No One’s Talking About This Lyme Risk… Transcript

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Introduction to Congenital Lyme and the Guest Expert

And so why would that be, like, after you've been able to modulate, you know, the inflammatory cytokines, after you've been able to, you know, get the infection out of, let's say, the endothelial cells for those that like to live there?

Why? Can you explain to our listeners how you would still be primed? Right. So again, with a fetal brain and like, you know, a full-term baby or adult brain, those structures are trying to mold and develop as they're being exposed.

So we get these very different pathways, many that we know. And interestingly, there are many that we don't know. We don't know long-term effect. of what will continue, understanding that the brain is still pretty plastic or moldable in children, but we do have this set of development neurologically that is occurring under a great duress.

And it's very tricky to understand sort of that really that long-term effect of it and what we're truly going to be able to mend. This is Doctor Talks, real talk from real doctors on the issues that matter to you most.

Welcome back to Lime Bites, rebuilding immunity, reversing inflammation, and redefining recovery. Today we're exploring a critically overlooked but increasingly urgent issue in the field of complex chronic illness, the silent transmission of Lyme disease and other vector-borne pathogens from mother to child in utero.

Despite decades of research confirming transplant central infection for pathogens like Borrelia, Bartonella, and Babesia, congenital transmission remains largely under-recognized in conventional clinical practice.

This oversight leads countless families without answers and children without adequate care. Joining us is Dr. Summer Delsignore, a board-certified pediatric provider and internationally recognized expert in integrative pediatric medicine.

With advanced training under Dr. Richard Horowitz and Dr. Kenneth Bach, and as a member of ILADS and MAPS, Dr. Sommer blends the best of medical science with whole-child healing.

She's also the creator of the groundbreaking Reset Protocol, designed to uncover and treat the root causes of pediatric illness. In this episode, we'll unpack how stealth pathogens cross the placental barrier, why certain pregnancies are more vulnerable and what the long-term consequences may be for children exposed in utero.

We'll also dive into diagnostic challenges, ethical dilemmas and the public health gaps that continue to leave families in the dark. This is Lime Bites.

Let the true healing begin. Welcome, Dr. Del Cignor. Thank you so much for joining us. Thank you for having me, Dr. Hinchey. Nice to meet you. Absolutely.

Tell our audience a little bit about why you got into doing what you do. You know, I was practicing primary pediatrics more than a decade ago, and I saw the pattern of events with children coming into the office with sort of nondescript symptoms, and they wouldn't get better.

They weren't healing. And we would notice that every time we treated certain infections for perhaps a behavioral component that there would be this remarkable kind of reset generally with their behavior, their general constitution, you know, cognitively they would improve.

Why the Speaker Entered Pediatric Root-Cause Medicine

And so I just started asking the why questions like anyone would and kind of led me down this rabbit hole to say,

hey, there's a better way of doing this than traditional medicine would have us practice.

And this is about the time that I connected with Dr. Horowitz, who was a wonderful resource and essentially kind of gave me the landscape of what he was seeing in practice and where the lack of treatment and focus was with children.

and started training with him. So it was really just having that healthy curiosity and wanting to heal those children from a root cause perspective rather than having them coming through that revolving door in the office constantly on medication and not healing, generally.

So that's kind of what led me down this path. And then here we are today. So what factors would you say really play a role in whether or not a pregnancy is going to be overly vulnerable to the presence of any of these stealth infections in the mother?

I think there are many factors involved. First, you know, really understanding the mechanism for Borrelia and how it would migrate is important to know.

So we're looking at things like Borrelia bructoferri. So it expresses these outer surface proteins that kind of enable it to tether to the vascular endothelial and it binds to these extracellular matrices.

There are certain lipoproteins that allow it to adhere to that endothelial cell under a slow condition. And then the mediation occurs where it's tattering to that endothelial surface.

It's then using a dragging motion where the spear can move along a vessel wall. It'll adhese to a transendothelial cell to migrate in. And this allows it to exit that bloodstream and then colonize the target tissue if it's joint or heart or brain or the placenta in this case.

So we have that occur. And then you have this, after this adhesion occurs, the migration through a, again, another, you know, sort of a, I guess, a modulation-type component occurs when there is a slit between those endothelial cells and the tight junctions there, and a host modulation, which is key, so it's going to kind of trick the immune system.

