Cerebral Folate Deficiency: The Silent Culprit Behind Neurological Symptoms Transcript

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Introduction and guest background

Hi and welcome to another episode of the Healing Lyme Summit. I'm your host, Doctor Myriah Hinchey. And today we're going to be talking about cerebral folate deficiency.

And it's possible links with Lyme and other co-infections. So here with me today is one of my great friends and fellow naturopathic doctors, Doctor Lindsey Wells.

Doctor Wells is the lead author on the recent publication on Foley receptor alpha antibodies in the pediatric Acute Onset Neuropsychiatric Syndrome, or Pans and Pandas.

She's also the co-founder of Lyme Heal Hers, which is an online summit focused on women and children suffering from vector borne diseases, as well as one of the first naturopathic physicians to receive their fellowship from the Medical Academy of Pediatric Special Needs, or Maps.

For more information on maps, you can go to Med maps.org. So welcome Doctor Wells. It is an honor and a privilege to have you here with me. Tell our listeners a little bit more about yourself and how you came to specialize in pediatrics and vector borne disease, and how you ended up being involved in and leading this study on cerebral folate deficiency.

Well, thank you so much for having me. So as you said, I'm a naturopathy doctor. I practice in Trumbull, Connecticut, and I knew early on in my career that I wanted to work with the pediatric population.

My heart was really with children with neurodevelopmental disorders like autism. I just find them to be so lovely to work with. And their families are amazing.

And so I spent a lot of time working with children with autism. And then that kind of shifted into working mainly with children with pans, pandas. And so early on in my career with Pans Pandas, you were seeing a lot of, for instance, strep causing the onset or a mycoplasma, and then what ended up happening was I was seeing cases that were getting harder and harder to treat, and there wasn't that waxing and waning presentation that we usually see around an infection.

What we were seeing is that these kids were in what I call constant flares, meaning that every day was pretty difficult for that child with some neuropsychiatric symptoms, whether that was separation anxiety, OCD, behavioral regressions, academic decline.

And it was getting really challenging for the children and the family members. And so we had to start exploring deeper, like what was going on with these kids.

And what we were finding was it was almost disturbing the findings of vector borne diseases with these kids with pans and pandas. And so it was mainly Bartonella that was coming up, which really made sense in regards to the neuro psych symptoms with these children, especially, like the rage and the aggression and the anxiety and the OCD.

But what's really also come recently, in the past few years, is also bbca coming up right behind Bartonella in my population. So that's kind of how I got into this was like just exploring deeper of like, what are these children suffering from and why are they like chronically having these flares and finding that the majority of them do have vector borne diseases.

So I had to, you know, learn how to manage those infections. And what was beautiful is seeing that these children and these families started to get relief because we found out what the infection was underneath that were contributing to the immune burden for them to be presenting with the symptoms.

So that's how I got into this field, and I feel very grateful and excited about it, that I can offer hope and answers to families who are experiencing this with their child.

Yeah, and we are very grateful to have you and all the work that you're doing and really like at large. This community is very lucky to have someone like you.

That does not stop digging at the root cause, right? Because, you know, for a lot of people it's like, oh, pans, pandas, there it is. That's the root.

And it's like, no, there's always something deeper causing that immune dysfunction that actually allows for pans or Pandas to occur. What are they? All right.

So today though we're going to focus on cerebral folate deficiency and the role that it plays. And for listeners that don't know it plays a role in autism spectrum disorder.

It plays a role in Pans and Pandas a lot of times. And also, you know, it plays a role in a lot of neuro degenerative as well as neuro psychiatric issues.

So even beyond what we're talking about, just to get it on the radar, it's implicated in depression and anxiety.

What cerebral folate deficiency is

Can be with Parkinson's can be and dementia stroke like there's there's just a much wider array of issues that it can cause here. But doctor, well, start out by telling us exactly what it is.

So cerebral folate deficiency essentially is just not getting enough folate into the brain so fully. We've probably heard about this nutrient, right. Being very important when, a woman is pregnant to decrease neural tube defects.

Correct. Can also be helpful for anemia. But what people don't realize is that we actually need folate in our brain to help with regulating neurotransmitters, to help with doing methylation, different pathways to help with detoxification.

And when we don't get enough folate into the brain, there can be consequences. Many of the things that you just, you know, mentioned on top of also seizures, schizophrenia, and then the biggest one is neurodevelopmental delays and autism.

