Introduction to PANS, PANDAS, and Molecular Mimicry

It all starts with an infection, but the infection itself is not just any infection. Once the infection occurs, we all make antibodies, but unfortunately in those patients who have a susceptibility to this self-non-self dysregulation, their antibodies are made against a part of the infection that looks like a part of our body in this case.

It's the brain. So when it attacks our joints, we call it rheumatoid arthritis. When it attacks our pancreas, we call it diabetes type 1. When it attacks our thyroid, we call it Hashimoto's.

But what if it attacks our brain? It's a biological response to an infection through molecular mimicry that must be treated as an infection-mediated immune autoimmune disease.

Welcome to Demystifying Pans and Pandas, the podcast where we uncover the mysteries, breakthroughs, and hope behind these life-altering conditions. I'm Dr.

Nancy O'Hara, a board-certified pediatrician, educator, and advocate with over three decades of experience helping children and families navigate the challenges of neurodevelopmental and neuropsychiatric conditions, especially pans and pandas.

These disorders can feel overwhelming, but here we'll break down the science, explore transformative treatments, and share stories of resilience and recovery.

If you've ever wondered what's possible for your child or how to find answers, this is the place to start. Let's dive in. Welcome back to our podcast on demystifying Pan's Pandas.

And I am so thrilled to welcome Craig Shimasaki, to the podcast today. Craig is an amazing researcher, scientist. He's the president and CEO of Moleculara Bioscience.

He's the adjunct professor at the University of Oklahoma. He's gotten a BS in biochemistry, a PhD in molecular biology and biotechnology, an MBA from the Kellogg School, and just on and on.

And He's a co-founder and CEO, as I said, of Molecularo Biosciences, and his research has been well documented. He's co-founded multiple companies, led multiple products through FDA approval, and now lends his tremendous amount of knowledge and passion and compassion to our field of autoimmune disease, basal ganglia, encephalitis, pans and pandas.

So Craig, thanks so much for joining us. Thank you, Dr. Herrera. It's a pleasure to be on here with you and the great work that you've been doing. I really appreciate it.

So tell me, because I don't even know this, how did you get to Moleculara? How did you found it? How did you do that? Well, that's a great question. And it's a kind of a serendipitous journey.

You know, I also help mentor some first time CEOs that are doing biotech companies and tell them about serendipity and how important it is.

Craig Shimasakiu2019s Background and Moleculararu2019s Origins

Well, Dr. Madeline Cunningham has been researching Sydenham, Korea and strep infections for like 40 years. And then Dr. Sue Swedo was involved with studies and finding out and defining pandas and pans.

And she contacted Dr. Cunningham about her work in strep. That led to one thing to another and another clinical

study that they did of about a thousand patients and she had five markers that she identified auto-antibodies that were attacking the brain.

Patients were getting well and recovering because they were treating the underlying root cause. The study was over and after the study was over, parents were telling other parents and for over a year, they were contacting the university asking for how do I get into this study?

And there was no more study. Then they began asking the governor and they got the provost and found out there was no way that they could help these patients.

So that's when she came to me and says, how do we help these patients because they're suffering and we have no way to help them. And that's when I said, well, I would need to have to start the company and there's a lot of stuff involved with that.

But first of all, let's make sure that there's something here that will actually impact the lives of lots of people. So that's really the genesis of it.

And that was about 13. I remember when it was just getting started, we were providing some of the blood for those original studies. So I remember it was almost 15 years ago now that I had a young man with autism who had gone Berserk, you know, literally the OCD, the ticks, the climbing the walls, the not sleeping, the urinary symptoms, all the symptoms we see.

And we could not figure out what it was going on with him. And we did the molecular Cunningham panel and his cam kinase was 206. That was one of the highest.

Yeah, exactly. And as soon as we actually treated him with intramuscular penicillin because he had so much gut dysfunction and one dose and he was a different child.

I want to start a little bit with this disease that we call pans pandas that I think really should be called basal ganglia encephalitis. And I've heard you on other podcasts and webinars do a such great job of talking about the molecular mimicry and what this really is.

Could you talk to our listeners that are really not understanding it as well? I'd like them to hear it in your words rather than mine. Well, and you know, it all starts with an infection.