How Tick-Borne Pathogens Cross the Placenta

So from a placental implication perspective, you know, Lospenta is very important, and it contains rich endothelial networks, a lot of fetal capillaries, maternal vasculature, and during a mother who may have bacteremia or other underlying infection that could be low level, you get that same kind of transmigration of that bug across the placenta, and that can easily reach the fetal circulation and potentially infect the tissues.

We have many, many cases of this occurring document of Borrelia DNA or spheruketes in the placenta and fetus. Unfortunately, it's during an autopsy tissue sample that we see it.

looking at things like Bartonella and Babesia, other vector-borne illnesses, you have a lot of the same major concerns. Bartonella will also invade the endothelial tissue and red blood cells, but it uses a secretory system to do so, but very similarly kind of hijacks that cell and uses it as a vehicle to travel in.

where, and then kind of looking on the flip side, like a babesia is not actively invading through the endothelial cells, but it's actually invading that red blood cell, which again, we know that there is that matrix within the placenta where it's easy to share any kind of pathogen from a mom to a baby.

And I think that really the inflammation is the major condition that you would see as a big vulnerability for a mom because you get that increased vascular permeability, you get disruption to that barrier, and nipple scintillation is a really great barrier.

And then that also helps to facilitate that transfer of an affected red blood cell or anothelial cell or free parasite into

that baby from mom to baby.

You also see sort of early versus late pregnancy, which is important for everyone to understand. And you probably understand this quite well, too, with the immune system and that whole teach one, teach two dominance.

Early in pregnancy, we're very inflamed. We have this very teach one dominance, and this is supporting implantation. They use the placental invasion kind of remodeling, tissue remodeling to kind of provoke it to kind of grow.

And you've got a lot of vulnerability at that time for the fetus because it's not protected, because you don't have this full, fully developed placental, you know, kind of fortress for that developing baby.

You shift into sort of second trimester and things settle out more chill, more anti-inflammatory, let the baby grow, feed, you know, develop as it should.

And then later in pregnancy, sort of late pregnancy, third trimester, later in the third trimester, you get this more anti-inflammatory switch to pro-inflammatory again to kind of prepare the mom for birth.

And so then you get that rise and all those inflammatory markers and you get sort of that, you know, the defense of the placenta, if it's mature, is improved, but inflammation still can affect it and affect the fetal brain and that amine priming.

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So join us at Pompano Beach or virtually from anywhere. Register now at LymeBytes.com. That's L-Y-M-E-B-Y-T-E-S dot com. So if one were to acquire a new infection, it sounds like the most dangerous windows would be during the first or third trimester.

But what if mom has a chronic Lyme infection or Bartonella or Babesia and she doesn't know it? What's the likelihood that it would be passed? Is there a different sort of risk of passing it along, whether it's Borrelia or Bartonella or Babesia?

LymeBytes Symposium Promo

Do you think there are ones that are more easily transmittable? Right. So I think that with a mom is probably more susceptible. So what I do know is that that inflammation plays a huge role in the ability to transmit anything across.

And if mom has subclinical unknown infection and it's sort of humming along, she's already inflamed. And what's important is to understand is that if the pathogen itself isn't transmitting over and Anecdotally, and from some of the data I've seen, I feel that it's perhaps Vibesia is a little easier to transmit to the baby just because of those red blood cells.

But I think that the inflammation itself can contribute to a lot of the damaging effect that you see on babies, regardless of the ability of that pathogen to cross over.

But to answer your question, I think Vibesia, we do see about a 30% rate of miscarriages with moms who develop Vibesia. Well, it's a pretty significant number.

It is. It is. And not being tested often until post. Yeah. What are your thoughts on, you know, we're still looking for, you know, the acronym, right, the torch, right?

We're still looking for those things prenatally, you know, which chlamydia is one of. And, you know, I would argue most people know if they have chlamydia or not.

Like, why is conventional medicine not screening?

Transmission Risk, Screening Gaps, and Pregnancy Management

for things like Borrelia, Bartonella babesia, you know, when they can have, you know, when they can cause such an increased rate of infertility, you know, miscarriages, stillbirths, et cetera, like what is going on?