But treatment resistant depression, dementia there's many different as I said consequences are manifestation two cerebral folate deficiency just not getting enough folate into the brain.

And what's very interesting is you can have normal levels of folate in your blood. So say you test your blood just for folate and you're like, there's plenty there.

But the levels look good. I must not have an issue. You can still have an issue and have cerebral folate deficiency because of particularly many different reasons.

But particularly what we're going to talk about is the auto antibodies not being able to shuttle that folate into the brain, leading to the cerebral folate deficiency.

Right. So you can have plenty of folate in the body. And obviously there are various forms of folate which we'll talk about. But for everyone listening, you know, it's not enough just to have a nutrient or a substance in the body.

It has to be able to get to and bind to the receptor to actually bring about the physiological change or action that

we're looking for. From that nutrient.

So why don't you tell us, tell us a little bit about the different forms of Foley and tell us about the receptors and how like what is the like Pappu physiology behind how this arises.

So this is very like a simplistic kind of pathway that I'm going to talk about. But essentially we get folate from things in our diet especially like green leafy vegetables.

That has to be converted in such a way that it can become bioactive, that the body can recognize it and plug it into its pathway. So it's going to go from folate to phonic acid to methyl tetrahedra, folate that methyl tetrahedral folate is the bioactive form of folate that again, the body can utilize in its pathways.

And so when we're talking specifically at the brain, the brain at the blood brain barrier is a very tightly regulated system. And we're very fortunate for that. Right.

So not anything can just enter into the brain. But at the brain there's going to be receptors. And I think of these as locks. And they have a specific key that can bind into it to be brought into the brain.

So there are receptors on the blood brain barrier for this methyl tetrahedral folate that methyl tetrahedra. Folate is the key. It can bind into that receptor and then be brought into the brain to go through those many different processes that I discussed previously.

But what can happen is that there can be abnormalities at that receptor site where there is actual auto antibodies that block the receptors that don't allow the methyl folate to get into the brain.

So essentially what happens then? That's when you have this cerebral folate deficiency and then may have those symptoms that we mentioned before. So someone could even be taking methyl folate.

But if you do not resolve the issues that are binding or blocking to the receptor and inhibiting the Foley, the methyl folate from attaching like the key to the lock, then you're still going to have the issue.

Absolutely. So a lot of people will say, well, I have the mgmt for mutation. And so I'm taking methyl folate. So I should be good right. That's not the case because when you have these autoantibodies there's two types in particular that we focus on or test for.

One is blocking where the antibody literally blocks the receptor. So no methyl folate combine to it. So it doesn't matter how much methyl folate you're taking.

On the other side there's something called the binding autoantibody. And that can bind to anywhere on the receptor and essentially alter its shape. So it doesn't function optimally.

So you can't get enough in. So really, what's interesting and what the research has found is that you essentially need to flood the system with the linick acid, which is a step above methyl tetra, hydrophobic eight to access a different carrier transport system in the brain to then bring in the clinic acid.

How that works. But then there's another piece to it too, right? Because that wouldn't be enough. So tell us about the molecular mimicry with dairy and how dairy is actually one of these underlying like root causes of of how this would even occur in the first place.

So there's two ways that some that we know of that somebody could have cerebral folate deficiency.

Folate receptors, autoantibodies, and dairy triggers

One is a non-genetic hereditary type of process that occurs. So this is something that somebody possibly could be born with. Right. And what we see is that there is some like mothers and fathers can have this and siblings can

have it even if they don't have the manifestations from it.

The other piece is that we can get this from consuming dairy products. And this is all animal dairy. You know, most of the research is just with cow dairy, but we can kind of make the assumption that it's all animal dairy.

So what happens is molecular mimicry that with cow dairy, what we know is, is that the folate receptor autoantibodies, those antigens are about 90% like homologous to what we have in the antigens in our body.

And so when we consume the dairy what happens is our intestinal our intestines will actually make those autoantibodies itself. And so when we're drinking the milk or taking the dairy product, we're just essentially causing our body to make more of those autoantibodies.

And then what that's doing is it's blocking the receptors on the brain. And so one of the biggest treatments besides giving high dose for clinic acid, is removing all animal dairy from the diet completely.

And it's so interesting. There's been some research not much, but looking at this to see how the autoantibody levels drop significantly once somebody takes dairy out of their diet, even when you're not giving high dose full and a gas at the high dose one a acid essentially just giving your brain the folate that it needs, right.

