Introduction to the Alzheimeru2019s Summit Interview

Hello and welcome to a very special interview for the Alzheimer's Summit. I am your summit co-host, Doctor Christine Burke. Over the last decade, I've helped many patients reverse their cognitive decline.

And in this summit, I am bringing you interviews with thought leaders who can help you understand how to prevent and reverse cognitive decline and Alzheimer's disease for yourself and your loved ones.

I cannot wait to introduce you to someone I have looked up to, and whose work I have followed and admired for years. One of our distinguished clinical luminaries in this field, Doctor Richard Horowitz.

Doctor Horowitz is a board certified internist and prior member of the HHS Tick Borne Disease Working Group and New York State Department of Health Tick-borne working Group.

As medical director of the Hudson Valley Healing Arts Center. He has treated over 30,000 chronic Lyme patients with classical and integrative approaches.

He has created an effective model to address those with resistant chronic illness called the M6 model and Dobson Combination Therapy, discussed in his bestselling book How Can I Get Better?

And Action Plan for Treating Resistant Lyme and Chronic Disease, and now expanded in his new book from Simon and Schuster, out later this year, Ending Chronic Illness.

Doctor Horowitz's new book discusses how all 16 factors underlying chronic Lyme disease and long Covid are also found in Alzheimer's disease, and he recently submitted an article to the Journal of Alzheimer's Disease Case Studies on reversing neuroinflammation and detail levels in a patient with chronic Lyme disease and post-treatment Lyme disease syndrome, using Dobson combination therapy.

Doctor Horowitz, I am so happy to have this opportunity to speak with you. Welcome. Yes. Thank you. I really appreciate you having me on the Alzheimer's Summit.

Very exciting. Very, very exciting. So today we're exploring one of the more emerging areas in Alzheimer's research how chronic infections can contribute to neuroinflammation and cognitive decline.

And I'll bet this is a connection that most people, at least most people outside of this summit, have never considered before. As you've said to me before, it really is paradigm shifting.

I'd love for you to help us connect how a disease like Lyme disease that most people think of as a rash and a joint pain, something you treat and move on from.

Like how common is Lyme exposure and how is that linking to infections as a contributor to Alzheimer's disease? Yeah. No, it's it's an important question.

So you know, the reason people need to pay attention to Lyme disease, which, by the way, is in epidemic proportion worldwide. BMC Global Health A couple of years ago, they reported that 14.5% of the global population, meaning one out of seven people walking around on the planet right now has been exposed to Lyme.

So the problem there is the testing for this disease, which we can go through, is not the most highly reliable in the world because there are so many different strains of this bacteria.

So if you are diagnosed with a cognitive problem, as you get older and they're calling it dementia, and in fact,

they're even calling it Alzheimer's dementia because they've done Pet scans, or they've shown you that there's beta amyloid, whatever that they're doing.

Most people will not normally think about the fact that Lyme disease can be one of the causes of Alzheimer's disease. So, as you said earlier, people need to put together that when you have beta amyloid.

And I think this is one of the things that escape people. Bacteria can cause amyloid production in the brain. Beta amyloid is not just showing up for no reason.

It's actually protective. It's shows up whether you have viral infections, bacterial infections, but it's actually protect your brain.

Lyme Disease and Alzheimeru2019s Risk

The problem is you create too much of it, just like then downstream with PTO, it starts creating inflammation in the brain because of these biological markers.

Lyme is definitely one of the causes of Alzheimer's. They've shown that the rates are 10 to 13 times higher. If you have Spy Kids. So they looked at Alzheimer's brain tissues and they found not just spyer Keith's like Lyme, but identical to Spy Kids from the mouth.

That's why you've got to go to the dentist and take care of gingivitis and poor Ramona's gingival and all those, you know, bugs that are there. But Lyme definitely has been associated.

And what's exciting, which I'm going to discuss with you today and this is brand new research. I just submitted an article to the Journal of Alzheimer's Disease case Studies, which was accepted with revisions, and I sent the revisions back yesterday.

We proved that we can reverse some of these Alzheimer's markers that are now showing up in the blood, like peto and beta amyloid, with a nine week oral antibiotic protocol.

This has never, ever been shown in the medical literature. This is very exciting because if you happen to be one of these people who is diagnosed with Alzheimer's and you have Lyme, this gives you hope.

Now again, we need a much larger study. This is a case study that I submitted. Right. So we can only go so far with, you know, how far we were going to make recommendations.

But still, as I started testing my line patients in the last year, I was finding detailed levels and beta amyloid ratios were off. And I don't think the Lyme community knows this.

This is something that doctors need to look at, and now we at least have one way we may be able to help reverse some of this neurocognitive decline. Yeah, it is really exciting.

And corroborating the paper that you've just submitted in our recent clinical trial, we also found a very high prevalence of these types of chronic infections and the Lyme and tick borne related infections in particular.

So you mentioned a moment ago that the testing is really variable. And as people who do this work, we certainly know that. But let's share a little bit with the audience about some of the challenges in the testing and how you recommend taking a stepwise approach.

Yeah. So the most important thing that people need to know about testing for Lyme disease is that it's a clinical diagnosis. You do not make the diagnosis based on a test.

