Introduction and Dr. Bajnathu2019s Background

Welcome to Mold, mycotoxin, and Chronic Illness Summit. Today we get to speak with Dr. Anil Bajnath. He's board certified in family practice medicine and functional medicine anti-aging.

And he's the author of The Longevity Equation and the faculty at George Washington School of Medicine. He's an expert in worker testing so much more and really just has this fascination with precision medicine, really using the latest technology to help each individual.

Thank you so much for joining us. Oh, thank you for having me. It's a pleasure. And it was just like this very small snippet of your bio. It's quite impressive.

Yeah, I like exploring things. Needless to say, you know, and the journey for me began so many years ago, you know, almost in my teenage years exploring, you know, dark field phase contrast microscopy and looking at on the nature peripheral blood and studying with a European biological medical centers.

And and really, you know, this narrative of these different mold illnesses and fungus is a is a real thing. And yeah, it's interesting to see how the science and technology has been, you know, kind of translated and expanded over the years to bring us where we're at today.

So I'm really excited to unpack some of the information with you today. Thank you. Yeah, I really love how everything's coming together. Like, explain some of the symptoms we see with Mycotoxins mold illness and then really understanding.

And it's such a detailed level of what's actually happening in the body all the way down to gene expression. The transcriptomics. I thought that they share a little bit more about your story, how you got here, and then wrap that into what Transcriptomics says.

Oh, cool. Yeah, No, thank you. Thank you so, yeah, you know, as you mentioned, you are involved with some of the research here at George Washington University and faculty, the course director of the omics of Medicine for the Fellowship in Integrative Medicine, where essentially I train other providers and fellows to connect the molecular dots from genome to path of genome and a part of that translational science process is the use of what's known as Transcriptomics.

And essentially what Transcriptomics is, is it goes back to the central dogma of biology that we all learned about in, you know, our science courses in high school, which is DNA into RNA and RNA into protein.

Transcriptomics and the Biology of CIRS

So when we're looking at mold illness, it's just very fascinating because again, with my story, you know, I got into a technique with that dark field phase contrast microscopy, as I was alluding to, and I came across different levels of research and publications from various physicians and PhDs that actually identified these different anaerobic fungal forms and peripheral blood.

And there's been some research to further quantify this and identify this with immunohistochemical staining. And I've identified signatures and patterns consistent with Aspergillus mucor and Candida within peripheral blood, but that doesn't necessarily translate to a true fungemia But more so I believe this could be coming from, you know, colonization and a dysbiosis within the gut that's bleeding over and making its way into circulation, thereby influencing the blood microbiome or the blood fungiome or fungi-ome or whatever.

And we're able to see some of those different factors. Furthermore, if my interest in omics led me to pursue training

with Dr. Andrew Hayman, who's my mentor at the University at George Washington University, and I learned about CIRS or chronic inflammatory response syndrome many, many years ago and was completely fascinated by it because there's always a subset of the population that is no matter what you do, you could balance your hormones, support their adrenals, get them on, you know, all sorts of supplements, and they're just not responding to these different interventions.

And it makes you question, what am I missing? And I really feel as though that that missing link is, you know, further defined by cirs, medicine. And as you know, this is a chronic, you know, issue that will lasting greater than six months that pulls into into the mix.

Various levels of the immune system are at the heart of this. You know, there's various inflammatory mediators from both th1, th2, th17 factors and with additional associations with coagulation, you know, being involved in this this equation as well as complement activation.

And ultimately, you know, at the heart of medicine there is a innate immune response to an antigen that's detected that is not being presented to the adaptive immune part of the immune system.

So at the heart of this, you know, it led me to training with Dr. Andrew Hayman. And again, he and Dr. Shoemaker are the ones who have, you know, kind of done all the research and brought me into the circle.

And, you know, we're we're leveraging the use of transcriptomics in clinical practice and essentially, you know, to further kind of illustrate what what that means clinically.

I would say, you know, if if you were to look at the transcriptome, I would make the analogy that if your DNA were the instruments to your own personal symphony orchestra, the transcriptome is a harmony coming off that instrument.

Certain things could be upregulated and red within a test or downregulated in blue, within the transcriptomic GENIE test. And essentially this is looking at how that environmental slash genetic interface is influencing DNA behavior.

So certain things are going to be upregulated. Downregulated based on these environmental stressors and responses and your body will, you know, kind of sing a very unique tune based on these various environmental stimuli.

So that's kind of what Transcriptomics is. And we use it to further define and map out some of these unique molecular patterns associated with with CIRS or chronic inflammatory response syndrome.

And as it relates to bio toxin illness. And what we have found in our research is that essentially 42% of people that are grappling with sirs are actually dealing with something called actin or myositis, which is a bacteria that behaves like a mold.

More specifically, it's a gram positive facultative, anaerobic spore forming filamentous bacteria that is very ubiquitous in our environment and can be colonized in a water damage building and within our skin.