But in order to bring down the autoantibodies, you have to remove the dairy. And then when you reintroduce the dairy, how it almost skyrockets. Again, those those folate receptor autoantibodies.

So that's a very important piece of the puzzle because a lot of times when people want to, you know, go through treatment, it's much easier to take a pill of phonic acid than it is to modify their diet.

But it's almost like you might get some relief from it, and you probably will, just not to the extent that you could find relief from symptoms and really long term treatment as the two of them together.

The removal of animal dairy and also implementing, high dose phonic acid. Right. And it's almost like, you know, this this can be reversible if you do both things together.

It might not be reversible to the point that you can eat dairy again, but you can resolve the issue by taking high dose splenic acid and avoiding all dairy products.

I just want to make one point to our listeners. So this is not something where you would go and have a food sensitivity test, like looking at IgG or IGA.

This is not something where you would go to an allergist and have an IGI towards dairy, or casein or whey, or any component of animal dairy. This is something completely different where the immune system gets triggered and confused because of the dairy and makes these autoantibodies.

So I just want it to be clear. If you've had dairy ruled out, you know, as an allergy, that has nothing to do with what we're talking about. Absolutely correct about that, because you're not looking for antigen antigen or the immunoglobulins against dairy.

You're looking for folate receptor autoantibodies. So they're completely separate. And you can have somebody that does not have a sensitivity does not have an allergy to dairy.

But yet they are very positive for the full receptor autoantibodies or vice versa. Somebody who is sensitive to dairy or allergic to dairy and they don't have the autoantibodies against the receptor.

So therefore they do not have cerebral folate deficiency. And that's also that's a great point. And another thing I want to bring up is and just kind of reiterate is the same thing with the Antifa mutation, I feel like a lot of people get hung up on that thinking, okay, well, if I have MTX for I have cerebral folate deficiency.

They are two completely separate things here. The MTA. GFR is a genetic mutation that you're not able to, you know, convert your full weight to the bioactive form as well as you should be.

So there you have a harder time going from folate to the methyl tetra hydrophobic when the cerebral folate deficiency is just because one of the biggest reasons is those autoantibodies.

And you can have autoantibodies without having MTX, or you can have MT AFA without having cerebral folate deficiency. So that's a very common question that I get as well or an assumption that people make.

I must have that too, because, you know, or my child must have that because they haven't cfAa. It's it's completely separate, right. So before we move on to talking about potential links between really severe neuropsychiatric symptoms in Lyme and the possible connection, tell me a little bit more about, I want to talk about why treating with clinic acid is more important than using methyl folate.

And then also I want to talk about testing for these autoantibodies. So the research for cerebral folate deficiency is really done with Luca Vaughan which is a clinic acid medication.

And so that's where we have the majority of our research. But also clinically we see much better outcomes using clinic than we will with methyl tetra hydro folate.

So for clinic acid, when you use high dose for clinic acid it has been shown to allow for the reduced folate carrier. So it's a different carrier that's on the blood brain barrier to shuttle in the folate.

So essentially that one gets activated when there's high extracellular. So a lot of folic acid in the system to move it in. We don't have as much evidence that that occurs with methyl tetrahedra folate.

And so I think there's only been like one study Urso that has shown that you could have the same effects. But again, clinically in my practice I have many children and adults with cerebral folate deficiency.

And we always see more resolution in their symptoms by using the high dose phonic acid. Okay. Great. And so now if someone wants to get tested to see if they have this autoantibody process causing cerebral folate deficiency, what testing do you recommend and explain the testing a little bit more?

The company in the lab name is religion. And you can go online at frat now.com to find information about the test. To find information about how to order a kit, you likely will need, physician name in order to get a kit, but you can inquire with them.

I think they also have a list of providers as well. So the providers who are ordering this test, if maybe you want to establish care for them, for yourself, for your child.

And what I also love about their website is they have a lot of the research out on cerebral folate deficiency. So if you do love to read the research, you can kind of find it all in one place there.

Are there any prevalence numbers in the in the Lyme community as far as like the prevalence? I haven't seen any studies, but I know that you look at those very closely with with your patient population.

Yes. So there is no research out right now in regards to cerebral for a deficiency in tick borne disease or vector borne diseases. And this is something that I'm very passionate about as I know you are too.

And I've been scrubbing my own data. And the data actually looks very promising that about 50% of people who have vector borne diseases are also positive for the cerebral folate deficiency.

