coming out.

That, Rob Lustig originally had mentioned this, and it actually does. It looks quite promising. They're using it right now for people who actually have a genetic mitochondrial disorders, and it does seem to support better mitochondrial function.

So I think epi catechin is another one that looks quite promising. Now question. When you were discussing and for those of you just tuning in, and I'm talking to one of the world's experts on dementia, doctor Dale Bredesen, about having an absolutely fascinating conversation on dimension kind of neuroinflammation.

So it's misfolded proteins, right? That's causing some of this problem, in park. So the things you're describing, is it changing the misfolded proteins or is just stopping the production of them so that the brain has a chance to heal from them?

So each thing has a different mechanism. So in the case, in the case of the Milo catalyst, what it's what looks like it is doing, is essentially enhancing energy production.

It may not be driving. Well, we'll see. Is it actually driving better? You know, this is a know polyphenol related is is it actually driving less? No one's looked yet to see.

Is there less oxidative damage? As you pointed out, things like glutathione, sulforaphane, all these things very good for reducing such damage.

Pathogens, toxins, and mitochondrial support

But each of these things has a different mechanism. So the euro lithium is helping you to turn these over and generate new ones, which at least for some period of time until they get damaged, they are going to give you better performance.

And this is what was noted by the guys like, oh, wait a minute, I my performance is better. Interestingly, as you know, exercise alone has helped, Parkinson's patients more than almost anything else.

So there is something positive, whether it's the Bdnf that's increasing, whether it's the GDF that's increasing, whatever it is doing that seems to be helpful for people.

And exercising is causing new mitochondria to be produced all the time. It's helping with insulin resistance. Right? I mean, it's doing it's really helping all the mechanisms that we're talking about.

Absolutely. And then interestingly, methylene blue has a quite a different mechanism recognizing you've got essentially the an inhibition at complex one.

So you're not getting the electron transfer down to complex three complex four and ultimately making ATP. You can divert around that with methylene blue.

So it's an electron acceptor that allows you to bypass complex one. So the two together are actually helpful. Now one of the fascinating findings in this research was that the when you start having this problem with oxidative phosphorylation, because of the complex one, you actually now switch into a glycolysis mode.

And as long as you allow glycolysis to be ramped up, you actually do pretty well for a while. So what they showed is if you simply inhibit glycolysis a little bit, a normal mitochondrion doesn't care.

But these ones in the Parkinson's situation completely fell apart because they were depending so heavily on energy from glycolysis. So as anybody looked at, I know some of my colleagues are using this.

You know, I use a lot of high dose methylene blue to stop the side effects of daptone. But I know some of my colleagues are using like 5 to 8mg, like for me, you know, Pathak methylene blue.

Other studies now on that low dose methylene blue being used in things like Parkinson's as somebody's done the studies on it at this point. So there are there have been study there are ongoing studies that you probably know there was a methylene blue study earlier that didn't show improvement as a, you know, as a, monotherapy.

I have to say, the problem with all of these things is that, like, I like one devenant tie peptide for your nose. Well, they did it as a monotherapy. Every single one of these things that's been checked as a monotherapy has failed because you they need to recheck it as part of doing all the right things.

And this is where I is going to be so helpful that you can look at thousands and thousands and even millions of people and say, okay, what are the things that seem to be the most helpful?

But we're exiting this outdated, old fashioned idea that we should just be looking one thing at a time, one thing at a

time. Well, of course, this is like, you know, taking a car that's got ten things wrong with it and just checking one thing at a time that's not going to make the car work optimally.

Again, you got to do all the things, together. And, and in fact. You're bringing that up. So I'm working with the Garland Consulting to finally get a multicenter, placebo controlled, randomized trial on some combination therapy.

And as I've with you and I've talked about this, the number one symptom that gets better from dapt, some combination therapy. Number one is cognition.

And what we find though is affecting the cognition is never one thing. So when we're designing the study, I have to figure out by the way, I'm speaking to a statistician, whether the number one factor we're going to look at is going to either be fatigue or cognition, but we have to figure out the secondary factors of these of the message model, because I have to look at Bartonella and all these other tickborne infections and parasites and mold toxins and heavy metals and leaky gut and food sensitivities.

And Marcel, I have to look at all 16 factors, because it's like going into a doctor's office with 16 nails that your foot and doctor pulls a nail and says, come back and tell me how your foot pain is.

Everybody in medicine? If you do one thing like in your world or my world, it's never enough. Although there is sometimes like a few of the nails or the big ones like you pull the Lyme, you pull the bark, you pull the mold.

And even if you have some mitochondrial dysfunction, some low adrenals, you'll say, oh yeah, yeah, I know, definitely notice the difference. You just don't get a full healing.