But the infection itself is not just any infection. There tends to be certain types of infections. Then actually, as you mentioned, molecular mimicry, it's really the identical amino acid sequence in certain proteins in infections that are identical to certain amino acid sequences in parts of our body, like in our brain or in our neurons or in our different parts of our receptors.

And so it starts there. So once the infection occurs, we all make antibodies, but unfortunately in those patients, who have a susceptibility to this self-non-self dysregulation, their antibodies are made against a part of the infection that looks like a part of our body, and in this case, it's the brain.

So when it attacks our joints, we call it rheumatoid arthritis. When it attacks our pancreas, we call it a diabetes type one. When it attacks our thyroid, we call it Ashimoto's.

But what if it attacks our brain? Do we call it bad parenting? Do we call it rebellious kids? Do we call it a bunch of other things? When in fact, it's a biological response.

to an infection through molecular mimicry that must be treated as an infection-mediated autoimmune condition, which you've described as potentially basal ganglia encephalitis, or now we know as PANS and PANDAS.

It's such a great reminder when you say it's easy when it's rheumatoid arthritis, when it's diabetes, when it's heart disease. We see it with myocarditis.

We see it with so many things. What's your feeling, understanding? Why is it so hard when it's the brain? It is one of those areas that really is the last frontier in medicine is the brain.

And it's not that really that long ago, if you go back into the 50s, when psychiatric medications actually came about, which is helpful for many patients, but in these patients, as you know, that's not the underlying root cause.

So they actually get worse. But if you go back a little farther, we were actually, you know, using what was called the fever therapy, and we were infecting them with different disorders to show that they can actually recover.

Some were being castrated, you know, the lobotomies were being done. So it is an area of medicine that is challenging, but it's no different from any other autoantibody attacking a different part of our body.

It's just that medicine and science have not caught up to it yet, and you don't want patients going through multiple doctors in years and sometimes decades before ever realizing that this is a treatable infection for the underlying autoimmune condition.

And you and I have talked about this and I've heard you talk about it before. And for me, I'm always looking for silver linings. And maybe one of the small silver linings of COVID is that more practitioners have seen COVID or long COVID being the manifestation of an infection causing the immune system to attack self.

So maybe we're getting more out of that. Do you agree? Absolutely.

How the Cunningham Panel Was Developed

In fact, I do think all the unfortunate things that have occurred with it, it has helped the medical community recognize and understand that infection can trigger the immune system to go awry and begin attacking parts of the brain, parts of the heart, parts of the other parts of different organs in the body, and treatment with immune modulators and anti-inflammatories actually are helping these patients get well.

So you're right. Unfortunate as it is, it has made the recognition of infection triggered immune mediated disorders much more well understood. And I think better received now that they're recognizing that this is actually what's going on.

I agree. And very early on in COVID, we had a couple of teenagers that had abrupt onset of neuropsychiatric symptoms, got admitted to psych hospitals.

They did a COVID test. It was positive, but they decided because of COVID to give that particular child IVIG. all their neuropsych symptoms went away.

And the doctor called me and said, I don't understand this at all. And I'm like, I do. It takes things like that for other practitioners to start to see it, I think.

Well, Nancy, I'll share with you one of the other areas of research that we're doing. And again, this research came from Dr. Cunningham's lab. Is that just like antibodies can attack the brain, they can actually attack the heart and cause atrial fibrillation, myocarditis, cardiomyopathy, and parts of chronic heart failure.

So we are doing clinical validation on five auto-mute targets around the heart that will identify and predict these patients that are suffering from an infection triggered, a neon mediated cardiovascular disorder.

that again will be treated with anti-infectives, immune modulators, and anti-inflammatory agents, and they can recover. So we're very excited about that because any organ in the body can be triggered to be attacked by an antibody.

As we know, it's hard to identify and diagnose what the underlying root cause is without an identification and a test. I do want to talk more about the molecular Cunningham panel because I think people really need to understand the

test better, the five antibodies that you're testing, et cetera.

So could you talk about that in more detail and what each one means? Yes, and so we think about PANS and PANDAS as you know, it's a clinical diagnosis.

