Introduction to Chronic Illness and Inflammation

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We all know the patient's not gonna feel all that well. What I ended up discovering over time with this 16 point model is that all of these factors drive inflammation.

And we know inflammation is the number one cause of all chronic disease and in many of the acute diseases also. So where's the inflammation coming from?

Welcome back to the Think Well, Age Well podcast. I am your host, Dr. Heather Sandison. And today's guest is someone who has spent decades asking questions where many others weren't and helping patients who felt they had run out of answers, where there was nowhere else to turn.

He is the founder and medical director of the Hudson Valley Healing Arts Center. And over the course of his career, more than 13,000 patients from around the world have sought his expertise for complex chronic conditions that have fallen through the cracks of conventional medicine.

Many people know Dr. Horowitz as the author of the New York Times best-selling book, Why Can't I Get Better? This is often referred to in my circles as The Lime Bible.

His groundbreaking work led to the development of MSIDS model, the Multiple Systemic Infectious Disease Syndrome Framework. And this helps clinicians understand why chronic illness is rarely just caused by one thing.

And most recently, Dr. Horwitz has published a remarkable paper that we've been talking about in my circles extensively. And it's exploring the reversal of Alzheimer's biomarkers in a patient with chronic Lyme disease using his Dapstone combination therapy.

How Dr. Horowitz Became a Lyme Specialist

and raising important questions about the role of these infections in inflammation and immune dysfunction and, of course, cognitive decline as we age.

So, Dr. Horowitz, I am so delighted and privileged to have you here on the Think Well, Age Well podcast. Thank you for joining us today. Oh, thank you, Heather.

It's good to see you again. You as well. You have spent decades treating some of the most medically complex patients that exist in the world. Literally, they're fighting in from all over to see you.

And I'm curious, what originally drew you to Lyme disease? When so many physicians, you know, kind of throw up their hands and say, this is too complicated, I'll refer out.

What made you dig in? I wasn't planning on becoming a Lyme doctor. That's one of the jokes in my career is, it's not like I turned to my mother when I was young and went, Hey, guess what?

Ma, I want to be a lime doc when grow older. I just ended up, you know, moving. When I finished my internal medicine residency at Mount Sinai in New York city, ended moving up to the Hudson Valley, New york, which back in 1987, and I didn't know it at the time, was the largest Lymedemic area in the United States.

So it was simply. I'm a board certified internist, I am here to help people. I like to think of myself now as more of a

medical detective trying to figure out why people are sick.

It's actually what drew me to internal medicine. But when people were coming in with rashes back in the 1980s and early 90s, we didn't really know much about Lyme at that point.

So I started on a journey to find out a lot of these patients who kept relapsing off antibiotics, why they got sick, that led to a 40 plus year journey seeing over 13,000 chronically ill patients from all over the world.

And I really, I credit, you know, my sticking with this, truthfully with my spiritual teachers who I met in med school. I was studying with the Tibetan Buddhist Lamas when I at the University of Brussels and one of my teachers told me Lama Gendun right before I leaving.

What do you want me to know before going out into the World as a doctor? And he said, Richard, The most important thing is exchange yourself with others and do for them what you would want done for yourself.

He said, if you do this, everything will go well. It turns out of course, that every time these patients were coming in, they didn't have answers. I kept looking for answers and you know, at one point I discovered Babesia, this parasite that was keeping people ill, a woman in a wheelchair for five years who couldn't walk, all of a sudden got up and walked.

You know, after a few months of therapy, um, another woman who couldn't talk, she had no, She was aphasic. She had, no words coming out of her mouth. Went to mass general went to all these, nobody knew what was wrong.

They said it was migraines turned out. It was Bartonella another, you know. Intracelular bacterial infection. So. The MSIDS model that you described, you know, it kind of grew from the ground up as I was just searching for answers as to why these people became ill.

And now it turned out, I've discovered that the 16 point model applies to Alzheimer's, that applies autism to ADHD, in fact, to most chronic diseases on the planet.

That's the, basically objective of my new book, Ending Chronic Illness, is to point out that there's other ways of preventing, diagnosing, and treating chronic illness, not just naming the disease and throwing a drug at it.

Right, right. So I want to hear about that framework, but you are known as a Lime Doc. And so I wanna dig into this a little bit more. There's chronic Lyme and there's what you described, that rash, and I'd love for you to even tell us what stands for.

So there's the tick bite and a rash that comes with it, and that's that acute phase. And that is recognized in conventional circles, but this chronic phase is less recognized and harder to test for, certainly harder, to treat.

Can you just kind of give us the synopsis of that for a minute? Sure. So right now we're having a tick explosion in the United States. If you go online, you'll see that ER visits are up at least 25% for tick bites this year.

I mean, it's a massive amount of ticks that are out there, likely from climate change, from the warming of the environment. So it stands for erythema migrans.

You're right. The early Lyme rash, which half the time looks like a classic bullseye and half of time, looks more like spreading red rash like, a bacterial infection like cellulitis.

Lyme Disease, Chronic Symptoms, and Transmission

Some people have been mistaken for like Herpes zoster, getting shingles or getting a spider bite. Half the time it does not look like a classic bullseye, but what happens is about 75% of the cases, 80% are going to get better with doxycycline or amoxicillin.

But about 25% and more, if you look at the literature, it even goes up to 48%. We'll go on to develop a chronic form of Lyme disease, which I still refer to as chronic Lyne disease.