But this, this idea of the one cause, one disease model or name the disease, throw a drug at it. It's it's got to be completely changed because at this point, about half of America has a chronic disease.

And as you early mentioned, it's it's about over 45 million Americans with preclinical dementia. And it's going to be more because of air pollution. Right.

And all the smoke that people are getting from the wildfires, this is only going to increase with long Covid, right? The oxidative stress to the brain is happening from all these different sources now that we never even had years ago.

Exactly. You know, it's interesting there been there have been, complaints. We had the 77 Nobel laureates who said that, this idea of having RFK running our HHS is a horrible idea, but I think no one has said, well, there's some shake up needed somewhere.

No matter who you like, who you don't like, whatever they are, I'm agnostic. Whatever is going to make things better. But you have to start by admitting that there are major problems with our health care system.

People aren't getting better. People have chronic illness. We have a massive bill. People are, you know, it's over $350 billion being spent every year because of Alzheimer's disease.

There are caregiver issues, caregiver, you know, trauma and stress. It is an absolute disaster. So whatever you believe, keeping the status quo is probably not the right way to go.

Oh, I'm 100% in agreement with you. And in fact, I've reached out to RFK privately, to let him know that when he basically, after January 20th comes in, he's inheriting a tickborne epidemic.

He's inhabiting an Alzheimer's epidemic. And the model that we're using, it needs to be change. We really need to go at this point to a multifactorial model of where inflammation is coming from with the downstream effects, and stop naming the disease and throwing drugs at it.

Look, I mean, Big Pharma has done some great things over the years. There's some wonderful drugs. I use them

myself all the time. But but they're the ones funding medical schools at this point, teaching doctors what to do.

And as much as we appreciate all the help of getting doctors through medical school, the model that they're using really, I would rather they put their money into chronic disease centers of prevention, you know, get people through prevention models doing what you talked about with exercise and with sleep and keeping down sugar, which is toxic to the entire body and dealing with infections.

Let's put the money into prevention and having people get money back on their taxes, right. If their hemoglobin A1, C is low and they're exercising and their Fitbit is telling me you're getting enough sleep instead of putting the money into the chronic diseases.

Right? It's it's kind of like we need a complete shift in the way we're doing medicine to prevent the dementia epidemic and the chronic tick borne epidemic that, in fact, what you're seeing and I'm seeing this completely overlapping.

Yeah. With what we now know, virtually nobody should be getting Alzheimer's. As crazy as that sounds. Start checking if you start when, as I mentioned earlier, the four phases.

You got the asymptomatic phase, you got the CI phase, subjective cognitive impairment where you can still score normally on testing, but you know, there's something wrong there that lasts on average ten years.

So the time for us to intervene in these first two is huge. The third is MCI. Mild cognitive impairment, which is one patient said is anything but mild.

Now you're scoring abnormally on tests. It's a relatively late stage. It's a little bit like telling someone you've only got mildly metastatic cancer.

It is a late stage of Alzheimer's. And then the fourth one, of course, is dementia. That's where you're having trouble taking care of yourself and things like that.

If we can get people to get checked, get these biomarkers that we talked about earlier, get on active prevention or early treatment, then virtually nobody needs to progress all the way to having dementia.

We should be able to make this a rare disease. And just as years ago, people said, you know, we got to do something about smallpox. We got to do something about syphilis, leprosy, polio, all these things.

They they had their heydays and they are now past scourges. These chronic illnesses should become past scourges. If we look early, we treat optimally and we look at the whole networks of these things.

And these can be eradicated mostly in the years to come. You know, you're giving a message of hope. And I do the same thing when I speak to patients, because we're finding we are reversing the vast majority of these symptoms when people are coming in, when we're getting to the underlying sources for where the inflammation is coming from, and we're addressing the downstream effects.

So, I mean, it's really a message of hope for people that we can't.

Combination therapy, trials, and hope for reversal

So I, you know, I hope maybe when we're both 120 years old and we have our ageless brain, you and I will be doing our, you know, our talk together. Okay.

Do you remember, Dale, when you and I talked back? Let's see how many years ago that would be about 50 years ago. Well, we had our talk about this. How are you doing there, Dale? You're still on your trend.

Who knows? Right? I mean, that would be fun, wouldn't it? Yeah. I'm. I'm 72 now, so I. I would love to see this,

when we're, you know, when we're a little older. So.

So here's my question for you. Let's say we take we have people coming in with cognitive decline. Would you treat all of them with DAP? Some. Would you include that for everybody.

Or if not, how would you single them out? And then how long would you typically be giving them DAP. So and for the people that are coming in with cognitive decline.