I know, I know, with perplexing, because we have so much data over the years to show that there is, you know, an infection rate that can truly affect a baby.

I think that it's under-reported generally. I don't think we have mandatory reporting for congenital Lyme, the VC of Artnella specifically. I think that, I know that we did have ICD-9 codes for it, I think maybe 2018, 2019, and then it was sort of silently removed.

So you couldn't track it. I think diagnostic limitations are there. Standard Lyme serology is not designed for neonatal or in-euro exposure. So we can test mom, we can test the corded blood, we can test the placenta after birth, we can continue serial testing of the baby.

But then again, a baby may already have an altered immune response and not show anything for many, many years. So we don't have a standard way of testing it to show it and show the correlation.

And with any kind of PCR testing or blood smear, half of the time the PCRs are negative in anyone who's chronic and or the level may be too low to capture with a culture.

And then you've got also that whole symptom overlap, clinical overlap where, you know, anemia and growth restrictions and stillbirths may be misattributed to other causes instead.

So, I think that those are the main concerns and issues that we see with that, is that there is nothing that really backs a provider who may want to help, who may not be comfortable in recognizing and doing diagnostics, but don't appear to have the backing to support them.

Right. So what would your course of action that you would recommend in a scenario where, say, a mother develops acute Lyme while she's pregnant, how would you suggest handling that pregnancy?

And of course, to all of the listeners and practitioners, this is not medical advice. You need to consult your own medical professional. But I mean, what do you suggest is the general course of action?

how would you try to protect the baby in utero for the rest of the pregnancy? And then, you know, what would you do for evaluation and testing on the baby once it's born or during birth if you're going to do cord blood or, you know, a sample from the placenta or what have you?

Right. Well, there are protocols that, you know, you certainly have to weigh the risks and benefits. And understanding that there's that 30% risk of, you know, a miscarriage or early term pregnancy, that's significant.

And so I think that we certainly should look at treating. I do treat moms along with their OBs, because I'm not an OB, and I certainly preface that to say, listen, I can't manage all of that for you, but Certainly, we can very methodically and cautiously approach this treatment to give you the potentially the best outcome for you and your child.

Certainly, treating all of those during pregnancy and again, weighing the risk of certain medications that are classified as dangerous. for them, and there actually are certain subsets of ments you have to actually, you know, leave alone that are hands-off.

And there's others that we can use. So you want something that's going to cross that placenta, which is counterintuitive to many, many people to say, oh, it won't cross the placenta.

We need it to. We know that that baby is most likely infected again, you know, first and third trimester. So there's the risk there. So there are, you know, you watch moms closely, you treat mom and baby, you do the best that you can to mitigate any inflammation and to help along understanding that inflammation is also a big contributor to the neurodevelopment, general development of that child.

So there are certain medications. I'm not sure we want to get into it. I think for the sake of this, we don't have to get into specifics. For anyone listening who's wondering, well, why not just wait till the baby is born to treat?

Why risk it when the baby is in utero and is developing? This is so not my specialty, but I would have to. I would have to imagine that this ongoing pro-inflammatory cytokine cascade, along with everything else that goes wrong once one of these stealth infections are in the body, it too is going to contribute to potential developmental issues with the fetus and the baby.

Absolutely, absolutely. And there are parents who do choose to treat afterwards, and it's completely their choice. It's their child. And that becomes more of the ethical dilemma with a lot of families is do we knowingly move forward with the pregnancy and avoid treatment because we're fearful of the potential knowing we have this high risk of miscarriage.

Or do we treat knowing that there's still a risk, you know, of miscarriage and or other potential issues later with that child as well? It's almost an incident.

Yeah. And then what do you think about the potential risk, you know, with having potentially the mother and baby be on, you know, if we're talking pharmaceutical antibiotics, you know, what that does to the microbiome and the vagina and therefore the microbiome that the baby's going to be inoculated with that really you know, ends up kind of being the basis for that baby's immune system.

How do we deal with that piece of if someone were to choose to do, you know, pretty aggressive pharmaceutical antibiotics during pregnancy? Right. And again, when the baby is born, that's the beginning of the battle.

So, we're just trying to prevent more of those congenital type conditions from occurring as we see that there are links with congenital heart disease.