This is obviously pre published, but it's, you know, part of my mission to get this out, this information out. And so I'm still pulling that and going to explore a little bit further.

Of the ones who are positive, what symptoms are they manifesting. Because my suspicion is these are patients who have tick borne diseases and are experiencing neuropsychiatric symptoms from the vector borne disease.

Testing, prevalence, and links to Lyme and PANS/PANDAS

So whether that's, you know, significant depression, anxiety, OCD, brain fog, memory loss like dementia type symptoms, bipolar type symptoms. Like this is what my suspicion is.

So but the prevalence looks very interesting here. And so hopefully I'll have that you know yeah. Partner with me Maria to do that. And we'll get that information out.

Yeah I would love to. I'm as you know fascinated in the same area. And yes hopefully by the time you know this is airing in April, we will be way underway to have those answers to share with all of you listening, because it really would be fascinating.

There is a lot of this vector borne disease patient population that have way more neuropsychiatric and cognitive issues than they do musculoskeletal. And again, it would be so interesting to see if this is part of what's playing that role.

Okay. So let's transition into talking about treatment. So obviously we've alluded to you know using high dose splenic acid and removing dairy from the diet.

What else do you look at when you are treating patients that have cerebral folate deficiency. You know it's a great question. You know I will look at some other markers too.

Like looking at other B vitamins. I'll look at their blood cell count. So liver and kidney function, their mitochondrial markers. But really the big treatment here is doing the high dose full intake acid and removing the dairy.

And just that in general brings down a significant amount of inflammation. Actually it helps improve their detoxification pathways. And it also makes their treat like the other treatment that they're doing more effective.

And we see this actually in the treatment resistant depression population where there's studies are ranging from about a third to over a half of patients who have treatment resistant depression have cerebral folate deficiency.

And when high dose formic acid is added to their treatment plan, they are finding it finding moderate to significant relief in their symptoms when added with their psychiatric medication that they were taking.

And so that's I also I'm finding that in my practice with people not necessarily using psychiatric medications, but with them just going through treatment, right of antimicrobials, more immune support, anti-inflammatories.

And it's like we're seeing more improvements in their symptoms adding in this piece than we are, you know, with not having it end. Yeah. And you know, it's important to talk about two I think I think sometimes the level of anxiety is literally debilitating.

So it's like it's hard for these patients to even be able to follow any simplistic treatment protocol. Not that, you know, treatment for vector borne disease is all that simple.

But I mean, it makes it very, very hard for them to carry out their their daily living tasks and so when you can bring down the level of anxiety or the level of confusion, you know, just those two things alone, it actually enables patients to then be able to look at the bigger picture and do a lot of like the dietary interventions and expanded protocols that it takes to really heal the body from Lyme and co-infections.

A couple questions. So when you say high dose full anycast at what does that mean? Because the typical supplement on the market is 800 micrograms. Right? Right. What are we talking.

The high dose in the literature is 2mg/kg up to 50mg per day. So we're talking 50mg for most people even even children. You know who it's significant at 2mg/kg.

And it's kind of surprising when you have, you know, a five year old in your office. And they need to be up to 50mg. But yet you then have to tell them they have to take, you know, to almost two bottles of what's available on the market per day.

Oh, yeah. Sorry. Per day. Yes, per day. I had I had a parent for a less than two year old crushing 15 tabs. Yes, twice a day. Yes. And they were I think their, their supplement was costing them around $600 a month.

Right. Just to take that. Yes. And so there is a pharmaceutical available on the market. It's called Luca foreign and Luca Veran, can be prescribed by physicians.

What I find is that it's very challenging to find a physician to prescribe this, because Luca born is classified as a chemotherapy drug because it's a rescue for chemotherapy.

It helps to replete the full Anik acid and the folate that's essentially depleted from going through chemotherapy. In my population. I have a child and they need to go on full Anik acid and maybe I'm working with a pediatrician or somebody who has prescription rates and saying, will they prescribe full Anik acid?

And then they look at the parent like, I'm not going to prescribe your child a chemotherapy drug. So that's one challenge. The other challenge is that we look over and that's available just through the regular pharmacy a lot of times actually has dairy product in it has some lactose in it.

So it goes against what we're trying to accomplish because we're also telling the family you have to remove dairy. But yet we're then giving the medication that has it that treats this condition and giving them dairy product with it.

So then the other option is to get it compounded and when you get it compounded, it can be extremely expensive and it can range depending on the price depending on which compounding pharmacy you're using.