But the clinical diagnosis, you need some kind of biological evidence in some cases to be able to say there's an immune response. And so these five targets are, and I'll name them off, two of them are looking at dopamine receptors, so dopamine D1 and dopamine D2.

And then we're looking also at lysoganglioside, which is the myelin sheath around nerves. And then tubulin, which is very highly concentrated in brain cells.

In fact, this is where these targets are isolated for our testing. And then the fifth one, which is a specialized test, which is called CAM kinase activation.

And this is where we grow a human brain cell, not the patient's brain cells, but a human brain cell and culture. And we incubate the serum of patients on top of these brain cells.

And then the antibodies, if they bind, they are capable of stimulating this enzyme called calmodulin calcium dependent cam kinase. And so why is that important?

Well, calcium calmodulin cam kinase up regulates the synthesis of three neurotransmitters, dopamine epinephrine and norepinephrine. So what you're having is if any of these are positive, you're having an antibody stimulate either the upregulation of these neurotransmitters or blocking the actic, the normal activity of dopaminergic activity or interfering with neuron transmission.

which would produce these different kinds of symptoms. And it's one piece of evidence that's very important in recognizing an underlying root of an autoimmune condition for these patients.

Yeah, and I think it's very helpful to have all five of those. And I've also seen you talk about how different symptom presentations do coordinate or correlate with each of the different measures.

Can you talk about that a little bit? Yeah, that's been one of the more fascinating things because now we have tested over 16,000 patients clinically, received data and information about their symptoms and their treatments and all those.

And through AI, we're working on an algorithm to predict treatment efficacy. But we've also found that when patients have antibodies against dopamine D1 receptors, what we see is more the psychiatric symptoms.

Patients will have different, even sometimes they'll have psychosis. but you'll see these psychiatric manifestations. When they're against D2, what we'll typically see is movement disorders.

So they'll have choreiform movements or they might even be diagnosed with ADHD because they just couldn't sit still or they would keep continuously moving.

When they're directed against Lysoganglioside, what we do is the coordinated with the tics and we also see joint pain for some reason. And then when they're against tubulin, which seems to be very frequent, we find complaints of OCD and brain fog.

And so we'll see a lot of that. And then when we see an activation of the cam kinase, it's tied in with what we see with a sympathetic nervous system activation.

Understanding the Five Autoimmune Brain Markers

So they will complain of easily startled, midriasis, fight or flight syndrome, anxiety, all kinds of activities that look like

an activation of the sympathetic nervous system.

And so the reason all five are important is because we don't know which ones will be positive and their symptoms tend to correlate with whatever ones they have antibodies against.

So it is an important reason why we test for all five, but it does help identify and correlate with symptoms. In fact, one of our nurses typically will spend most of the day consulting with the physicians who place the orders.

And all we have to do is look at the test results and we'll be able to describe the symptoms. And many of the doctors say, do you know my patient? How did you know my patient?

Well, it's just here in the results. And so that's been very helpful to see that correlation. Yeah, and I think that's very important for people to understand.

Let me ask you a couple follow-up questions on what about the positivity? So, for example, if a child is in a flare, meaning a worsening of symptoms, or if they're coming to you at a time when they're doing well, do you feel that has an impact on the test?

It can. Now, we do ask that either the patient have symptoms or be in a flare or not be, you know, at a, let's call it baseline. Because you can still have that, but you won't show as elevated as you normally would.

And these are metabolic tests, meaning your antibody levels will increase and decrease throughout, you know, a course of a syndrome. So we want to be sure to be able to capture that.

But yes, it is important that the patient be symptomatic. If they are in a flare, that's great. But as long as they're still symptomatic and haven't fully recovered, because we do take post-treatment recovery tests just to show that they're back to baseline.

And it is an important element to remember. Okay. And so you do, that was a follow-up question I had. You do recommend following up once the child is better and follow up to that follow-up.

How long after, for instance, if they got better because they had IVIG or plasmapheresis, how long after? Those are great questions, Nancy, because one, if the patient is well and they're fine, you don't necessarily need to take that.

Although we do have many people who do take it so we can demonstrate and we do case studies and we do publish them. If a patient may be recovered to like maybe 50 or 60 or 70%, but you're not able to get them past that, it is opportune time to see what is going on.