Now, if you were to go on the CDC site or speak to the health departments, they want what's called a two tiered test. They want a positive Elisa. And if that Elyse is positive, they're going to do a Western blot.

The problem is that most of the Western blots are looking at one strain. So when when they first originally did this, the B 31 strain, which is a lab strain that goes back 34 years ago from the from the Dearborn conference, we now have multiple strains of Borrelia the like cousins of the original strain.

And if you do a western blot using one strain, you're obviously not going to pick up the other eight major strains. So we don't I don't ever do these days local Western blots, I use what's called an immuno blot from my genex, which was just approved by the FDA, by the way.

So the FDA now has approved a Lyme immuno blot. There is an IgG. So the way I do the clinical diagnosis is we have a validated screening questionnaire that we published back in 2017 and the International Journal of Medicine.

And I did this with Mariella Satara from the State University of New Paltz. My good friend, Doctor Phyllis Freeman, PhD researcher. And we looked at 1600 people who had a chronic, fatiguing musculoskeletal neuropsychiatric illness.

Right. They were diagnosed with line. But we also looked at normal people who said they didn't have it. We were able to create a questionnaire and get it statistically validated.

And you can find the questionnaire on my website can get Better icon. If you look under symptoms and questionnaire, you'll find it there and you can download it.

And the way this questionnaire works is if you score over 63 on this questionnaire, you have a very high probability of having Lyme. If you score 45 to 62, you have a moderately high probability between 25 and 42.

Mild probability below 25. It's not likely that you suffer from Lyme disease. But the key and this is really important with people with Lyme disease. They have migratory pain.

There are only seven diseases in medicine that cause migratory pain. So if you fill out this questionnaire and you have good and bad days when the symptoms are coming and going and you don't know why, like there's no reason and your joint pain, your muscle pain or your nerve pain, tingling, numbness, burning, stabbing, vibration senses they just move around your body for no good reason.

One day you've got it in your hands, and three days later it's in your shoulder. And four days later it's in your legs. It's moving around and you're thinking, I'm crazy, but you're not crazy.

That's what Lyme actually does. So if you are tired and you have migratory pain or migratory neuropathy with cognitive issues, then that migratory aspect tells you that Lyme is going to likely be one of the causes, because the other six causes of migratory pain are not generally seen in clinical practice.

It's things like lupus, hepatitis, Crohn's and ulcerative colitis, gynecologic righties, writer's disease, and acute rheumatic fever. Now, yes, in 1 in 1000 shot you may have these diseases, but generally when you go to a doctor with it, it's going to be Lyme.

So it's a clinical diagnosis. And the labs are to confirm the clinical diagnosis. So I like telling people to play a game called Lyme bingo.

Testing Challenges and Clinical Diagnosis of Lyme

And what this means is whether you do a Western blot or my preference in immuno blot from my Gen-X, if you have any one of the following bands that shows up on your immuno blot bingo, you have been exposed to a Borrelia species.

Those bands are 23. The outer surface protein C 31, the outer surface protein A 34, the outer surface protein B 39, and the 83 slash 93 band. You only need one band on an immuno blot with a high score on the Horowitz M6 questionnaire, which again you can find on my website, can't get better.com.

It tells you that you have been exposed to Borrelia. Now understand in my world, if you have cognitive issues and memory problems and you've been diagnosed with Alzheimer's even by F-18 Pet scans, the problem is, is that and this is what we just published in the medical literature, we found that all 16 factors that we normally see in chronic Lyme disease or post-treatment Lyme disease syndrome, meaning infections, bacterial, viral, parasitic, fungal, environmental toxins, microbiome issues with the gut, leaky gut and mast cell disorder.

Vitamin mineral deficiencies, sleep disorders, mitochondrial dysfunction, Potts disorder, Nami, a hormonal dysregulation. All of the things we found in Lyme have all been found in Alzheimer's disease, which means we now opening up like a whole nother avenue to hopefully get people better by saying hold on.

Toxins. Heavy metals are found oftentimes with Lyme. If you've not spoken to your doctor, if you have a Lyme diagnosis and diagnosed with a demented illness, some of these patients that we've treated, they're mold, they're metals, they're Lyme, they're Bartonella.

Their cognition gets better. In fact, some combination therapy, which I published in this article, it's number one effect is to improve cognition with memory concentration.

So it's it's really an exciting time. But keep in mind the tests are unreliable. And in my new book that will be coming out later this year called Ending Chronic Illness, I just got the book.

It just showed up from the publishers in Henley Chronic illness. It's exciting. In this book, I explain in great detail how to use these tests because you can have an immunofluorescence assay and Elisa AC6, Elisa a western blot and immuno blot.

There are a lot of indirect tests that you can use for Lyme and even direct tests like PCR nano trap, where you find the bait in the urine. Fish testing is one of my favorite, which is an RNA test.

We usually use it more for Bartonella and Berbizier, but the point being, don't rely on one set of tests. If you think you have Lyme, you've got to make sure that you know it's a clinical diagnosis.