Um, but it's otherwise known as post-treatment Lymedisease syndrome. And so what this is basically is, is Borrelia burgdorferi, the agent of lyme, disease is the great imitator.

So these people come in with. drenching night sweats and day sweats if they have babesia, this malaria-like parasite that gets in quite often. They have a chronic fatiguing, musculoskeletal illness with migratory joint pain, migatory muscle pain migartory nerve pain tingling numbness burning stabbing sensations and they can have chest pain shortness of breath oftentimes memory concentration problems with word finding problems difficulty falling asleep waking up in the middle of the night Neuropsych symptoms, suddenly they're depressed or anxious.

They even develop things like OCD, bipolar disorder, schizophrenia. So Lyme is really the great imitator. And the problem is the CDC said there was at least 476,000 cases just a year or two ago.

Medicare rates are seven times higher. We're probably dealing with a couple of million people per year. And many of these people go on to chronic Lyme.

And now we know that, you know, according to what I've seen, a lot of those people who've developed what they call chronic fatigue syndrome, myalgia-concept myelitis, fibromyalgia, even some of the long COVID patients, it turned out it wasn't just like part pieces of virus, the spike proteins or active virus that was making the mill.

It was Lyne. it was Bartonella. All 16 MSIDS factors that we'll discuss in Lyme today apply to long COVID. I published it a couple of years ago in the journal Microorganisms.

So it's a chronic fatiguing musculoskeletal neuropsychiatric illness, very prevalent, great imitator. And as you said, the testing can be negative in a lot of these patients because they become immune suppressed.

Lyne and Bartonella. can suppress the immune system where you don't make antibodies. If you've taken antibiotics early on in the course, it'll abrogate your immune systems to not make anti-bodies.

I mean, then you get long COVID, your T cells get exhausted, and then get mold toxins like gliotoxins that suppress your system. And lo and behold, you got all these chronically ill patients coming in that are looking for answers.

You mentioned that rash, this erythema migrans rash and how it can look different. It's not always the classic bullseye rash. But also my understanding is some people will never get a rash or if they get to take bite on their scalp, they might not see the rash it might be hidden.

Also, my understanding is that it's not just ticks, right? There are other vectors, the other insects, spiders or fleas. Certainly with Bartonella, flea come up a lot, but there's, I think sometimes people pigeonhole it into I've never had a tick bite, so I couldn't have Lyme.

But there are ways that we can be exposed. Can you go into a little bit of that? Yeah, so mostly for Lyme disease, it's usually by a tick bite, but maternal fetal transmission from the mother to the child can happen.

Blood transfusions will not happen for Borrelia burgdorferi, But just about every other tick-borne disorder I could mention today, whether it is Ehrlichiosis or different tick borne viral infections, Babesiosis, Bartonella, they can be transmitted not only from mother-to-child but also by blood transfusion.

I mean, even Babesia can be transmitted by solid organ transplantation. But Lyme is mostly going to be seen from tick bites or maternal fetal transmission.

And that's tough because there we have seen parents who have transmitted congenitally LyME to their children. Oftentimes they're misdiagnosed. They're told they have autism spectrum disorder, but it's actually congential LyMe.

Bart, on the other hand, which you mentioned, Bart has a huge number of vectors, and Bartonella actually looks like Lyme. It similarly causes fatigue and headaches and joint pain, muscle pain.

Neuropathy, memory concentration problems. So it looks Lyne, but the vectors can be ticks. There's a Eurythpean species, Bartinella Bertilisi, transmitted by Ixodes ricinus ticks, it's still debated how much ticks are transmitting it here in the U. S., but fleas, cat scratches, cats bites, mosquitoes, spiders, kids, which are biting flies, even the triactome bugs, some of the newer vectors on the planet right now, transmiting Chagas disease, they can transmit Bartonella.

Bartronella is a big problem. It's oftentimes misdiagnosed, but it's a problem, especially in children that develop new onset of seizure disorders or neurocognitive issues.

So most of those vectors are mostly for Bart, not so much for Lyme disease. Got it. Okay. And I want to go into this IMSIDS model. So I wanted you to explain first what it is, and then why do you feel like it's such a big paradigm shift compared to, say, what you learned in medical school?

Yeah, so we learned in medical school, and you may have learned the same thing, but it was kind of the one cause, one disease model from Cox postulate or Pasteur's postulat that we learn.

And of course, this goes back to the late 1800s when there was a bacterial infection like strep causing glomerulonephritis or rheumatic fever. But what we find now in our chronically ill patients is there's never one thing wrong with them.

It's like going to a doctor with 16 nails in the foot telling the doctor you have foot pain. And if the Dr. doesn't find all the nails or finds one or two nails and says, Hey, come back in a month and tell me how you're feeling.

We all know the patient's not going feel all that well. So what I ended up discovering over time with the 16 point model. is that all of these factors drive inflammation.

And we know inflammation is the number one cause of all chronic disease and in many of the acute diseases also. So where's the inflammation coming from?

The MSIDS Model and Root Causes of Inflammation

So the first main six factors on the MSIDS model, which I call rivers of inflammation going into an ocean of information, is number 1 infections, Which includes a bacterial infections like Lyme disease, Bartonella, B, parasitic infections like babesiosis.

C, viral infections, like you can have long COVID and reactivate Epstein-Barr virus or herpesvirus 6. Or D, fungal infections. You may have a candida overgrowth in your gut or actually get fungus in you body.