We are seeing, you know, encephalopathy, encephalitis occur as a result, visual disturbances that may be permanent in nature. So, you're trying to prevent the bigger organ system failures and disruptions.

understanding that, yes, there's a good chance you're going to wreck the gut, and we're going to have to really rebuild from there. And, you know, there are certain probiotics you can give to moms, but there's some ones you can't touch while they're pregnant because it can become pathogenic for the child.

So you do your best, again, like I said, to mitigate risks. We know that there are certain herbs you can use, but there are some that we can't touch that I would love to be able to.

But it's a risky take. It's a risky take. So you really start early on aggressively rebuilding that gut and getting that,

you know, de-inflame, get the detox going and repair.

Breastfeeding, Newborn Evaluation, and Early Red Flags

It can be done. But again, the journey is just beginning. Are you suffering from Lyme disease or another complex chronic illness and aren't sure who to trust when it comes to herbal supplements?

Hi. I'm Dr. Mariah Hinchey, founder of Lyme Core Botanicals. As a naturopathic physician specializing in complex, chronic, infection-driven illnesses like Lyme disease, I needed herbal medicine I could truly trust.

That's why I formulated Lyme Core Botanicals. where our herbal tinctures are handcrafted in small batches right here in Connecticut. We use the whole herb, never isolates, to preserve the full spectrum of medicinal compounds.

Every single step from sourcing to extraction is done with precision to ensure maximum purity, potency, and consistency. These are the same herbal formulas I used to heal myself and have used for years to help my patients and family members heal too.

And now I'm making them available to practitioners and patients everywhere. Lyme Core Botanicals, herbal medicine you can trust from a doctor who lives this work.

Learn more at Lyme Core Botanicals. That's L-Y-M-E-C-O-R-E dot com. So say that you've done your due diligence during pregnancy, you've done the best that you can do with testing during birth, after birth, and you're still sort of coming up empty.

What would you say could be some of the initial clinical red flags, either in infancy or early childhood development that would lead you to want to work that child up again or maybe just treat them?

Right. So there are several symptoms that could kind of clue us in. Initially, it would be recurring pregnancy loss. It's so if we're going way back before mom is having a child to term to say, why?

If it's an otherwise healthy woman, why are they not carrying this child to term? Why are they having so many losses? So it would be great to start there in an ideal world.

if there are maternal febrile illnesses in pregnancy without a known cause. So if you're going through a torch workup, a classic torch workup, and you're coming up empty-handed with texoplasm and rubella and CMV and herpes and EBD and all those things that are classically involved, you kind of expand upon that and you look into other things.

You're looking for Zika, you're looking for Parvo, which we run that for everybody, now COVID, and then expand beyond that with the tick-borne illnesses.

But if you see things like enlarged spleen, enlarged liver, if there is ongoing intrauterine growth restrictions, that's a big one. If you're saying intracranial calcifications, and this could be on ultrasound, you could see that if, you know, after, you know, development, the head, so hydrocephalus, microcephaly, you know, any abnormality there, that's definitely a red flag for me.

you know, moms hearing baby to term if that baby is anemic has jaundiced beyond what we typically see for the cytopenia, failure to thrive kind of situation.

You want to look at those, you know, those key factors again with diagnostics. But understanding that it's challenging, especially with a mom whose immune system is a bit tilted during that period of time.

And then for a newborn, if we do make it to that newborn phase, the baby still has mom's innate immunity, so you're not really capturing anything from that child until the two or three months, at least, of age.

So it's really doing very thorough history-taking and diagnostics by some basics. and rolling out what it isn't and then making almost like an educated guess or clinical assumption that, hey, these symptoms appear to fit within, you know, these infectious disease symptoms, and we've rolled out everything else kind of scenario.

It's a challenge. It's a challenge. And so what about, do you worry that if there wasn't transmission, you know, trans placentally, that potentially it can be transmitted through the breast milk?

I've had a lot of people call and ask that question and it's like, it's such a tricky tricky question to answer, right? Because I feel like, you know, they're going to get so much from the breast milk and, you know, if possible to breastfeed, we should absolutely be breastfeeding for as long as possible.

Right. How do you deal with that? Well, what we know is that you do get spirochetes that will pass through breast milk. What we don't know is what level of spirochete load would it take to become pathogenic?

to mom and baby, or to baby, not mom, but mom and baby. And we don't really have that data to know. And there are so many, you know, beneficial factors in breastfeeding.