Is there still autoantibodies or maybe it's an infection instead? That's when we do see a lot of retesting. Now your question about post-treatment with IVIG, and here's one thing that we have found because we have tested lots of IVIG.

And if you think about this, a thousand patients or more, many thousands of patients are contributing to these. it is not unusual that you might find some of these same autoantibodies in these preparations.

And we have occasionally seen that. However, recall that these are not endogenous antibodies, they're exogenous antibodies. So your own liver and everything will be filtered and eventually removed.

So what we recommend is to post-treatment test anywhere from, let's say, six to 12 weeks, if you can, the longer the better, just because you might run into maybe the potential of some results that might represent more of what's going on with the antibodies that are in the IVIG than what's going on in the patient.

Now, that doesn't always occur because not all lots like that. But it's better because you want to see what's the steady state data of the patient rather than maybe something due to that.

And as you know, the half-life of IVIG is kind of guessed around 45 days. So you want to look at that. Okay. And then from an insurance perspective, it is rightfully an expensive test, but some of my patients, and I have at least noticed that a couple more are getting some insurance approval.

And then secondarily, are you finding any insurances are accepting the Molecular or Cunningham Panel as a reason to do more expensive treatments like IVIG, plasmapheresis, et cetera?

Yes, and both those questions are important because we know that there is an expense to this and so we as a courtesy to the patients bill their insurance if they submit their insurance information.

How? All of our patients get some type of reimbursement. Because we're out of network currently, a lot of times it'll go to their deductible. So we do bill and for insurance and we do help them and then We usually cut checks back to patients at the end of each month.

Now, as far as getting coverage for, let's say, IVIG, we have had many instances in which the tests have been used. We call it the autoimmune brain panel now, formerly known as a panel, but they use that as evidence that there's a biological reason to use IVIG.

And many of our clinicians have told us that it's been the difference between getting insurance coverage and not. As you know, the ID insurance is always challenging anyway, but as long as there's some biological support, they've been finding that this is very supportive of getting treatment.

I have recently listened to and spoken to the president of Claimable, which is a company that's helping more people get needed interventions covered by insurance.

And he mentioned that specifically this autoimmune brain panel will call it rather than the Cunningham panel. But I understand how now we're not calling it the Cunningham panel anymore.

We're calling it the autoimmune. And maybe in that way, it's a good thing. because the autoimmune brain panel may help more insurance companies to see it as what it is.

Yes. And I decided to name it the Cunningham panel in the beginning. I believe that people would recognize for research and they would do that. Now that we're at 16,000 patients beyond that, most of the clinicians say, Cunningham?

Who is Cunningham? without having to go through that, but she's relieved and we decided to make it more generic and we're adding more things to it as we're working on the research.

So that's the reason we made the change. That's great. And is there any other research you do want to talk about? Craig, I know you mentioned a couple of things, but any of the studies that you, other studies you've done that you want to mention?

Yes. So back to the autoimmune brain panel and a couple of others, but there's been a few published studies that We've worked with some clinicians on one that comes to mind is a 16 year old girl who was diagnosed with schizophrenia at Cincinnati Children's Hospital, who was in and out of treatment facility and was on the land of pain and many of the drugs that you would expect, but not so treatment resistant.

Test Timing, Retesting, and Insurance Coverage

So they were gonna put her back into the institution. And Dr. Bermstein, who's an immunologist there, knew about our tests. He had ordered it. She was highly positive with four of the five.

And he ordered plasmapheresis. And in the published study, case report, it said within two weeks, she was back home playing tennis, off of a land of pain, and back home, it's still normal for this day.

So after I read that, I reached out to him and asked him for a blood specimen post-treatment. And he said, well, we thought about it, but she was well, so why would we want to cast?

I said, well, right. So he sent it to us. And sure enough, we were able to show all our antibodies. And she's still healthy and doing well. And so those are just really gratifying to see that the patients are doing well.

She's done other things. Immune-mediated disorders triggered by an infection, as you know, can attack multiple different organs. And there's thoughts about lupus and chronic fatigue and other things that are also potentially believed to be immune-mediated and infection triggered.