As you know, they've even found it in the brains of Alzheimer's patients. So number one is infections and when you mix these infections with environmental toxins, like mold toxins in heavy metals.

I mean, there are ultimately hundreds and thousands of environmental toxins getting into everyone's bodies, but the ones I usually focus on that do a lot of the damage, and we can detox a land of other chemicals in detoxing mold, is mostly mold and heavy metal.

When you convicts infections and toxins like this, you get a massive amount of inflammation in the body. And then you combine it with microbiome abnormalities in that gut.

You've got the wrong type of bacteria. you don't have enough short chain fatty acid bacteria to lower inflammation. The fourth point is leaky gut with mast cell activation, where you'll get things like lipopolysaccharide that gets across the intestinal barrier, causes that blood brain barrier to get leakey.

Then you have inflammation of the brain. So points three and four is microbiome, leaky gut, mast cell. And then the fifth and the sixth are vitamin mineral deficiencies, because your body has to detox all these chemicals and deal with the inflammation from infections.

Number six, insomnia, where people just don't fall asleep or keep waking up in the middle of the night. That happens from Lyme, but even young thin women have come in with sleep apnea.

Men can have benign prostatic hypertrophy, women in menopause. There's a lot of reasons why people don' sleep. But the point being, When you combine these infections with toxins with microbiome issues, with leaky gut and mast cell activation, With vitamin mineral deficiencies and sleep disorders.

I mean, even in sleep, you know this with Alzheimer's, the glymphatics, You need those glymphatics to take the garbage out at night. Otherwise you get these pathological proteins of, P-tau and amyloid beta that accumulate.

Those are the six major factors on the MSITS model. And they have downstream effects from the inflammation. So for example, the mitochondria in your body have nothing protecting them from all this free radical oxidative stress.

In Alzheimer's, right, and what you've been looking at, we know mitochondrial dysfunction was one of the hypotheses, but where is it coming from? Right?

So infections and toxins can drive it, But we also see hormonal dysregulation. People come in with low adrenal function. low testosterone, women go into early menopause.

We get immune deficiency or autoimmune reactions because as your immune system is trying to fight Lyme Borrelia, you get a lot of autoimmunity in the body with anti-nuclear antibodies and rheumatoid factors and anti gangliosides.

So you combine this with deconditioning and liver issues and neuropsychological issues It's basically a lot of inflammation in people and Lyme, as I said, it's the great imitator because most of the patients who've come to see me over my 41 year clinical career, they were not all diagnosed with chronic Lyne.

They came in with a diagnosis of long COVID or chronic fatigue syndrome, myalgia concephalomyelitis, fibromyalgia. You know, some of the cases of dementia that have come in, in fact, weren't what I would call true Alzheimer's dementia.

It was Lyme induced neuroinflammation driving those biomarkers, which we'll talk about. So in this day and age, when someone comes in with an illness, I don't find anymore that it's one thing making them sick.

That model we learned in medical school is pretty much outdated. so, you know that, that's what we're finding is really needed to get these chronically ill patients better.

You know, when you have people frustrated with the conventional system and, you know it takes them a long time to get in. It's like the uphill for every ill, but that's what's covered by insurance.

How do you help patients navigate sort of this frustration of, that functional medicine, IMSIDS type framework is outside of what I can afford. And I don't mean to put that all on you, But it's something I struggle with.

We want to help as many people as possible with dementia, but it's not covered by insurance. And so how do you help people square that? Is there an empowering message that we can leave, that people can take away today around this?

I want take a moment to share something really exciting with you. This is something that's been a long time coming for us. At Solcery, our mission has always been to make dementia rare and optional.

To give people real tools, real answers, and real hope when it comes to brain health. But one of the biggest

barriers we've seen over the years is access.

So many people need this care. They're motivated, they're ready to do the work, but they simply haven't been able to afford it. And this is why I'm incredibly excited and proud to share that we are now participating in Medicare's Guide Program.

This means that for the first time, eligible patients can receive ongoing support for cognitive decline, Alzheimer's and dementia through Medicare, with significantly reduced out-of-pocket costs and support from caregivers.

We are not just opening the door, we are expanding our team to meet this need. We now have two new nurse practitioners trained in our functional root cause approach to brain health.

They're working alongside us with our teams to deliver this care in a way that's personalized, comprehensive, and deeply supportive. This is about reaching more people earlier and staying with them longer on their journey.

It's about supporting caregivers who have been carrying too much for too long. And it's about making sure that the cost is no longer a reason that someone doesn't get the care that they deserve.

If you or someone you love is experiencing memory loss or cognitive decline or has been diagnosed with Alzheimer's or dementia, I want you to know that there are options and that now there's more access than ever before.

You can learn more and see if you qualify by visiting salseri.com or calling 760-385-8683. This is just the beginning and I'm so honored and excited to be able to bring this announcement to you today.

So, fortunately for me, almost every protocol I have published in the medical literature over the years are generic oral drugs that are covered by insurance.

Amazing. Oh yeah. In fact, if someone comes in, I mean, probably drawing like 35 vials of blood on the first visit, but fortunately Quest LabCorp bio-reference, the local labs cover it.

People usually do not end up with bills because of the coding, right? If you're coding correctly most of Um, the coding covers it. They don't end up with big lab bills and Dapsone combination therapy, which I pioneered for the treatment of chronic Lyme.