I don't discourage it. So I would definitely encourage it. And again, it's a personal choice. And we're going to deal with it. We'll deal with whatever is there.

I think that from an infectious transmission perspective, the baby most likely already has it.

Neurodevelopmental Effects and Autoimmune Mechanisms

I don't think the breast milk is going to compel the situation, honestly. Yeah, I mean, yes. It's a tricky question to answer, but again, I think there's so much more benefit.

Right. Agreed. So can you talk to us a little bit about how in utero exposure or transmission would actually cause, I guess, I don't want to use the word cause, but for lack of a better word, can lead to neurodevelopmental disorders like autism spectrum disorder, ADHD, speech delay, motor delay, et cetera.

Absolutely. So what we know, we have a lot of case reports and we don't have really large cohort studies. I know that Dr. Brian Fallon has a lot of research in this area and suggests that there are certain packagins that cross the placenta, reach that fetal central nervous system directly, and they can disrupt that neuronal migration, the way we myelinate, and the way that we develop synapses in the brain.

So that basic structure is affected from that direct impact. So Borrelia itself, Borrelia burgdorferi that causes Lyme disease, can localize in the neural tissue.

It can disrupt white matter development and activate the glial cells. We have things like Bartonella that will infect the glial cells as well. We see that in animal models and then infect neural endothelial cells.

And Babesia can cause a microvascular damage and that can reduce like oxygenation to the brain. So, infection during pregnancy can cause the systemic maternal inflammation as well, and that also alters that brain development.

So, we have inflammation and the bug at the same time. It's all happening. So, it can disrupt ongoing neurogenesis. It can cause the cytokine storm, essentially mom to baby, and you get this sort of cytokine-mediated sort of microglial kind of primary activation or sensitivity.

And then that's what's going to increase that risk for autism spectrum and mood disorders and then the other cognitive components, executive dysfunctioning, PANS, PANDAS, autoimmune encephalopathy, whatever you want to call it.

So that's kind of what you see. So expand a little bit and loop and, you know, I know you just touched on pan-

spandas, but like from an autoimmune perspective, talk a little bit more about how that pathophysiology works.

Sure, sure, absolutely. So with the exposure from an infection, you kind of get some of that mammary, molecular mammary that occurs. There's an immune priming perspective where the fetal brain immune cells remain sensitized to an exposure of whatever that might be from a vector borne illness.

And then that peripheral immunity will remain kind of at odds with an inflammatory shift. So you get this shift in balance from TH17, TREXELs, that sort of thing.

And then what happens from a molecular memory perspective is pathogens can then express antigens that are structurally similar to the neuronal proteins that we have.

And this generates like this cross-reactivity of antibodies. And this will then start targeting other structures of the brain, like the basal ganglia, dopamine receptors, tubulins.

It'll trigger that neuropsychiatric symptom, OCD ticks, anxiety that we see with that auto-nyosophilopathy. you have this persistent immune activation, even after the bug is cleared, technically, if there's something acute.

If it's a cleared or in a very low level, you still see that persistent immune activation. And we know that there are certain established bugs that are on that torch, you know, list of testing to do CMV, HSV, Toxoplasma, but also we add to it Aurelia Bartonella.

And, you know, so we have this inflammation, we continue to promote autoantibody formation, continues to drive T-cell dysfunction, and it's just sort of this cycle that happens.

I know that Dr. Suido proposed this to hit hypothesis where these things are happening congenitally from infection in utero, from the inflammation, and the fetus is sensitized to that immune system and their central nervous system.

And then later you get sort of the second immune challenge, the second hit, whether it's an additional viral infection, an additional, you know, vector-borne strep, what have you.

And then you see that sort of cycle continue with that neuro-inflammation-driven autoantibody production scenario that we see with auto-mancephalopathy.

And then do you see this ever progress like into seizure activity, epilepsy, things like that? I do. I do. It's interesting. There's a few pathways that involve that.

There is definitely the direct infection like you would see with other non-controversial type vector borne diseases where you develop this blood-brain barrier disruption, the cytokine storm, microbial activation, cortical irritation, and then you get all these misfiring situations with neurotransmitters and the inflammation, and then it blocks certain receptors.