We're also seeing and working on a project here. of early onset immune mediated autoimmune Alzheimer's. There's now over a hundred failed clinical trials that are trying to target the tau and amyloid.

The belief now is that these proteins are immunopeptide that the immune system is trying to protect the brain. And there's now evidence that many are saying Alzheimer's is really an autoimmune condition, which we believe that.

And so we can identify early and intervene. That's one of the things that we're working on as well as the cardiovascular, the immune mediated cardiovascular panel.

So we have got a lot of things in the works. It all takes money. So we've written a number of grants. We have a lot of support people, but it's just great and amazing because they're what motivate us.

Yeah. Well, I think when you talk about Alzheimer's, I also think about the other end of the spectrum because in autism, particularly regressive autism, we look at a lot of those children as infectious triggered or immune mediated or autoimmune disease, not all, but I wonder after the Alzheimer's study, to look at some of those children too because we've seen it clinically certainly.

Absolutely and we have been looking at the ICD-9 and ICD-10 codes of all those 16,000 patients that have come to us and when they're positive what we've found that a very high percentage of them have been diagnosed with ASD and prior to that we're actually seeing even some Parkinson's cases.

So as you know, these are all clinically defined syndromes, but underlying the etiology via different biology. And so that's why it's so important to be able to know.

And if you do, you treat the root instead of treating the symptoms. Yeah. Yeah, and going to that root cause, you know, that brings us to my mentor, Dr.

Sydney Baker, and that was what Sid trained me in. You know, it's not about name it, blame it, tame it medicine, meaning we name it Alzheimer's, we've got to put an Alzheimer's drug on, or we name it autism, we've got to put an autism drug on, and that will cure all that ails them.

It's really about looking at the root cause because there may be more similarities between those with Parkinson's, Alzheimer's, diabetes, arthritis, autism, Pan's Pandas, then there are differences because the root cause is so similar.

Yes. And as you mentioned, and we've heard from him speak, I took away two TAC laws and those TAC laws was if you're sitting on a TAC, the first TAC law is the treatment is not to add bill every four to six hours.

The treatment is TAC removal and again, getting to the root. And I loved his second TAC law, which is if you're sitting on two TACs, removing one TAC doesn't remove 50% of the symptoms.

You have to treat all of the underlying causes, which is what you do. You look at, you know, infections, inflammation, what are also the immune modulatory things you need to do.

And then these patients have the remarkable recovery. So it is important to treat the whole person. Absolutely. And

two things along those lines. One is as we're talking about, this is an immune system disease.

It's a disease that where there's, you know, glial cell overactivation, cytokine overactivation. And often I hear from patients, oh my God, they have this infection, this infection.

No, they have an immune dysregulation. Yes, and that's important because most of these patients in our studies, we've seen they have multiple infections.

Usually there's one offending that triggers this molecular mimicry, but because of the immune dysfunction, they become susceptible to all of these other infections.

So typically you'll see them with multiple infections. And then also they can be susceptible to, let's say mold toxins, heavy metal, other things that they don't clear out.

And so then you've got this complex mixture of often it's challenging to know where to start unless, you know, the clinicians have training and understanding.

Research Beyond PANS: Heart, Alzheimeru2019s, and Autism

And the other thing that came to mind when you were saying that is I always say the gut is not the second brain, it's the first brain. And I've heard you talk about exposing our children to dirt.

And one of the things that I talk about a lot that in developing countries, there's not the level of autoimmune disease that we see in our country and our listeners have heard me talk about it so many times.

Can you just address that a little bit too? Yeah, you know, autism now, what is it? One in 31 now. And, and so there's a lot of things that contribute to that, but I believe that there are certain things like we call the hygiene theory.

Uh, and we, we do protect our kids, which is important from eating dirt or doing anything or, you know, they drop their pacifier, it's sterilized. You know, you don't just, you just stick it back in their mouth and you know, that's good.

The five second rule. But what we find is that. You mentioned the gut, which is required to have these bacteria inside of us. If they are not there producing the different types of chemicals and the compounds that are there, that they do not help keep our system healthy.