And so there are some challenges here in regards to treatment. Yeah. Yes okay. So how long does it take if someone is very diligent. So let's say they remove dairy 100% from the diet and they start taking the appropriate dose of volcanic acid.

In your experience, how long does it take before you start to see a change? You know, it's a spectrum, right? And so sometimes I'm amazed by how quickly I can see it.

Like I can see it by the next appointment, right. Which might be eight weeks. Or realistically, I think it's going to take at least three months. The reason being is with dairy in the system, I think, you know, the immune response to that can take some time to go down.

Right. So I tell patients to realistically give it at least three months. But it's surprising because a lot of people will see it before then. And I think even in the research when they're looking, say again at treatment resistant depression, when they're looking at the trials, it's not very long trials.

It's still around that three month mark, like the 12 weeks that they're kind of seeing what the improvements are. So that's usually around the time. However I do treat for longer than that though.

So just because you get, you know, the results that you're looking for around, then I will continue to treat for a minimum of a year, most of the time, depending on what the result is.

I will treat longer than that. Okay. And is there anything that you would like to share from the study that you were the lead author on? What? Our audience.

Yeah. So the the study that we published was the first in the population for Pans and Pandas. It was also looking at

prevalence. And so what we found was that 63.

Treatment with high-dose folinic acid and dairy removal

8%, almost 64% of children who were in our, study group were positive for the cerebral folate deficiency due to having either the binding or the blocking autoantibodies.

What was so interesting about that, that the kids who were positive, where we had some that had some overlapping comorbidity with autism, and when we looked at the children with autism versus the ones who had just pans and pandas, their levels were actually much higher than the children with autism, which is really fascinating being in my world and in my community, because cerebral folate deficiency is so prevalent in children with autism.

So it's like, what does that mean that these children with pans pandas have much higher levels than the children with autism? It was just that that's interesting into question mark.

And I don't quite know. But the other thing that was interesting is that those who had higher levels of, for instance, the binding autoantibody, they were more likely to have tics.

And so this could be something that's, you know, more prevalent maybe in the Tourette's population we don't quite know, or the children who have childhood tick disorder, which is very common.

Are they the ones with the cerebral folate deficiency? And is that then predisposing them possibly to having immune dysregulation. And they could present with pans pandas down the line maybe I don't know.

So that was fascinating. What we also found clinically is the children who had cerebral folate deficiency when we treated them again with the high dose splenic acid and took out the dairy, they had significant improvements in their symptoms.

And so this was the OCD, the anxiety, the tics, the depression and what I'm most interested in as well right now is looking at speech, this fluency. So about 50% of children with pans will have some form of speech, just fluency.

So that could be stuttering, that could be mutism, that could be putting in words and not really being able to get their thoughts out. And I've had some cases in my office that those children that present with that are positive for the cerebral folate deficiency.

And when we put in the high dose phonic acid, for instance, the stutter completely resolved. And so this is something that again, I just find fascinating in the population for pans pandas.

And it really made me curious because my population really is the ones that have underlying tick borne disease with pans pandas like, how is this playing a role in tick borne disease and not just pans?

Pandas at that. If you know and how this all comes together, really the symptom relief that we can get these people, regardless of the infection in the immune dysregulation because like you said, even being able to bring down the anxiety, being able to bring down that brain fog and helping with mental clarity could change somebody's life and regard like their how they are able to get through and move through treatment.

I mean, it's just going to improve daily living significantly, right? And if you look at all of the other actions of folic acid in the body too, right, like you're right, like it could actually explain why some cases of even if we just looked at Lyme as, as that vector borne disease, why some patients can have such severe neurological conditions even with neuropathy and things like that.

Right. Versus, you know, somebody else. Yes. Question. Because I so this just popped into my head, I would imagine that a lot of our listeners are experiencing the same.

So what's the harm in taking a high dose splenic acid like is there are there potential side effects? Is there a

downside? Is there a point where you know too much?

Is too much? And what would that look like? Talk us through that. Yeah. So what can happen when you put too much phonic acid in too quickly? Is there can be some side effects, mainly agitation and irritability.

That's the number one that I see. And the second one is insomnia. So in the literature they're also going to put aggression down. But I don't see that clinically.

So it's the agitation irritability and insomnia. Essentially what's happening is that your the neurotransmitters are almost kind of like depleted from not having this folate in the brain.

Right. And so all of a sudden you're flooding the system with that. And what you're kind of overwhelming the synapses occurring at, say serotonin, neurotransmitters the dopamine neurotransmitters.