It's all oral generic drugs. You go to your CVS pharmacy or whatever it's covered by insurance. What's not covered is. you know, the probiotics for the protocols or some of the folic acid supplements I'm using or using glutathione to help protect you from Hertzheimer reactions.

But honestly, when you consider that most of these people have been to 10, 20, 30, 40 doctors before seeing me, I have developed these protocols in a way that they're short, they are fast.

I get people better and they're out of the office so that we can get new people in. But it is mostly covered. Your correct, for example, a mold test from real-time laboratories can be a few hundred dollars.

It won't be covered, but what I find is if we give the code for mold, once the test comes back positive, and they apply for their insurance. A lot of times they will get money back, but you don't always get it upfront.

But fortunately, the way I've developed these protocols, most of the things are actually covered. So, you're correct in the functional medicine space, of course, there can be a lot tests that aren't.

The heavy metal test we've used most are in serum, which won't cover it. Even the six hour urine DMSA challenge from doctors data was 60, 70 bucks at the time.

It was not a highly expensive test. I'd say probably the biggest issue is if you're not a Medicare patient, getting

some of the Lyme testing, like we can get it through the local labs again, ELISA, western blots, all of their regular tick-borne infections are covered again through local lab.

Ehrlichia, anaplasma, babesia. Bartonella, Rocky Mountain spotted fever, Q fever. The covered. But the problem is there's so many strains of Lyme disease.

We usually have to use specialized labs like hygienics. Again, they will discount them, but you'll probably pay about $300 to get an IgM and an IGG immunoblot.

It's not nothing, but it's thousands and thousands of dollars. So when I have to establish the diagnosis, I had a questionnaire on my website, cangetbetter.com cost no money to fill it out.

And it gives people a pre-test probability of what is the likelihood you're actually going to have Lyme disease. Um, and if you score over 63 on this questionnaire at cangetbetter.com, it tells you, Hey, I really should get tested.

Now you can get testing through the local labs, but if it comes back negative, you then really need to do some of the specialty testing because a lot of times we need.

Fish testing, which is fluorescent in situ hybridization. It's an RNA test. very useful for strains of Babesia and Bartonella. But if you just start with an immunoblot from iGenX, or even look at the bands on a Western blot through a local lab, there's a game I play with patients called Lime Bingo, which is if one of these numbers on the Western Blot or an Bingo, you've been exposed to Lyme disease.

And those numbers, people can take notes on this, is the 23 band, 23 is outer surface protein C, the 31, outer-surface protein A, 34, out-of- surface-protein B, 39, very specific band for Lyne, and the 83 slash 93. Now, a local Western blot, if there's a 31 could theoretically be due to Epstein-Barr and autoimmunity, but in real life, It's, it's live like 99% of the time.

And 41 is super common. It comes up on absolutely everyone, but non-specific, right?

Making Testing and Treatment More Accessible

Yeah. So, so there's ways of getting through these tests without spending a fortune. Honestly, I find that most of them, the monies that people have spent is usually before they've ever come in to see me because they have been to so many doctors getting tests, trying to figure out what's wrong with them and never got to the source.

Yeah, and then not being able to work, right? Being a burden on their family potentially. The caregivers can't work. It gets very expensive, not just directly paying for medical costs, but it's all the opportunity costs.

I just want to take a minute to applaud you and thank you. for being aware of that piece of this and for making this as accessible as possible for people, because in these circles, it is challenging and people deserve good medicine.

And so thank you for finding the ways to make the labs accessible and to makes the treatments accessible, and creating that as part of your protocols.

I really think that that increases the impact and it's not easy, right? It's easier to just spend more money. Well, I recognized early on that we were in the middle of an epidemic of tick point disorders.

And now, of course, what's really interesting is now we're putting together the epidemic Alzheimer's disease and the Epidemic of Lyme disease, there is maybe, you know, a reason why there's a bridge between these two.

As you know, they've talked about in the medical literature and autopsy studies for years that they would find Borrelia burgdorferi Lyme in brains of Alzheimer's patients with biofilm, with amyloid beta, but it was always someone who had died in an autopcy study that had never been in a live human being.

So I recognized early on in this worldwide epidemic, I had to find a protocol that was not IV. that was oral, generic, that any person across the globe could take it.

And I published it in open access journals so that anyone could go read my literature. They don't have to come to see me and get this protocol. I've done medical detective substacks, which are also free to sign up, where I explain these protocols.

In my new book, Ending Chronic Illness, it'll be out this October. Congratulations. We have a section on the three B's, Borrelia, Bartonella, Babesia.

where I explain these protocols like a cookbook, that anyone can pick up this book and understand how to do these protocol, s that if God forbid I should not be around tomorrow, people would be able to take this and run with it to find answers for Lyme.

I mean, my wife was sick for 25 years. She's eight years in full remission since she did the double dose StaphZone protocol. That's an incredible personal testimony and story that's phenomenal.

Congratulations on your book. Thank you for making this an open book, like literally, it's a book about how to do this. I'm curious, as we shift the conversation into dementia and Alzheimer's and this clinical case that you published around the biomarker reversal, how often do you think that these infections are only one part of a larger puzzle when it comes to age-related memory loss?

So based on the factors, in the article that I recently published, this was just a month ago in April in The Journal of Alzheimer's Disease Reports, what I found is that just like I published for Lyme, that there were these 16 MSIDS factors making people ill.