So it's the perfect storm for an encephalitis. which can then lead to seizures as secondarily. There's a trickier one where you get this more immune-mediated autoimmunity, and it's some tick-borne pathogens will trigger that autoimmunity, autoimmune antibodies against that central nervous system antigen.

And you're talking about certain receptors, again, dopamine receptors, myelin antigens, NMDA receptors, and they can mimic also that autoimmune encephalopathy or encephalitis.

you get these refractory or pneumon-set seizures as a result of it.

Seizures, Blood-Brain Barrier Damage, and Brain Priming

So you'll see elevated autoimmune markers like acanthiamase if you do a Cunningham panel or some of the other testing that would look for autoimmune antibodies, auto antineuronal antibodies specifically.

And then another pathway that can occur from a seizure perspective is a vascular involvement, almost like many strokes in kids. So you get this vasculitis that can occur.

And you see it with a lot of more intracellular-type bugs like rickettsia. We see it a little bit with Bartonella, a lot of babesia. So you get this cerebral vasculitis and micro-infarcts, and then you get these focal seizures or kind of epilepsy-type scenarios.

And as a result of that, depending upon the bug, so let's say babesia, that can trigger things like hemolytic anemia, you get these shifts with electrolyte balances as well.

So it's sort of coming at you from all angles. And, you know, the seizures typically are not responsive to typical medications that you would see. It doesn't follow that path, that, you know, that voltage pathway, you know, voltage gate pathway that you normally see.

So, we're looking at treating more with anti-planetaries to reduce a seizure. So, it's counterintuitive to say, hey, give a monkey turn or a celery. You know, to help it versus, oh, we're not on, you know, gabapentin or we're not on other, you know, medications that aren't seen to be coiling it.

So, interestingly, From all those exposures that could contribute to that seizure, that microglial priming and remolding has completely changed that neural circuitry of that system.

So this can occur after E. coli pathogens, after colonic inflammation. That person may always be susceptible to some sort of a seizure-like pathology.

And so why would that be, like, after you've been able to modulate, you know, the inflammatory cytokines, after you've been able to, you know, get the infection out of, let's say, the endothelial cells for those that like to live there?

Why? Can you explain to our listeners how you would still be primed? Right. So again, with a fetal brain, unlike a full-term baby or adult brain, those structures are trying to mold and develop as they're being exposed.

So we get these very different pathways, many that we know. And interestingly, there are many that we don't know. We don't know long-term effect. of what will continue, understanding that the brain is still pretty plastic or moldable in children, but we do have this set of development neurologically that is occurring under a great duress.

And it's very tricky to understand sort of that really that long-term effect of it and what we're truly going to be able to mend. Right. So I mean, one would reason that the longer someone has had the infection, and I mean, this is true, obviously, for anyone, but especially in kids, the longer they've had the infection because their brain is developing and growing and making these neural networks, that potentially the harder it can be to reverse some of that.

And it's funny because I always say, you know, kids are the easiest to treat because they don't have the baggage right, that adult we have. But on the flip side, when an adult brain has already developed, right, and the blood brain barrier has been somewhat sealed, you know, so it's interesting.

You've painted a different picture in my head than what I've thought about before. So I love it when that happens. You know, why don't talk to our listeners a little bit about the blood brain barrier and how it's affected in all of this.

So the blood-brain barrier is essentially a junction of very tightly woven epithelial cells that prevent infections and other type of pathogens and toxins and things from crossing over.

The brain is an immune privileged area, other, you know, highly high volumes of immune path, you know, immune markers there because we don't want to seize every time we have a cold or have a fever.

So we don't want it to be highly reactive. So we have to have something in place to protect it. And that would be that barrier. And as long as that barrier is intact, We're golden.

You don't get crossover. It's a nice fortress. But when you have some of the stealthy bugs, like I mentioned earlier with Borrelia and how it can attach those cells and how it can create inflammation and kind of loosen the junction between those epithelial cells, you get sort of this pathway in.

And then once they're in there, again, it's immune privilege. So then there's really nothing there to really combat it, say like if it were systemically outside of that area.

So, once the integrity of that blood-brain barrier is breached, then many, many things can pass through. And the situation is that if you have inflammation there, inflammation begets more inflammation.