So, you know, there is the, the dirt cure book. I don't know if you've read that. Oh yeah, definitely. Well, you know, interesting just by having her child go back to, you know, growing vegetables in the garden, maybe washing them off, but don't, you know, sterilizing everything.

And his gut began to heal and it began to just improve dramatically. And I think one thing that's important in this gut brain connection is the same endothelial line, the junctions between the endothelium in the gut and the blood brain barrier, which is also the same endothelial lining.

When you heal one, you're healing the other. So if you think about it, in order for something to get past the blood brain barrier, they have to be able to have a leaky system, which is why you see leaky gut, leaky brain, because the same end of the aligning, it is no longer functioning properly.

So all this back to, you know, healthy lifestyle, healthy diet, getting these probiotics and whatever it is, however you introduce them, get back to healing the gut.

And Auckland, that's the first step in getting them started on the path to recovery. Yeah. And, and I so agree with you. I mean, we have to keep in mind that anywhere from 20 to 36% of our children may have a restrictive eating disorder and may well have, you know, due to contamination fears, choking fears, real OCD.

piece of their puzzle. And we have to be conscious of that when we talk about changing their diet and all of that. I work at a medical center in Brooklyn, which is, you know, bereft of any dirt, bereft of any parks or anything else in this little microcosm.

And I'm always talking to them, get a window box, get a plant box, a vegetable garden in your apartment, in your home, because children need to be in the dirt.

They need to be diversifying their microbiome. And I think what you're saying about the leaky gut, leaky brain is so important for people to understand.

Yeah. Well, you're probably aware of that study that was published looking at the spectrum of bacterial species in autistic children spectrum of bacteria in healthy children.

And the study found that not only were there different species in the autistic children, but it was a narrower group of these than was in the healthy group.

Right. So this speaks to this microbiome issue, you know, we don't know what we don't know until we really recognize that there's other things going on that contribute to this complex disorder.

Yeah, absolutely. And some of the work by Sabine Hazan in COVID, you know, also shows us how COVID impacted the gut. And I was thinking about COVID a little bit more as you were talking too, because one of the things I think that we unfortunately did was we increased a lot of our kids' anxiety.

contamination fears and isolation and that cleaning and whatever may have been necessary. I'm not putting any judgment on that, but I think we did a disservice to the diversity of our gut in how much we reinforced that necessary cleanliness.

Yeah, there's some element of protection, but then there's the recognition of when you can go over for it. And you know, the aftermath of that, if you look at the, you know, even in the UK, the resurgence of scarlet fever from streptococcal infections was just amazing because, and we saw RSV resurgence, we saw influenza resurgence, all of those.

And then you recognize that there hadn't been enough exposure to the kids during this time. So you see this resurgence, but then also the mutations in these organisms allow it to be able to have opportunistic types of effects.

There are some long-term far-reaching effects of a lot of different things, but the important thing is to really diagnose and get to the root of whatever the disorder or the disease is, and then treat the root and so many more people get well.

Yeah, absolutely. Root cause medicine, that's what functional medicine really is. And I hope more and more of us get back to it. Now, the molecular test, the autoimmune panel, only practitioners can order that at this time, correct?

Correct. And the reason we do that is one, we want a patient to be able to have the ability to talk to their clinician about it and be able to have a plan as how to be treated because information is good, but if you don't know what to do with it and there's nothing to do, that's not good.

And the other, it also helps with our regulatory status because we want to stay within compliance, but we firmly believe that Having a good clinician who knows what to do with the results is so important to getting the patient well.

So yes, only licensed and prescribing practitioners can order the panel. And as you said, you do have nurses, clinicians that can help practitioners because we do have several practitioners who listen to this podcast.

You do have people on staff that help them understand it in case they don't have knowledge about the test. Yes,

absolutely. And in fact, probably half of our orders came from a patient telling their doctor, Hey, I've heard about this.

Gut Health, Microbiome, and Environmental Triggers

Here's information. And then we could follow up if they're open to it or put them in touch with an onboarding or a webinar or just a one-on-one. Because we're giving them help.

This is the reason we started the company. Kiddingly, but not necessarily tell. people were nonprofit organization, not because we intended to be, it's because the test doesn't cover our costs yet.