And in 2024, I've published in journal Microorganisms, these sixteen MSIDs factors were associated with long COVID. Now I published in this journal that all 16 mcids factors are associated with Alzheimer's dementia.

Now, from my perspective, what do I think are going to be the most important factors? I do think it's multifactorial and, you know, You can tell me your take from having been in the field for so long, but I Do think It's probably a combination of bacteria like Lyme combined with viruses, whether it is herpes virus 6, 7, 8, or herpies virus 1, Or, EBV.

I DO think its a Combination of Bacteria and Viruses. that are probably along with these environmental toxins. Jaminerology was publishing years ago, they would find pesticides in the brains of people driving amyloid production.

Knowing the number of hundreds and thousands of toxins getting into people, and knowing how bad these infections are, And knowing that these environment toxins are completely destroying the microbiomes of guts, where we now don't have that short chain fatty acid bacteria to lower inflammation, I think those first six factors on the MSIDS model that I discussed with infections, toxins, microbiome issues, leaky gut, not getting to sleep adequately and vitamin minerals are probably at the top of the list.

But, you know, if you happen to be a type three diabetic with, people with high insulin spike, of course that's going to a major factor. if you're one of those people.

But even dysautonomia, right, was playing a role in some of these patients. Mitochondrial dysfunction is playing the role, but of course my take is, is that the infections and toxins that are causing the mitochondrial disfunction.

So, you know, with these anti-amyloid drugs, I applaud them for trying to use them, But it only lowers it in the Cochrane review that came out one week before my article got released.

It basically said they're not really changing the clinical course. They're lowering these biomarkers, they are slowing it down maybe, but we don't really have good answers and this is the first time.

By the way, when I submitted the article, I didn't realize this was the 1st time anyone had ever reversed p-tau-217 completely. I was completely ignorant of this when i submitted it and then I realized like, oh this actually a big deal because the anti-amyloid drugs were lowering it by 23% over 6 plus years.

I lowered it by 63% in nine weeks, but it's because in my patient, it was obviously that the p-tau was being driven. In fact, by Borrelia burgdorferi.

So I do think it is going to be multifactorial. I don't ever think in Alzheimer's dementia, you're going find one factor, But there might be one that is at the top of the list.

Like in, my patients, I treated her heavy metals. We treated here for Q fever. I indirectly treated her for BART. She had metabolic syndrome with an elevated hemoglobin A1c.

So she had other factors that could have caused memory loss. But we kind of addressed her B12 was borderline, but we addressed all these, right?

Lyme, Alzheimeru2019s Biomarkers, and the Dapsone Case

So ultimately, I think in her case, it was infections, I think if we were to do thousands, thousands of patients, and this is what I hope will happen, the neurologists out there, if they would have checked for all 16 MSIDS factors in their dementia cases, it may turn out that there's two or three of four factors that are the main ones driving it.

But my concern with the Lyme epidemic now showing a relationship with Alzheimer's epidemic, we have to make sure that Borrelia burgdorferi is not there, because then you may actually have a treatable cause.

We won't know until we do a multi-standard placebo-controlled randomized trial, the old standard of medicine. This is one case study. I can't go too far on what the implications are.

But knowing that in the 375 Dapsone cases I published over 10 years, the number one statistical effect was always memory, concentration, word finding.

It was 70% of the people said the No. 1 thing that got better from Dapzone. was memory concentration, and whether that's because it was an NLRP3 inflammasome inhibitor lowering inflammation in the brain, which, by the way, might really be a reason to think about repurposing this drug, even if you don't have Lyme disease, you know, maybe it's chlamydia pneumonia, right, that is driving it and maybe these same drugs will help.

We won't know until someone really takes the time to look at all 16 factors, use Dapsone independently from infections like Lyme as a repurposed drug to lower neuroinflammation and see what we have.

But the beauty of this at least gives hope, like atleast we had a new research. You know, I opened the door a little bit to get some light into a, you know to a field that really needed it.

Yeah. And it was a PTAL-217. That was one of the biomarkers that was reversed. It basically went from positive to negative, right, and reduced by, you said, 63%. That's phenomenal.

Were there other biomARKers you looked at? Was there an NFL or A-Beta ratios? Did you look at other things? So we did. So her p-tau 181 was normal. Her beta amyloid 42-40 ratio was normally, but what was fascinating about the case, and again, I learned all this while I'm going through the literature.

And the p tau 217 came down, her beta 42 40 ratio is improved. When I published the article, what it showed is. is that the Quest Labs has this really beautiful graphic, and you can see that graph is going up.

And you know, for the listeners who are listening, you don't want a low beta-42-40 amyloid ratio, it's the 42 protein that's this sticky form that is really causing a lot of problems.

You want it actually to go higher, that what happened in our patient. It actually showed from Quest Lab that there was less amyloid production showing up, as the p-tau 217 went down, but she had a normal neurofilament light, normal pta 181. Her initial beta amyloid 42-40 ratio was normal, But improved.

You could see it was actually getting better after we did the Dapsone nine-week therapy. And what was your reaction when you saw her labs come back? Well, I was jumping up and down for joy.

I mean, literally, a kid in a candy store going, because I didn't know it was going to work. You know, she was one of the few patients at the end of my 41 clinical career who had never done Dapsone.

And she'd been putting it off for 15 years. She had not taken any antibiotics for 15 years. So I was really excited because I really, you know, I recognize like, Oh my God, this is such a wonderful result.