And it's sort of this vicious cycle until you're stopping it from treatment or, you know, in some ways trying to kill off that bug or reduce that inflammation to tighten it back up.

And my blood-brain barrier, as you know, too, is pretty fluid and or vulnerable. And many kids up until they're, you know, well into college or out of college, up until their 20s.

Yeah. So what sort of misdiagnoses do you see? Oh, goodness. So if we're looking at congenital infection, the misdiagnosing is usually from a neuropsychiatric component.

You see a lot of autism spectrum, ADHD, autoimmune encephalopathy or PANS, PANDAS. I know people use those words interchangeably. Mood or psychiatric disorders, seizure disorders.

And the symptoms are not specific enough. with many pediatricians who lack exposure to vector-borne illness patterns to, you know, trigger them to think that it may be something else.

So they're looking at symptom-specific and, you know, the congenital infections right now from a vector-borne perspective are not in the standard torch testing that's out there right now.

So that isn't that next step for many people to think about. Right. So what do you think can be done? What systemic changes need to happen in our health care to try to turn all of this around?

I mean, obviously, screening prenatally, right, it would be huge. None. You know, if I had my way, I think every vector-borne disease, whether you're talking about a pediatric patient or adult patient differential diagnosis, like, it should be in there, right?

It should. Yes, it should. Absolutely. So, yes, I do think surveillance is a key. I think we need the right diagnosis codes to be able to track it. And that comes back to something political that is far beyond me at this time.

But we definitely need those codes so providers can put those in, we can track it, we can see those trends. I think that we need congenital tick-borne registries, you know, that we can also monitor and use this as educational tools and find the gaps in medical care.

Misdiagnosis, Systemic Change, and Ethical Challenges

We have policies for vector-borne diseases. Mosquitoes seem to be much less controversial because we have it for Zika, you know. And there's even things for syphilis, which really is similar, but there's lack of a mandatory maternal screening protocol.

And, you know, I love to see some pretty robust screening protocols prior, so during family planning, if possible. to say, hey, let me put this on their radar, and have you ever been exposed, and let's get some testing, and here are the risks and benefits associated, and here's what you should do if you're bitten by a tick, you know, during your pregnancy.

I think we definitely need to do all of the screening and education prior. And then from a community outreach perspective, I think that we need to educate our OB, GYN providers, our midwives, our duals, our pediatricians, you

know, generally, and really beef up the education in those endemic areas.

Interestingly, over the winter, I hosted a kind of a coffee talk and dinner. I was prepared to wine and dine any OB who wanted to attend. to think at the table.

I don't have any major agenda other than to get an idea and get your sense of tick-borne. Where's your head? You know, how do you view this illness? And I couldn't get one OB to the table.

Oh, you're kidding me. And the response was, and we kind of did some door-to-door, almost like troubling salesmen. Hey, come on, this is really a big deal.

No one's going to have you on a microscope. I just want to talk. And they would say, no, we don't have a problem. We don't have a problem with that. Well, you don't because you're not screening.

You're not testing. Right. You don't know that you don't have a problem. You don't know that you don't know. And you're only seeing, you know, your only interaction with that baby is during the pregnancy.

There's no follow up, you know, afterwards. Right. They don't see it. And then I think, you know, we need the funding for that translational research because we talk about, you've mentioned it, I mentioned the data.

The data is anecdotal. It's case studies. Right. It's case studies from providers willing to put it out there, but we don't have that translational research to say, hey, Animal models show us this.

We see it time and time again in mouse models and things like that. But translating that into research with moms and babies, we don't have large cohort studies to be able to really have some teeth to it and feel confident about it and develop tried and true diagnostics to really hone in there to identify it.

Yeah. So how do you deal with the ethical challenges, you know, in dilemmas when it comes to treating a child that you suspect, you know, having a tick-borne or vector-borne infection and not having, like, this standard of care to fall back on?

And I mean, there's this standard of care, but it's, you know, it's atrocious. It's part of the reason of why, you know, everything is can lead into a chronicity state.

Right, right, exactly. I mean, there is, you know, a great deal of parental uncertainty that you definitely have to overcome. There is an emotional component to it, especially if you're suggesting that it's congenital.