Look for those bands on the Western blotter, immuno blot and also check for co-infections, because many times the Busia Bartonella are associated with Lyme and they will cause the same cognitive dysfunction that you see in many of these Lyme patients.

Yeah, those are all that is such a useful framework for people, because it really simplifies something that can be quite complicated in an application, and I appreciate that so much.

I'd add that, you know, I find often what I've been terming latent diagnosis so people that don't necessarily meet the clinical diagnostic criteria but have evidence of exposure on the immuno blot testing.

So we know that their immune system has been exposed to that bacteria, but they've never had the classical symptoms or the symptoms like you've described in the standardized questionnaire, which we use all the time in our practice.

So it's an interesting thing where I think more people have had exposure than we realize, because I don't know that everyone necessarily develops symptoms of disease.

What what is your thoughts on that? So what's an excellent question? I mean, I think the people who develop disease are usually number one, the ones whose immune systems have been affected.

So first of all, Lyman, Bartonella, both affect the immune system. We have many of our patients. We published this years ago in the journal Health Care in 2018, that about 20% of our patients have chronic variable immune deficiency.

They don't have the antibodies, immunoglobulins to fight infections, or 85% of them have subclass deficiencies,

again impacting the way they fight infections.

But then a lot of these people have had long Covid and they get T-cell exhaustion. So not only are there B-cells that make antibodies, but the T-cells that fight viruses and cancer have also been affected by some of these viruses.

So many of these patients, their immune system is affected. So if you get exposed to Lyme and your immune system's not working correctly, you are going to get sicker.

The other ones who generally get it are the ones that have had multiple tick bites, and the ones with multiple co-infections, and especially the ones with more toxins, because more toxins also suppress the immune system.

So if you're loaded with Clio toxins and aflatoxins and tricot thickens and as we'll run on all these toxins we're finding in people, it's similarly affects your immune system's ability to fight.

So the sickest people in our practice are usually the ones that are loaded with toxins. They don't just have Lyme, they have co-infections. They have leaky gut.

They have the wrong types of bacteria in their gut that don't make short chain fatty acids. So they've got inflammation and that affects their blood brain barrier.

And they start seeing a lot of inflammation affecting their cognition. So it's usually people that have multiple factors like multiple overlapping causes of inflammation and downstream effects.

But you're right. I mean, there are people who have a healthy immune system could get bitten by a tick. They might have very, very mild symptoms. But you have to be careful because some of these people, you know, they're a little tired or the little achy and they think, oh, it's the aches and pains of daily living the way the IDSA sometimes describes it.

And I'm not saying that's not possible, but in my world, these infections and toxins are getting into a large percentage of the population. And and now knowing that really Bergdorf, where I live, is associated with Alzheimer's disease and raises your risk of it, according to what I've read in the literature by tenfold and the statistics, because I just submitted this paper.

And I think people need to know this over 55 years old, the risk of you may have discussed this in the summit so far, but the risk of dementia is up to 42%.

I'm going to repeat that figure 4 to 2%. Now that's ridiculous. They know that the rates are doubling of Alzheimer's dementia. But the problem in medicine and you know, this, Kristine, is we label illnesses and we throw drugs at it.

So what you're going to label someone as a dementia case? Here's your namenda right. Here's your aricept instead of saying, hold on, this dementia case may have all 16 factors underlying it.

I need to go look with a Lyme is there, but Busia, Bartonella there are viruses reactivated like we see with long Covid. You've got to go through the 16 point message model to see.

But the risk is enormous for people to have cognitive challenges as they get older. So I think the model just needs to be shifted the way we're looking at it right now.

I totally agree with you. And I think even even taking that a step back, like we've talked about before, where we want to be looking for these things at a some level, not necessarily as deep a level as we need to when people are chronically ill, but we need to be looking at these factors before the symptoms develop, because it's so much easier to correct them before people have accumulated all 16 factors on the insides map.

And by the way, and this is where it gets tricky if you ask most doctors when they go, if someone comes for a

history and physical, which you and I have done over the years, you know, go for your colonoscopy, get your mammography, get your prostate exam, check your PSA, there's bake, check your cholesterol, diabetes, hemoglobin A1, C.

But what is not being done because they don't know how to use the markers is Peter 217 Peter 181. Neural filament light and beta amyloid ratios 4240. Now those four markers I just mentioned, which are now available to request in LabCorp.

In this past year, before I started moving over to just consultation medicine, I started checking my line patients, and I had 50 year old women who said, yeah, you know, I've got a word finding problem here and there.

And the Peter 217 marker was elevated in the blood.

Latent Exposure, Immune Dysfunction, and Risk Factors

Now, that is one of the most significant markers that you are going to go on and have a demented illness later on. And I will tell you, and I'm guesstimating because I have to go through the records.

But it's about 50% of the patients I was checking. Their beta amyloid ratios were off. I mean, just so you know, if you do these tests through your lab core, it's kind of a first a an elevated P 217 or 181 is not what you want, but a low beta amyloid ratio is what it's a little reversed when you look at the lab tests.

It is. But but just know we're finding these markers, these neuro markers. Right. That are implying you may go on to getting demented illness. And probably half of our patients.