But I didn't know the full implications until I started writing up the article for review and then realized that it had never been done. And that the, the Alzheimer's trailblazer study that they had done back, four years ago and in JAMA lowered it by 23% in six years, and I'm lowering it.

By 63% and nine weeks. It's like. Okay, you guys were trying to pull out amyloid, but maybe the reason it wasn't always working was because Borrelia burgdorferi or Chlamydia pneumonia or other bacteria were there.

Now, honestly, if I was a pharmaceutical company making these, I would want to do a parallel study to see how effective these drugs are once you start hitting Lyme.

It could be that if the amyloid and p-tau is due to mold or due the heavy metals or do to these other emsids factors, these drug may actually end up being much more effective than they are right now.

So that's a study that definitely needs to be done. Yeah, couldn't agree more. And I think some people will hear this and assume that we're saying Alzheimer's is caused by Lyme disease.

But I want to make sure that you're describing this, Dr. Bredesen talks about the BREDESEN 7. My framework is the six primary level drivers, causes of neurodegenerative disease.

And so, I think that we want to make sure that, we put all of this back into that framework. Lyme disease generally has been, underrepresented, particularly in conventional communities, and this chronic form, this longer form Lymedisease.

And what we're saying is that, yeah, that might be one of the primary drivers for some people of dementia, of neurodegenerative disease, because of role of infection triggering the inflammatory process.

So, okay, we're in agreement. Absolutely not. I mean, the literature says, if it's right, that maybe a quarter of the cases, somewhere between 25% or a little higher of what's now being diagnosed as Alzheimer's, because when I saw this patient with p-tel…

By the way, I had many patients before I finished clinical practice, probably half of them were showing up with these Alzheimer biomarkers. So the problem is, at the time, It's Borrelia and it's other things driving, you know, these pathological proteins driving your inflammation.

But then I realized if my patient had gone to a board-certified neurologist with a high PTAL-217, she would have been diagnosed with Alzheimer's, not Alzheimer due to Lyme disease, because the American Academy of Neurology doesn't even look for Borreli-Burgdorferi as a cause because it has all been autopsy studies, it's never been proven in a live human being.

So yes, I mean, clearly your framework and Dale's framework, and even mine, they're all, as you know, kind of synchronous. We're talking about the same thing.

It's only going to be a certain percentage of these cases, but it now means if you've been diagnosed with this, at least something you need to look at, because there might be potential solution.

Again, until we do a really good randomized multi-center placebo trial, we're just not going Right. And I think if hopefully all of our listeners here have heard this before, but amyloid and p-tau are antimicrobial, right?

They are there to protect us. Inflammation is also there amyloid can get stuck in the on position, p-towel can be stuck on the position and so we want to be able to turn those off.

But the way to try them off is not to just take them out, not just to reduce inflammation, just extract the p towel or just the amlyloids, but to ask the question of why?

Why is the brain needing to protect itself? why is there an inflammatory cascade being turned on in first place? And so you've found associations between all of these 16 MSIDS factors and Alzheimer's disease.

I think we've gone through six of them. And I want to make sure we go through the other 10. Then I'm curious, which are the most important ones in your mind, particularly when it comes to aging and age-related memory loss?

Mm-hmm. So the 10, so after the six rivers of inflammation, which again is infections, environmental toxins, microbiome issues with the wrong type of bacteria driving inflammation.

Leaky gut with mast cell activation, vitamin mineral deficiency, sleep. Those are the main six. The 10 downstream effects are mitochondrial dysfunction, which you know, mitochondria hypothesis and the amyloid hypothesis, and autoimmune.

There's all these hypotheses with Alzheimer's, but from my perspective, it's like, well, these infections and toxins and sleep disorders drive mitochondrials.

So that's number seven. Number eight is dysautonomia. People will get definitely POTS dys autonomia, we see immune dysregulation with immune dysfunction where some people have chronic variable immune deficiency, but we also see a lot of autoimmunity.

So immune and auto immunity are some of these factors. We see neuropsychological issues, so neurological symptoms, psychological symptoms. The liver, It turns out with these people who have non-alcoholic steatohepatitis, NASH, or fatty liver, one third, which is one-third of the world's population, they have metabolic syndrome and insulin resistance.

And we know that these blood sugar spikes with insulin will affect the brain, right? It's one of known causes of how you can get inflammation in the brains.

Then there's people that become deconditioned over time from their illness. Those are really the main factors, you know, in, the 10 downstream effects of, how this affects people.

And in my view, the biggest ones ultimately are going to be infections and toxins, microbiome sleep, as we talked about earlier. But that doesn't mean in an individual patient, like hormonal dysregulation, you get inflammation in the brain.

It affects the hypothalamic pituitary axis. and the hormones get dysregulated. And we know with low estrogen, you go into menopause, there's memory loss.

All of this may play a role, and in some people, it may be that the hormone are playing a big role as is dysautonomia, as his immune dysfunction autoimmunity.

I don't think you'll ever probably find two people that will be exactly the same, but for me, the broad brushstrokes. is that it's probably going to be, because in my world with chronic Lyme, when I treat the infections and toxins, that

usually is the biggest factors that get these people ill with Chronic Lyne.

And as I said, 70% of these who've taken Dapsone combination therapy for chronic lyme say memory, concentration, cognition. is my number one best effect of doing this drug regimen, and it's durable.