So we're doing it because we see so much significance of how patients would not be able to get well if you didn't do this. And we do believe because of that, that the other finances will take care of itself.

But yes, it is important that a doctor practices. And we also have several people from around the world that listen or watch the podcast. And I know it is available in England.

It's not yet available in Australia, correct? We actually do get specimens from over 50 countries. Some of them are a little more difficult. The clinician actually will figure out a way and we'll work with them to get it over.

But we do have AONM who help facilitate from the UK and around there. another group in Portugal and even in, believe it or not, Ukraine, and another one in Poland, and then another one we have out in Belgium.

Wow. That's great. Those are all places I've been to lecture, so we know they get the root cause medicine piece, so it's great that they're facilitating the use of this test there, because I think it is really important for people to understand that like arthritis, like diabetes, like thyroiditis, the brain has the itis, has the inflammation from these infections.

And if we can figure that out and treat it appropriately, these kids can get better. Yeah, and we believe that pandas and pans are very specific. So an adult can't have pandas or pan, but an adult can suffer from the same biological condition and be responsive to treatment in the same way.

So it's very important to make sure that they fit the criteria for a lot of reasons. It is also important to recognize it's a medical model that will help us understand so many other conditions and help in understanding how to better diagnose and treat patients who are suffering from these chronic illnesses that Yeah, and such a good point.

It does happen in adults. It's just the P is pediatric. Let's not call it that, but it is the same thing, especially if it was undertreated, misdiagnosed, missed entirely as a child.

But even with other diseases, it can still happen in an adult, even if it didn't occur as a child. Yeah, that's why I also like the usage of the term basal ganglion cephalitis.

There's been publications about Cunningham and others have shown. these antibodies will attack the basal ganglia and stain to it and certain receptors involved because that takes away the other components of let's say whether it's strep or not or whether or not it's a pediatric disorder or not and maybe even some kind of encephalopathy but I think terminology kind of gets us into the problem of you know denotation and connotation you mentioned items yes all that means is inflammation no matter what you attach to it but if you say encephalitis that is something that sometimes people think okay well this is not an encephalitis uh you know there's no viral or bacterial attack in the brain you know it's not a direct uh attack it's a secondary immune All that aside, I think it's just important to realize what's really going on and then treat the underlying root that you always talk about.

Yeah. Well, Craig, thank you for all you do to help so many of our families figure it out and get the help that they need. Are there any last words you want to leave our families or practitioners or any of our listeners with?

Just that there is hope and I know as we have talked to so many parents and so many patients who often have said

this is the first time they've ever had hope that either I'm going to get well or my child is going to get well.

And it is through perseverance and it is through recognizing and looking for the clinicians and physicians and the practitioners who know and understand and help like themself that they get their family better.

And we have just seen over and over again that don't give up. There are answers and your child or your family member will get better, but get to the underlying root, beat the root.

Hope, Root-Cause Medicine, and Closing Remarks

And you'll see that and we've seen that with over 16,000 patrons. So we do know that. And it's one of those areas that we hope will soon just be common knowledge.

And then it'll be the first thing people will think about and there won't be as many years to decades of searching for answers. Well, Craig, thank you for all you do.

I'm glad you met Madeline and I'm glad you started this company and for all you do in our community, both with the science that you present as well as the compassion you give to all of our family.

So thank you. And thank you Dr. Herr for what you do. I see your book back there and demystifying and I recommend that all of our practitioners because it's so well put together and it's very clearly laid out.

So thank you for what you do and we'll look forward to working together and helping more people. Absolutely. Well, thanks everybody for being here and thank you again, Craig, for everything.

That's it for today's episode of Demystifying Pan's Pandas. I hope you're walking away with insights, tools, and hope to help you and your child on this journey.

If you found today's conversation valuable, be sure to subscribe so you never miss an episode. Share this podcast with anyone who might need it. It could be the lifeline they're searching for.

And if you have a moment, leaving a review helps us reach even more families who deserve answers. Also, for more information, training, and community, check out our website, drohara.com, and join our annual membership.

And remember, every step forward, no matter how small, brings us closer to healing and understanding. Until next time, be present, be hopeful, and we look forward to seeing you next time on Demystifying Pan's Pandas.