Now this article that I just submitted, which should be published soon in the Journal of Alzheimer's Disease Case Studies, what it showed is in this woman.

And by the way, I very few of these patients, most of my patients have gone through some combination therapy, which we can discuss in a second. It's a nine week oral generic antibiotic protocol I developed.

It took me ten years tweaking this to get it tweaked for Lyme, where it's quite effective. At least 50% of the people will go into long term remission if they don't have the Busia Bartonella mold.

What we found in this woman and this is highly unusual. My practice Eliza positive C6 Eliza positive Western blot IgG am positive CDC, CDC, ICC, Western blot positive, CDC positive, I am immuno blot and a rash.

This woman had every marker of post-treatment Lyme disease syndrome. I could never have found a perfect case. And she was 15 years with these symptoms after being treated with doxy sight clean.

And she also had evidence of Q fever. Cox yellow Brunetti or licky of the Busia Bartonella. So she had multiple infections. 15 years later she's got joint pain.

Rheumatoid factors are elevated and she has peaked out to 17 levels high in her blood. And I said to her, listen, your grandmother had Alzheimer's disease.

Let's check your EPO levels. Turns out she was 33. She did not have the genetic variant going on to its house. And I said to her, listen, I don't yet have evidence that that some combination therapy will lower these.

But there has been research published by IBA, Shopee and colleagues that Lyme disease, Borrelia burgdorferi will be under biofilm. It creates amyloid in p tau in the brain in people with Alzheimer's and Parkinson's.

But these are brain tissues of autopsies. Never in vivo, never a live person study. We gave this woman the nine we Dobson protocol and we tested her markers three months later.

The rheumatoid factors, which at one point were 33 I think over 20 is high, completely reverse back to normal with less joint pain. Joint pain got better.

But what's amazing is the p tau 217 that was elevated at 0.33 reversed going down to 0.12 into the normal range after a nine week oral generic protocol.

This is never, ever been seen in the medical literature. Now, this is exciting because if roughly and we don't know the numbers, but if you read the literature, there are guesstimates that up to 25% of the Alzheimer's cases are due to expire.

Kids. Now that's a combination of Lyme disease, Borrelia, Bergdorf Rye, and dental call as far as kids from the mouth. But nevertheless, adoption protocol would still be treating this.

So we might have. And again, it's one case study. We need a multicenter, placebo controlled, randomized. Right. We know and I'm applying for these things so I can prove to the world we have answers.

But this is exciting because this is the first time it's ever been proven that we can lower down P2 17. And what I didn't know until going through the literature, when you lower P2 217 in the blood, one of these markers that you may go on to Alzheimer's dementia.

It doesn't mean you have lower P2 levels in your brain. It actually implies you have lower beta amyloid ratios. And that's what we saw in this patient.

Her beta amyloid ratios, all they they were normal. They improved after doing DAP shown meaning the load of beta amyloid went down after we did the nine week home protocol.

Now again, we need to prove this in a larger population. But this is very exciting for the the doctors out there who say, gee, I've got an Alzheimer's case.

Did I check for Lyme? Did I check for Bartonella? Did I check more? This gives us a new paradigm of kind of look at these diseases and maybe make headways so that we're not giving someone a life sentence.

Absolutely. And that is a dramatic and remarkable result that you just told us about. That's really amazing. Congratulations on. That. Yeah. Thank you.

You know, it's been many years. What I'm finding interesting, I've been in medicine now for 41, 42 years. And what's interesting about this, as a board certified internist who moved when I finished my Mount Sinai residency in internal medicine and moved to the Hudson Valley, New York, the highest Lyme endemic area in the world, 41 years later, I've seen 13,000 chronically aligned patients who've come to me from all over the world.

But what's interesting is the model, in looking for answers to this chronic Lyme population, what was completely unexpected was that in looking for answers for them, creating the 16 point model, I then was able to apply it and I published this on Long-covid two years ago, that all 16 factors for chronic Lyme apply to long Covid.

All 16 factors apply now to Alzheimer's disease, right? So in my new book, An Ending Chronic Illness and going through all these diseases where we're now finding the 16 point M since model is there.

And by the way, Borrelia Bergdorf, right, shows up just like, by the way, the Alzheimer research this past week they discussed chlamydia in pneumonia.

Right. It's been talked about for a while that it's a potential the beauty about some combination therapy is I am mixing tetracyclines with the zits from mycin with rifampin with that zone.

I'm actually doing antibiotic protocols that would head chlamydia pneumonia. So it's possible that this protocol will be hitting multiple bacterial infections at the same time.

That may actually give us some results, right? No matter whether it was mycoplasma pneumonia, whether it was chlamydia pneumonia, because it's affecting a broad range of intracellular bacteria.

So, you know, it's kind of exciting that you do one thing in medicine, which helps a group, and then you start expanding it out to other areas. And that's why I'm so appreciative that I'm able to speak to you about this.

On the Alzheimer's Summit. Because Alzheimer's disease, the numbers are doubling, you know, in the next couple of decades. And it's like, we've got to find better answers than what we have right now.