And that's why I think it probably is going to work in the Alzheimer's space is because I don't see people relapse from their cognitive issues when they've done Dapsone if the other MSEDS factors have been adequately treated.

You know, I hear the same thing from patients who have done the hyperthermia treatments. What are your thoughts about hyperthymia treatments for Lyme, for treating Borrelia?

What the Alzheimeru2019s Findings Mean Clinically

You know, it's an interesting hypothesis because they found early on that if you heated up the body temperature to at least 104 degrees for two hours or more, you could kill spirochetes.

So I think there's definitely a group of people who go to Germany who do hypothermia who will get some help. But here's my biggest problem. Lyme and Bartonella are what are called biofilm persistent bacteria.

So they go into hiding under these biofilms where your immune system can't recognize them and your antibiotics don't penetrate easily. And these persistent forms are a type of the bacteria that go in to dormancy.

So that's why when you stop ivirocephaline, I mean I've had patients take IV drugs for Lyme for a year, not with me, but with other doctors come to see me.

And within one month off the iviocefin, the symptoms are back. It's like, well, why didn't iviorecephin kill the bacteria? Because ivioricephin does not hit these biofilm persistiforms.

The way I designed the protocol is looking at the tuberculosis leprosy drugs, because TB and lepercy are biofilm persistobacteria. That's how I came up with the idea of doing this, cause they use rifampin and Dapsone to cure lepersy.

So it wasn't all that brilliant. All it was is, hey, I wonder what happens if I add a tetracycline to rifapin in Dapzone, Cause tetracyclines hit Ehrlichia, Anaplasma, Regensia and it turned out it, was a home run, you know, out of the park with this.

So I do think that hyperthermia has its place, but ultimately I've seen too many people relapse. So, I think what it does is it probably lowers the load because you will knock off a certain amount of the bacteria.

But ultimately, i don't know that there's any proof that hypothermia is affecting the biofilm persister forms, just like a lot of these alternative therapies like SOT therapy.

Some people, S. O. T therapy will shut down these bacteria for a while, It comes back later on, right? Because you're just putting kind of like a lock in the key to shut off the bacteria for a while.

But if you understand like TB, you need to use four drug regimens specifically for this type of bacteria. Don't you that Lyme and Bart are a similar type bacteria?

that, so I think these other therapies have a place, but from my perspective, not only are they highly expensive, you were talking about, what it costs to do some of these integrative therapies, SOT therapy can be $3,000 a pop, if you go to Germany, it's 15,00. Daps on therapy is nothing like that.

It's a generic old drug regimen. I mean, don't get me wrong, You might pay a thousand bucks for supplements and folic acid, But it is NOTHING like what you're going to pay for some these therapies and you've actually got a durable answer.

So, I do think there is a role for these things, but I just don't think it's getting to the source of where these bacteria are hiding in the body. Got it.

For listeners who have either a diagnosis of mild cognitive impairment or maybe they're APOE4 positive, they've got high risk family history, maybe even somebody with Alzheimer's disease and dementia, what practical lessons do you want them to take away from the paper you recently published and also from your work and its implications for Alzheimer?

So the main points for me is when I did this deep dive into the medical literature, again, discovering the 16 point model applies to chronic Lyme, it applies too long COVID, applies two Alzheimer's.

When I get a one year deep-dive into this book, what I discovered is the same 16 MSIDS factors was applying to ADHD, autism, Alzheimer's, allergies and asthma, the three B's.

C, cancer, cardiovascular disease, chronic fatigue syndrome, myalgia, fibromyalgia, hormonal dysregulation, immune disorders, autoimmune disorders including rheumatoid arthritis where the 16 factors were showing up in these patients.

Same for MS, multiple sclerosis. All 16 factor shows up as well as mood disorders. Depression, anxiety, OCD, psychosis, bipolar disorder and these viruses.

So it's kind of like, hold on. We're in the middle of a chronic disease epidemic where 60% of Americans have at least one chronic illness. 25% Americans, have two or more chronic illnesses.

Just this morning, a paper just came out that we are like one of the worst countries in world with our healthcare system. I don't know if you saw this.

It was just, came up this, morning that, you know, we're spending more money on healthcare than almost any country in, the world, and we not having great results.

So what is it about this chronic disease epidemic that can explain it? And again, the take home message for me is inflammation is coming from multiple sources.

For those people that have gotten viruses like COVID, which then caused long COVID. Yes, it may be spike proteins, but in our experience, the VGF, the vascular endothelial growth factor, which is a sign of endithelium inflammation, was also coming from Bartonella.

And we found that when we treated Lyme and Bart and affected the microbiome, they've shown in long COVID the wrong type of bacteria in the gut, bifidobacterium, people will get better.

So the take home message for me is, as we get older, I mean, just turned 70 years old. Now I feel great for my age and I'm doing a lot of things to block inflammation and open up anti-inflammatory pathways.

They call message for me is if you want to live a long, healthy life, you got to figure out where the inflammation's coming from. It's not enough to go to the doctor, check your blood pressure, your cholesterol, get a mammogram, a colonoscopy, all good.

But for people over 55 years old, if the NIH now said, which is on their website last year, 42% of Americans over the age of 55 are going to become demented.

It's like, what? Does not have to be. I mean, and nobody at the top of the healthcare chain is going, oh my God, four out of 10 people listening to this podcast are scheduled to become demented.

Like that is just not acceptable. So my take home message is look at 16 M SIDS factors. Again, they're not all going to appropriate for every person. But you want to make sure you're treating infections.