There are many moms who will feel extremely guilty and blame themselves, and you see all of those cycles of grief. They're angry, they're sad, you know, they're And here we're presenting this without any true confirmation most of the time.

It's just a, hey, clinically, this is how it's setting out. I think that there's a huge medical liability as a result of that lack of surveillance and policy.

and innovation with diagnostic. So medical providers, it's a big challenge when you're recommending a treatment based on a clinical suspicion alone. If you're not one of the lucky ones to have a positive test, to put your neck out there and say, hey, we're going to treat your child with long-term antibiotics and herbals.

And we may make them miserable for a period of time, but it's okay. They'll get better, you know, and trying to convey that to them and then, you know, maintain some level of evidence-based practice, you know.

And so it's a challenge. But we're looking at a lot of gray zones between, you know, what we see in experimental science and research and then what we're seeing in actionable diagnostics and clinical care.

It makes it tough too because I have a lot of patients who, you know, when they go back to their pediatricians, you

know, they're still dealing with pediatricians that say, you know, there's nothing that you can do even about autism.

You know, it's just, you know, you were born with it or you have this regressive case and it just, it is what it is. And then, you know, every other symptom that kind of comes about, it's like, well, that's normal for children on the spectrum.

And it's like, no. I mean, it might be seen often, but that doesn't normalize it, right? We shouldn't just be expecting these things. And so it's challenging to be offering another explanation and solution when a lot of other pediatricians are actually saying, no, these chronic vector borne diseases don't exist, and they have nothing to do with the diagnosis.

All right, absolutely. And then if you just look, common sense, looking at the rate of autism, it's in epidemic proportions from 2010 of 300% or more.

I've lost count. And that's just not how epigenetics works. That just isn't how it works. So we're looking at either larger burdens of toxins and infectious disease exposures.

and inflammation from those toxins. I never bought into the, well, we're just surveillance. Our surveillance is better. Don't trust that. We've been surveilling for years and we continue to have the climb every year.

We're seeing these increases. Yeah, I don't believe that. I don't believe that autism is a static condition. However, we do know that there are some people who are genetically predisposed and just the luck of the draw, yes, perhaps they are without any other infection or any other insult, but most of the times the kids have layers that we can peel back and improve.

Yeah, I often say, like, straws in the camel's back. And, you know, each straw that you take off allows, you know, the child and the immune system and all of the systems to kind of come back one more step.

Right. Exactly. Exactly. So our bodies are these wonderful things that can heal themselves if you give it the opportunity. And especially with children, again, that brain is still developing.

well into their early 20s, and we joke to say, hey, that's why kids or young adults in college do kind of some really stupid things because their friends are a little bit on it, exactly.

I wonder if I'm two, I did it too. And so we still have opportunity to affect and improve upon that molding and upon that development neurologically. So not all is lost.

Closing Thoughts and Episode Outro

Now, if you're still here, there's always something that you can do, always. I believe that no matter what age you are, if you're still here, there are interventions.

Usually, there's something missing that your body needs to function properly, or there's something there that's causing your immune system to go haywire.

And oftentimes, it's both. Right. Agreed. One leading to the other. Thank you so much for being here with us. Do you have any sort of last words of wisdom that you would like to share with our listeners?

Yeah, thank you for having me. I think that just, you know, always be vigilant and never give up hope, and always ask those why questions. You know, once you get to the root of something, things seem to fall in place.

From a treatment perspective, clarity comes, and from that, you know, you certainly will develop a nice pathway of healing, whatever that looks like for you.

Yeah, and it's different. It's all an individual journey. Yeah, absolutely. Well, thank you for being here. So for all of

our listeners at home, if you would like to hear more from Dr.

Summer Delsignore, you can check her out at the Lime Bite Symposium. She's going to be doing a full presentation on congenital vector-borne diseases, how to diagnose, what you can do about it.

That's November 14th and 15th in sunny Pompano Beach, Fort Lauderdale, Florida. So you can join us virtually or in person. And I just want to thank you for listening to Lime Bytes.

And I hope this brought you one step closer to true healing. If it helped you, please share it with someone else who needs to hear this too, because together we all heal stronger.

We'll see you next time. Bye-bye. Thank you for tuning in to Doctor Talks. We hope today's episode has enlightened and inspired you on your path to optimal health.

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