Absolutely, absolutely. This is really exciting work. Let's talk let's talk a little bit more. So you've talked about how you studied this medication called DAP zone.

It has both antimicrobial and anti-inflammatory properties. And we know that inflammation is a really central issue in Alzheimer's disease. So talk talk a little bit more about the DAP zone how you're applying it.

Neuroinflammatory Biomarkers and Reversing Alzheimeru2019s Markers

What maybe some of the cautions are how how do we how to make decisions about this. Yeah. So what's really interesting about DAP zone and Alzheimer's disease.

Is there was a study published by doctor Lee in Korea, goes back a couple of years, and he had over 3000 Korean patients with leprosy. Now DAP so normally is a drug with rifampin.

And this is by the way how I figured out how to use DAP shown for Lyme disease, rifampin and DAP zone. It's used for leprosy for anywhere between 6 to 12 months, depending on whether you have multidrug resistant leprosy or regular.

But this particular regimen was used in the leprosy population and over 3000 people, and they followed them for 15 years. What they found is that the leprosy patients called Hansen's disease leprosy, who took rifampin and DAP.

Some part of the protocol I'm using, the levels of Alzheimer's disease was like six times higher if they did not take the option and much lower if they did.

So the theory behind this is that DAP zone was acting as what they call an Nlrp3 inflammasome inhibitor. So in other words, we've got these inflammatory pathways in our body.

And we know by the way, that every chronic disease you look at from autism to ADHD to Alzheimer's, allergies, cancer, it doesn't matter what you name it is due to chronic inflammation.

So with chronic inflammation, in this particular case, what they suspected was happening with DAP zone is that it was acting on an inflammatory pathway called the NLRB three.

Inflammasome also showed up with Covid at the time when when Covid was out there in its worst form, and that it lowered down inflammation in the brain.

And that's how Daptone acted. Now, it's possible that in my patient where we reversed our 2017 and improved beta amyloid ratios, it's possible Dobson was acting as an Nlrp3 inflammasome and had nothing to do with heading line.

We don't know this until we do larger studies, but the interesting thing about it is that's probably one of the ways that that zone is affecting the brain, is it has excellent central nervous system penetration.

And I love the fact that DAPs have had already been published, lowering Alzheimer's rates years before I ever published this case study. In over 3000 people in Korea.

So, you know, taking this forward now about Appsumo, we discovered about ten years ago that Lyme was a what's

called a biofilm, persistent bacteria like tuberculosis and leprosy.

This came out of John Hopkins University. And about at the same time, it was a shopee's group at the University of New Haven, Stanford. They were kind of coming out at the same point, Kim Lewis lab at northeastern, looking at the fact that why does line keep relapsing?

Because we used to give cell wall drugs like amoxicillin, Herceptin, cystic drugs like flagella or plaque with all grapefruit seed extract, intracellular drugs like zero.

And it would help, but the patients would relapse. After we stopped the drugs, we thought it was what were called the cystic forms or L forms cell wall deficient.

But in 2016, when Hopkins discovered this, I went to the medical literature and he said, oh, you mean it's a persistent like tuberculosis, leprosy. And because I had treated a lot of TB when I was doing HIV medicine at Mount Sinai, this goes back into the 1980s.

At this point, 70s and 80s and residency. I looked up the Iron Age rifampin periods and amid all the TB drugs I was using, and I came across them. And the thing about Dobson that struck me is, number one, it's anti-inflammatory.

Every symptom you get with Lyme, or even in Alzheimer's is due to inflammation. Number two, great penetration into the brain. I never have to use I. V.

drugs anymore for lime. I just use these oral generic antibiotics. We get autoimmune phenomenon in Lyme. Dobson has autoimmune effects. It basically lowers down autoimmune symptoms in the body.

It hits BBC. It has somewhat of an anti-malarial effect. Right. So it has all of these different effects in the body. But you're correct. You're asking me about the side effects.

But it's not an easy drug to use. Mainly because like I call this do no harm. H is for hurts, A is for anemia, R is for rashes, and M is for meth. Hemoglobin Amia. So what does this mean?

This means that when you start using dapt shown in this nine week protocol, you're going to start killing off the bacteria and getting what are called heart Simon reactions, which is an inflammatory reaction.

So your symptoms get worse temporarily. Your fatigue, your joint pain, your neuropathy, your brain fog. You know it all can get worse for a temporary period.

We have to detox you with things like high dose glue to iron out, catalyzing the body to make it better. But the worst side effects is the of harm is a and anemia and meth hemoglobin Amia because the are for rashes even though it's a sulfa drug people have told me oh I have a bactrim allergy.

I can't take sulfa. My thoughts is all trimethoprim. They actually can take naps on 99 out of 100 times, and I give them like an h1 h2 blocker, Zyrtec, Pepcid.

They're usually fine, but the anemia alone interferes with folic acid metabolism in the body. So I give massive doses of both. Luca Vaughan and L methyl folate to help stop the anemia from getting worse than it normally would be.

So when you take dapt stone, if you have a hemoglobin, let's say a 14, when you start, you're going to be roughly ten. When you get to the end of the protocol.