A lot of my patients didn't know they had mold when they came to see me. 90% of chronic glial patients now are testing positive for mold. Mold suppresses your immune system.

It can lead to cancer, right, with gliotoxins and aflatoxins, and trichothickines. These are hidden factors. So my first message is that MSID's model, I believe, to change, it's a paradigm shift for how we look at chronic disease medicine.

And the next step is, you know, if the NIH were to take randomized multi-center trials, take this model and apply it to all these chronic illnesses because we're not doing all that well right now with chronic illness in the United States.

And I think it's a starting point to say, okay, let's really great science and look at these 16 factors, right? Bobby Kennedy recently said, Let's get everybody off SSRIs and psych drugs.

But why are people all depressed and anxious, right? I mean, it might be social, what's going on in the United States and around the world, but it's also these infections and toxins.

It's all 16 emcees factors causing mood disorders. So that I think for me is really the major factor. And then for over 55, I would be starting to check people for Alzheimer's biomarkers and do like a mocha test, you know, do a simple cognitive test in your office and start to these biomarks because now we may have options instead of just anti amyloid drugs or improving the anti-amyloids drugs by treating some of these factors.

Yeah, I couldn't agree more. It's a really exciting time and it's the way to stay optimistic, right? In the face of these statistics around how much complex chronic disease there is and how many people are suffering, there are clear paths forward.

And so for anyone who is feeling overwhelmed or discouraged, because I think that that is a way to describe this path. So will you please share? I know it's not out yet, but I'm sure it is available for pre-order.

Where can people find it? What is it called? And when does it come out? So the book will be out in October. It's called Ending Chronic Illness, a revolutionary new program to reverse inflammation and heal persistent disease.

And what I did in the is I have sections on prevention and treatment, but it's also natural treatments. It's using nutraceuticals, it using vitamins and minerals for prevention.

Practical Takeaways and the New Book

Its looking at diet like the Mediterranean diet and how to adjust the mediterranean diet, which is so beneficial with exercise and sleep for helping to stop cognitive issues later in life.

So there's a whole section on natural methods but also pharmaceuticals. Like if you have migraines, I have no problems using a Jovi and, you know, some of these drugs out there, but maybe you don't need them.

Maybe, You know you, have a mitochondrial dysfunction in the brain and you Coke you 10 and B vitamins and. You need to be on a low histamine diet because that's how my wife stopped from migraine is just getting off histamines foods because she didn't realize she had mass cell activation.

So. What I do in the book is I go through all these major chronic diseases and explain how you can use the best of both worlds, the Best of Pharmaceuticals and what modern medicine has to offer, but also how to sometimes get yourself off these drugs and use natural methods to stay well.

Cause in my case, my whole family died of cancers of different types. I have no one left in family. The last survivor. And it's in part why, again, when I wrote the chapter on C is for cancer, cardiovascular disease, chronic fatigue, I wanted to make sure people know, because everybody knows someone getting cancer these days.

It's like, why is it happening? Infections, toxins, microbiome, it's the same issues. But if you go to your doctor,

they're not going to tell you to look at these things.

So it basically, the book will be out October 13 from Simon & Schuster. You can go endingchronicillness.com to go look the the Book in Pre-Order. And really what I hope will happen is people will go their doctors, give them even a copy of the book if they have not gotten it, let them read it because I believe it's going to be the paradigm shift.

I've seen so much chronic illness in my lifetime that we have to do better than what we're doing right now. And I didn't expect, by the way, when Simon and Schuster, you know, gave me a contract to write it.

Why didn' know that all these chronic diseases were going have all 16 factors. This was a complete surprise to me, just like when I did the search on Alzheimer's and discussed, that, all, 16 M SIDS factors are associated there.

So, It should get people hope and it should give them like new avenues of healing. And every story in the book are personal patients who've come to see me over my 41 years that I've gotten off Crohn's and ulcerative salitis drugs, or I reversed their MS because it wasn't true MS.

It was Lyme induced or Bartonella induced neuroinflammation with demyelination with mold. It describes in detail how to use the model. And I think it's going to benefit a lot of people and give people hope.

I'm sure that it will. Dr. Horowitz, thank you for being here today. But more importantly, Thank you, for the decades that you have spent advocating for patients with complex chronic illness.

And for, you know, not giving up, for continuing to ask the difficult questions that challenge conventional thinking so that we can find real solutions.

And I think, whether or not people agree with our conclusions, your work, it just reminds me over and over, and inspires me that like curiosity, rigorous investigation, compassion, humanity, And this willingness to look deeper are where the breakthroughs start and and how people really, again, get those outcomes, gets those solutions.

So thank you, thank for your work and thankyou for you time today. It's always a privilege to talk to you. That's my pleasure. Thank you for having me on today, really great pleasure for me too.

If you've learned anything from this podcast episode today with Dr. Horowitz, please share with others who might benefit. There are so much unnecessary suffering happening, whether it comes to Alzheimer's or other complex chronic diseases.

So please help us spread the word so that others can get the help that they deserve. Thank you so, much. Until next time, be well. If you enjoyed today's conversation, please take a moment to subscribe, leave a review, and share this episode with someone you care about.

It's one of the best ways to help others discover tools and inspiration for aging well. To stay connected, get bonus resources and never miss an episode, head over to drheathersanderson.com and join my email list.

Until next time, keep thinking well and aging on purpose.