So you know, of course I have a specialized practice because people come to me after they've seen, you know, three dozen doctors and they failed the treatment.

So mine is a little bit different because I am seeing lime as a factor in all of these patients. Right, right, right. But but there's going to be what I, what I described to some people as you can think about it in terms of it's either lime themselves, you have lime in 16 overlapping factors driving inflammation or you have not lime since, which means you could have Bartonella without lime, or you could have herpes viruses in your brain without lime, with toxins.

And and no two patients will ever be the same. I mean, this is why precision medicine is needed, because, we're going to find some people that it's the viruses and the pesticides and the mitochondrial dysfunction causing problems, but others it's the viruses, pesticides, mitochondrial and Lyme and leaky gut.

The point being, we need a model to screen through, you know, the six overlapping causes of inflammation and downstream effects. Otherwise, you're not going to get to all the causes of why it's happening.

So, you know, to get back to adaptation, I mean, for me, it's been a miracle drug. And I've had people I have a woman in Arizona who just flew out to see me is she's in her 70s, and she never wanted to do a lot of the higher dose steps on therapies.

I put on one minute of cycling a day, just 50mg. Great penetration into the brain stopped neuroinflammation and 25mg of DAP zone, which in my world is like homeopathic.

Some. She stayed on it for 18 months, just swore that her memory got better and she came off and she noticed there was a slight decline, but she still noticed she was still better than she was from when we started a couple of years back.

And what's interesting is because snapshot does lower neuroinflammation. It's an interesting theory that, you know, I'm thinking for myself, like, because we know that microglial activation is one of the major parts of the brain, apart from astrocytes and everything else causing inflammation is low dose naltrexone.

You know, at 4.5mg with maybe a touch adaptation and a little melatonin and things that decrease neuroinflammation, right? That'd be something that's helpful for people.

But you bring up an interesting point with that zone. There may be a place for it, low dose, not necessarily high dose in people with neuroinflammation when they don't have Lyme.

But of course, we're going to need big randomized trials to be able to see where that falls. But I can tell you that it's an amazing drug for neuroinflammation and reversing cognition.

I hope, by the way, because of the talk we're having today and we're designing the randomized trial, I will be calling you because when we design the trial, I'm going to want to integrate these three biomarkers you talked about, right.

For Alzheimer's. We're definitely going to want to integrate these biomarkers. And I'm going to want your input because Aim and clinic is one of the people that are participating in the randomized trial apart from Jonathan apart from Mount Sinai.

So you know, I don't know that we'll be doing F-18, Pet scans and things, but we will be looking at these

biomarkers. So you and I should actually speak at another point about what you'd like to see in the trial, because we can definitely overlap, what you and I are doing.

There's no reason not to at this point because I think looking at those neuro inflammatory biomarkers before and after treatment, it will be absolutely fascinating to see what gap some combination therapy and addressing the variables does for people.

No question. And we know we're in the middle of a trial, as you know, right now at six different sites around the country, we did an interim analysis.

It's clearly positive. So things look good. I kind of wish we had included low dose DEP zone as part of the overall treatment for this, because I think that there's a real place for this.

We are included in mine so that whenever you wanted to get out of your trial, we'll put it into my randomized trial when we finally get this off the ground.

Very interesting. Well, happy days scale. I wish I have to tell you, you and I could be speaking. You are just fabulous. You're a great speaker. You speak so clearly about what's going on in the body.

I think people are just really this one. I think knocked it out of the park. And I really would encourage people to get your new book because this is something we all need, the ageless brain.

Right. So now. In March. Yes, it's coming. It's coming out in March. So this will be around the time that our Lyme summit is actually coming out. So this is perfect timing for this.

So for those of you who've tuned in, The Ageless Brain by Dale Bredesen. Absolutely. Please go get this book. Because what Dale has discovered and what I'm discovering in the message, we're finding overlapping causes, he's coming at it from another perspective.

But he has amazing research at this point of how to protect our brains, and I do. I just like you. I think there's hope for people. We do not have to live with these kind of chronic illnesses.

You're finding it in your field, I'm finding in mine, but they're clearly overlapping and we're really making some great progress. So thank you so much for taking the time today.

This has been wonderful. Always love to talk to you, Richard. Thank you so much. Great to talk to you and look forward to further discussions. Yes. So for those of you again have been tuning in, this is Doctor Richard Horowitz.

I'm co-host for the Doctor Talk Healing Lyme Summit 2.0. You've been listening to doctor Dale Renison, one of the leading world's experts on Alzheimer's and dementia.

Again, his book Coming Out Ageless Brain, and hopefully you'll be tuning in for another episode soon. Thank you for joining us.