Rarely it will even drop six grams to eight. That's usually the most I've ever seen. It'll always come back to normal. But if you're a woman who starts at 12 because you're iron deficient, you want to make sure you don't have any iron deficiency anemia, no B12 deficiency folic acid.

Before you start home. So sometimes I have to start and stop the protocol because of the anemia. If a woman is starting with a low hemoglobin. But again this is important.

The labs always come back to normal. The hemoglobin is when you don't carry oxygen well in your blood. So we added in methylene blue which by the way has studies on Alzheimer's.

You may have discussed this in the summit. They're using low dose methylene blue now for mitochondrial dysfunction which is one of the hypotheses not just the amyloid hypothesis.

It's one of them as to why people get dementia. We use methylene blue because it's very effective at reversing meth hemoglobin. But methylene blue is also a persistent drug.

And it works with rifampin. And centrum acts to help Bartonella. So when I put together this protocol, which took me about ten years of tweaking, it, we have found the exact right, you know, amount of methylene blue of daptone, etc.

to basically give you a very good possibility of getting you into long term remission. But the key with this is you have to be you have to have an enzyme called G6, PD glucose six phosphate dehydrogenase.

If you don't have this enzyme, and I've only found it missing in like 2 or 3 people in 40 years, you can get worse anemia and worse mouth hemoglobin reactions from using the protocol.

So but most people will be able to take it. But it's it's not an easy protocol. I mean I tell people this is like cancer chemotherapy. It it is not easy, but it is well worth it.

And my wife is now going on eight years in full clinical remission. She was sick for 25 years. She took 50 of Daptone felt great, relapsed after a year.

PCR positive for Lyme in her blood. It went all right. She felt good with 50, but it wasn't enough to knock out the line. I gave her 100 for six months.

She went, oh, I feel great. We stopped it. She relapsed. Now this is before I knew the correct dosage. The correct dosage is 100mg of DAP on twice a day for four weeks.

When you finish double dose stop, someone with a four day high dose stops on pulse. Just four days at 400. If you don't have the Busia part, you don't have mold.

And this is what I did for this patient, by the way, that we just published in the Journal of Alzheimer's Disease case reports. That's what she did was an eight and a half week protocol finishing up with high dose Tap zone.

Dapsone, Inflammation, and Treatment Cautions

And then three months later, her her PTL levels were better, her beta amyloid ratios. In other words, she reversed her Alzheimer's markers using this 8 to 9 week oral protocol.

So all of these protocols, by the way, they are published in my medical detective Substack. I have a free Substack for people called Medical Detective.

If you look at the five Bartonella sub stacks, the entire protocol in detail, your doctor can read this and also read the article we published in the journal microorganisms back in 2023.

In 2024, this entire protocol is published so that you don't need to come to me for a consult. I have given away all of the information that I've ever had for the benefit of humanity and people out there.

So, you know, I do have a consult service, but I've tried to make it so you don't need me. Yes, I have consulted you before. This is this is really I mean, it is an incredible paradigm shift in therapeutic, not just in therapeutic

intervention, but the timeline of treatment.

Like as I'm listening to you talking about this nine week protocol and how quickly people can show improvement compared to at least in our chronic Lyme patients in the past, the years of treatment that it often took to try to get people to the other side of that disease, with the persistence and the biofilm support for the, you know, bacteria to persist.

It's really it's really. Yeah. And you know, I, I submitted and we're going to be actually looking for other grants. But and I don't want to go into this in detail, but we did submitted our 34 NIH grant because I'm absolutely convinced of what I've discovered.

And the reason we know it works. We have a culture study in culture. This was done through Iva Shopee's lab, where we showed that adaption alone lowers the biofilm persist or forms a Borrelia.

But when you add, for example, rifampin to that zone, which they did with leprosy, it lowers the biofilm load even more. When you then add a tetracycline like minnow or doxy, it lowers it even more.

And then when you add your fourth drugs, it from acts lowers it even more. So when we designed the protocol, we have culture studies showing that a for drug regimen works better than three, works better than two.

You also will never get antibiotic resistance when you mix these drugs together. We use multiple biofilm agents in the protocol, but we also have an animal study from Tufts University in mice where they showed that these animals, these mice that had chronic Borrelia burgdorferi infections after doxy, they gave them rifampin and DAP, shown in a cured them.

The mice, the mice, it was gone. Now we don't like to use the word cure with Lyme disease. But I will tell you I now have patients years and years in remission.

If I didn't cure them, I knocked out 99.9% of the bugs to the point your immune system is handling it. But I think for the people out there, again, going back to because it's an Alzheimer's summit and neurocognitive problems, keep in mind that every point on the 16 point M since model right is now associated with Alzheimer's.

So you have to look for the bacterial infections like Borrelia burgdorferi, chlamydia, pneumonia, even to fever, which this patient had. Cox yellow has been associated with Alzheimer's.

Herpesvirus one, two, seven and eight right have been associated Covid even sometimes will cause it. Parasitic infections can cause it. Fungal infections can cause it.

But mold toxins also cause neurocognitive problems, right? As do heavy metals. They've been associated even with neurocognitive decline. So please don't accept the diagnosis of dementia without going through all of these 16 points with your doctor and finding out where is the inflammation coming from and then reversing it.