And it can cause an increase in some of those symptoms. Right. And so that's when you go too fast and too much. And so I think kind of staying in that weight dependent range up to 50mg is very safe.

But it's how you go about implementing it. That is what you need to watch. So usually I start at much lower doses than what their weight dependent dosage is and move slowly and at.

Sometimes I move faster than they think some of my other colleagues like I'll keep going up every week, and then if somebody has those symptoms, I just bring it back down and hold there for, you know, two weeks before moving up again.

Right. But sometimes then, like the literature, what they're saying is like, you can go like every month kind of increase it up. But my patients and my clients.com, you know, a lot of them are really suffering.

Right. So I try to get them the released that I know is possible. And a quicker time frame because I'm able to help them through. Okay, you're experiencing this.

Let's back down and let's, you know, try again at this time and kind of give them the instructions again in the literature, what it's saying is that it is a very safe intervention.

And if you are suspecting this, that it might be worth doing a trial of putting it in for a period of time without necessarily doing the testing. Right. So you're saying you have a weight dependent target dose that you want to reach.

Where would you start? Like how would you derive your starting dose and how many weeks do you typically like for the typical patient, you know, do you take to achieve that target weight dependent dose.

So you're going to start at X and you're going to arrive at that target dose. But like over how many weeks. And you know do you do you double every week or two weeks.

Like how do you work up to it. Yeah.

Dosing, side effects, and closing advice

So again it kind of depends on the person and how big they are, but usually like and how sensitive they are. That's another thing, right? If you're somebody or your child is super sensitive, they react to everything.

You're going to want to start lower than what I'm going to say right now. But I think 7.5mg is actually a pretty good place to start. And so I'll do that for a week and then every week increase it by 7.5mg up into the weight dependent.

Personally, I find 30mg to be like a sweet spot a little bit for people and I'll see something around that time. And if I'm seeing an improvement, I will kind of go quicker to get to the end goal.

But you don't want to rule out that the intervention didn't work for you unless you're kind of at that weight dependent dose for, you know, as I said, a period of time, which minimum is going to be three months, I really think it should be up to a year for a treatment there.

Okay. All right. Well, any last words of advice that you'd like to leave our listeners with? I would love to keep talking, but we are pretty much out of time.

Okay. Well, this has been wonderful, but I think the biggest thing is this should be explored. If you or your child are experiencing neuro psych symptoms associated with vector borne diseases, this could be an additional piece that's not necessarily infection driven, but perhaps there was a susceptibility that occurred beforehand, or a dietary intervention that you could implement to get relief for those symptoms.

And so there's still so much information that we need to figure out regarding cerebral foye deficiency in this population. But as I said, when looking at my data, it is very interesting and also promising that this could be just a piece of the puzzle that's occurring for you to get you relief from these persistent symptoms that you're having just to make life easier for you while you're navigating this vector borne disease.

And this illness. And so I encourage people to explore further, and I wish more practitioners also would be testing for this, because it can make a difference.

And the outcomes of treatment plans. Yeah, absolutely. So are you taking new patients? I am taking new patients. But there are listeners how to find you.

So you can go on to my website, which is Lindsay Wells Indeed.com. And so all the information about my practice is there. And you can also find me on Instagram, Lindsey Wells and and I do love to post about vector borne diseases.

I love to post about Pans and Panda Cerebral folate deficiency. And you can find more information there. And you can also check out my paper that you know, I did write in regards to the Pans Pandas population, and hopefully you'll see more coming out.

About cerebral folate deficiency specifically for this population. Awesome. Any upcoming events you'd like to share? This upcoming event that we have going on is the Lyme Healers Event.

This is a free online summit and it's focused on women and children who have vector borne diseases. This year, we're going to be focusing more on neuropsychiatric manifestations of vector borne diseases.

But it's a free online event and you can sign up for free at line heal her, stop. Awesome. Yeah. And by the time this actually airs, that will have been over.

So you'll have to catch the replay. Yes. Which will do you for that? Yes. Sometime in the summer of, 2025 I guess by then. So yes. So we will definitely have an encore going on, but you can always check out the recordings on the website.

Thank you for that, Mariah. Yeah, absolutely. All right. So thank you so much for being here, Lindsey, and for all of our listeners at home. Thank you for joining us.

And I truly hope that this helps you on your journey to healing Lyme. Take care. We'll see you next time. Bye.