And I'm treating a patient now from Massachusetts, his F-18 pet scan showed that he had, you know, a high burden of amyloid. The neurologist put him on lecanemab.

He wanted to start him on the count of Mab. And I said to him, listen, I, I'm a little bit worried about shrinking your brain and brain bleeds and side effects.

And it's never been shown that long term. This is going to be what people need. The neurologist never checked him for Lyme of Bartonella. His Bartonella fish was positive.

His Lyme was positive. He has mold toxins. So it's like you just started giving him a drug for Alzheimer's without looking at the underlying inflammatory causes.

And now we're following his PTO and beta amyloid as we keep treating him with pulsing for the Bartonella. Now,

again, I'm finishing up my practice. I'm only doing consultation medicine going forward.

I mean, again, you can read about it. It can get better.com for any people interested. But please understand I'm not looking for work. I'm very I'm very happy to play with my new golden retriever puppy and my wife and play ping pong and have a life, but I will be on a book tour later on for ending chronic illness, because this book, for me is a summation of my life's work of everything that I have discovered, and it will tell people how to diagnose and treat not just the three BS Borrelia, but Busia, Bartonella, but how this model applies to all chronic diseases and many line patients, by the way, have ADHD, right?

They have been diagnosed with a demanding illness like Alzheimer's. So it's a paradigm shift that I. I hope that our new health care system at HHS, I spoke, by the way, to the acting surgeon general, not the new one that's coming, but I actually spoke to her two weeks ago about my findings.

So I'm trying to get in at the high levels of HHS and also to get an NIH funded study so I can prove to the world that we have answers and maybe even let them look at this for chronic fatigue syndrome, because in chronic fatigue syndrome, or 16, since factors are associated in fibromyalgia or fibromyalgia.

Exactly. So it's kind of like doctors are naming diseases and throwing drugs at them instead of saying, well, hold on, every major chronic disease has inflammation underlying it.

We now know there are 16 factors. And by the way, there's a roughly 2000 scientific references backing up this book. They will all be on my website on Can't Get better.com, because we couldn't even fit them in the book.

The books are ready 640 pages from Simon and Schuster. They said we're I put this in the book. You better put. So it's like your doctor if they need science.

And I think this is how people don't trust science these days. Right? I think people have become confused of what do I trust? You need to have a doctor in the trenches for 40 years.

Who knows what works and what doesn't work, right? You know this. It's not enough to say it's been published in PubMed. People have to come to you, Christine, and go, hey, I read this, but does this, like, link up with what your experiences?

And I think that's how we're going to get people's trust back is having doctors like you and I in the trenches who say, you know, something gets published and it does work or it's published and, you know, I have not found it to work so well.

I think that's where we need to go on this. Yeah, I agree with you. And I think, you know, your point is so well taken that we've really adopted in medicine this approach of just identification and treatment instead of recognition and then searching for why, how did we get here?

How did this body become diseased? We've kind of accepted the you know, the acute model of medicine, like the strep throats and the acute pneumonia, and that model where there's one thing that causes it and a treatment that's going to reverse that.

And we've tried to apply that to all these chronic diseases that have these multifactorial inputs, inputs like you have mapped out for us in the episodes.

Yeah, yeah, yeah. And I think again, and I think this is going to happen, I think this is a major finding for me and even the Lyme community, the Lyme docs out there, the people have been doing this for years.

They are not checking these new Alzheimer's markers that you can get from quest and LabCorp. I was shocked to see that the PTL 181, the PTL 217, the beta amyloid ratios and neuro filament light, they were off and at least 50% of these patients.

Meaning it's not just enough to say, hey, we're treating you with antibiotics or herbs or whatever we're doing. We want to see these biomarkers, right?

We want to see these biomarkers go down to protect your brain going forward. And so I'm hoping that this new article that I just submitted and and in my book and in chronic illness, it'll stimulate right, the scientific community to say, hold on.

We're we're all doing this a little bit differently. But, you know, it's possible that shot therapy and I. V. ozone and some of the things out there does that.

But does it we now have a bar to set, which we know the dapt zone gets into the brain and looks like it may reverse these Alzheimer's biomarkers. Do these other therapies do that.

So we're going to need much more studies to prove this. But it's kind of an exciting time where we now may have answers that we just didn't have before for people with these chronic fatiguing neurocognitive illnesses.

Yeah, absolutely. And I'll share with you in our precision primary care division in our practice. We have been doing it up to 17 levels as a screen, and we're probably I would need to go back and check my numbers as well.

But just off the top of my head, I would estimate we're probably at about 10% of those that are abnormal in people who are 50 and older. So we can we can already identify before they have symptoms that these changes, the end result, if you will, of the chronic inflammatory pathways are already starting to happen.

It gives us an opportunity to intervene. And because of things like the M6 map and having, you know, and potentially this type of treatment, right, we have ways that we can change this trajectory.

So it is worthwhile to find out. It's absolutely worthwhile to be tested.

Broader Chronic Illness Model and Closing Remarks

Whereas before, you know, why would you get tested when it was just going to have an inevitable decline? That's what's changed. Dementia is no longer inevitable, right?

And I think that's you made an extremely important point, which is you need to get tested for these markers, because in the past, all you could do is here's some aricept, here's some Amanda, you didn't we didn't really have in our toolbox all the things we have right now.

Now knowing that all 16 emesis factors are associated with cognitive decline, you can work with your doctor and look at these factors and find out and then see, well, what can we do.

Do we need to pull them all? Do we need to treat the bard? Do we need to use dobsonian? And I think even with Dobson, what's fascinating is here you have a study from 3 or 4 years ago and over 3000 people showing that the Alzheimer's rates with zone were like 5 to 6 times lower.

And yet no one took a generic drug that's been out for 50 plus years and said, hey, why don't we try repurposing this medication? The dose they use, by the way, in that study was 100 of that.

So I'm using 200 to 400, but 100. My wife, even when she took a hundred years ago, went, wow, my brain, my body, I mean, it works. Well, what if that was enough for people where we gave them a little extra folic acid?

You don't get a lot of meth hemoglobin at 100 of daptone, and you can use glutathione to lower it. What if that was enough for people? Right. And it's never been looked at.

And I it's just it's amazing to me. And I think part of this is big pharma who's just looking for, you know, monoclonal antibodies. And look it's all great to do this stuff.

But don't ignore things that are right in front of you that can benefit the global population. When we're in the middle of an epidemic of Alzheimer's, just like we're in the middle of an epidemic of Lyme disease.

Oh my gosh, I know I'm just so grateful for this work that you're doing, for your contributions to this field, for everything that you have gifted to the scientific community from your website and your books.

It gets it's really incredible. And I just feel so honored to know you. I want to ask you if there's anything else you want to add that we haven't covered yet, because this has been an incredible discussion, incredibly informative, and I'm just so grateful to you for your time and sharing all of this.

No, I mean, I think the most important point for people is go get tested at this point with these new neuro inflammatory markers. It was not something I learned as an internist, as I said.

I mean, it would like check your blood pressure, check your cholesterol, diabetes, go for I would not have normally checked. You're saying it's about 10% of your population and I'm guesstimating.

It's about 50%. But again, with mine, I'm dealing with really severe neurocognitive issues. And people keep in mind that it's not like you can check this and say, there's nothing I can do. There are things you can do.

And I think that's the point is we give people hope that we now know that there's a new approach. You want to look at mold. You want to look at every fact you're on the map.

When I looked at it, leaky gut mass health disorder, mitochondrial dysfunction, Potts disorder, the infections, whether it was viral, bacterial, we now have answers for these type of things.

They did a study and they prove recently that some of the antiviral drugs like Pam site glitter did not work right for Alzheimer's. They just published just a month or two ago.

But that's because and I don't think this is where the science needs to change. We're not looking at this diseases from a multifactorial perspective. It's kind of like you're naming the disease and then you're going, it's a virus, or we're naming the disease and saying, here's the drug.

What we're not doing is saying that every one of these chronic, fatiguing musculoskeletal neuropsychiatric illnesses has 16 underlying inflammatory factors.

And we need so this is the paradigm shift. I just want to highlight this is what will be talked about in my new book. You can look at it on the website.

And in chronic illness.com we just got the website up. And please let people know about this, because I am so excited to share this with the world and I really look forward at the Islets Conference.

You know, later this year talking to my colleagues, because my colleagues need to be doing this. They need to be checking these markers and checking to see now with a new standard of care, a new bar that we're setting is what you're doing, whether it's herbs, whether it's ozone.

So t daps on whatever it is we now need to start comparing for the benefit. The Lyme community, what we're doing in the same thing for the Alzheimer's community.

Right. This is a new way of approaching Alzheimer's. Dementia is, you know, 42% of adults over 55 years old. There's a chance you're going to get it. I looked I was reading the literature when I submitted this article.

I was absolutely astonished. I didn't know the rates were so bad. So I'm glad you're doing the summit and raising awareness of this, but know there is hope and there are some.

Now, a paradigm shift kind of on the horizon that I think is really going to benefit people going forward. I do too.

And you have an upcoming Healing Lyme summit as well, don't you?

Yes we do. Doctor Maria Hinchey and I will be doing the upcoming Lyme summit, and in fact, we will be talking specifically about protecting the brain and neuroinflammation.

That's the focus of this year's summit because of this article that I submitted to the Journal of Alzheimer's Case Studies, which is brand new information that the Lyme community will now hear.

So, yeah, it's exciting new stuff because we're really making headway. I mean, that's one of the things we'll be sharing in our summit, too. Excellent, excellent.

And that's an inmate. Correct. That will be airing in May. That's correct. Yeah. So stay tuned. You can look at a Facebook doctor period Richard Horowitz will be posting it.

I'll probably even be discussing some of it on the Medical Detective subsection. And so that's how to get more information on it. Beautiful. Oh my gosh.

Well, thank you again so much for your time, your generosity with your knowledge. And to my viewers, if you have enjoyed this discussion today, please join some of our other sessions where we continue to dive into my passion, the root causes of cognitive decline.

See you soon!