So that's 42% of what we're seeing here based on the molecular signature with GENIE that is involved with mediating the process of CIRS. Additionally, other 38% is that we see is is dealing with endotoxins and these various forms of inflammasomes and only 7 to 10% of our studies are actually have a true signature for mycotoxins within our clinical presentation upstairs.

So 42% actino 38% ish endo and 7 to 10% mycotoxins we see as influencing cirs. And again, I. Just want to pause here because what this tool does then is as you're working with your patients, it really helps you to define what treatments that are necessary next, because you do different things that are just showing the mycotoxins versus the endotoxin versus the actinomyces Yes. And keep in mind, this tool is meant for research use only.

So it's not intended to like really diagnose or treat, but it does it does provide us with additional information that

points us in directions that might have been obscured through other forms of testing.

So you have good ways to to look for the endotoxins or for the actinomyces And I know there are ways to look for it in regards to actinomycetes you could identify throughand via biomarker testing, you know, the company and via biomarkers tests in.

A certain amount, but not in the body.

Actinomyces, Environmental Testing, and Legal Considerations

Right. In the environment, Correct? Yeah. And they also have the skin colonization test as well. Yeah. So there's what we find is that a lot of people are actually colonized with actinomycetes on their skin and the actinomycetes is actually releases several different forms of toxins, including of a medicine.

And mycolic acid. And this actually activates complement and pokes holes in the cells and triggers this whole inflammatory response. So the way we kind of map out actino is through a company called EnviroBiomics and we look at what's known as dominance index and prevalence index.

And and that gives us some further information at connecting the molecular dots from, you know, the transcriptome to the entire biome, so to speak. Yeah.

And so let's explore this actino a little bit, turn this a little bit more and then come back to the just the other areas because we really haven't covered this on the segment so far.

And I think it's a really important piece that I want people to know about. So can you just walk us through the thought process that you have of like next steps and what you would recommend for patients when they come back for the positive Transcriptomics on the gene test for actin or Mice's.

Yeah, and that's going to be very specific with what's called TGF beta receptor signaling on the on the GENI test along with map kinase signaling. So, you know, there are these different secondary messengers that are upregulated in response to the actin and meiosis specifically and this unique signature.

And essentially, you know, again, this is bacteria very ubiquitous in the environment, especially in water damage buildings. As we know, these water damaged buildings are very poorly microbial in nature.

So it's not just, you know, mold that we're seeing. We're seeing all sorts of fun things within this colonization and this bacteria that releases this off gases.

It's volatile acid and gives, you know, a very weird smell. So typically you see mold, you smell active. No. And so depending on, you know, the amount of areas within the home, there's a very specific cleaning protocol that needs to be followed in regards to even vacuuming the mattress with a HEPA filter vacuum twice weekly, washing your sheets twice weekly.

There's skin, decolonization protocols out there as well that that are being explored right now to help decrease the microbial burden that when it's when it is has a higher human habitant index, so to speak.

And so it just really depends on what we find there clinically. So it's very interesting to see, you know, sometimes again, what we see is, you know, the genetic haplotype correlates to the protein, the transcriptomic signature with the proteomic signature with the and via biomarker signature.

And we connect those molecular dots, so to speak, and see how it all kind of translates. So yeah, there's very specific cleaning protocols for the extent of my OCD.

Which a lot of times I think gets missed when we're just looking for the mold. Correct? Correct. This is really important. And, you know, to speak about, you know, some of the again, I deal with some of the legal cases tied

into sirs and water damage buildings.

And, you know, this could be a very tricky area of medicine. And so there's a lot of legal factors that come into play. And, you know, you need to have good data.

And unfortunately, you know, it's it's it's looking at some of the urinary mycotoxins tests. You know, that's not going to be, you know, clinically applicable in the court of law.

It's not FDA approved. And if that's even brought into the equation, that actually could vary your case because it's it's not a FDA approved test. And there's a lot of criticisms of the urinary Mycotoxins test versus some of the more objective data that we're able to get that can't be argued or refuted comparatively.

Data is that. That's going to be the combination of, you know, the different environmental testing and then, you know, walking through the the research behind surge medicine.

In regards to the HLA haplotypes, we know that 22% of the population carries with it a very specific haplotype that predisposes them towards these environmental hyper sensitivities.

And then, you know, again, making the appropriate case diagnosis for SIRS. As you may know, it's clearly defined based on the 28 GAO and 2018 consensus for what is defined as a meaningful exposure.

And we know that there's these 13 symptom cluster categories and you need eight out of 13 symptoms. There's also the use of the visual contrast sensitivity test, which is, you know, also pulled into the mix for for diagnosis.

And you would need three out of six objective biomarkers to further define, you know, a SIRS diagnosis and as you may know, there's multiple papers published on this over 25 publications with over 4000 patients that, you know, kind of really give this a little bit more credibility in regards to the legal aspects.

We're dealing with a mild illness, so to speak, or sirs as a whole. And this has been further validated with two randomized controlled, double blind, placebo controlled trial study publications as well.

So the research is is starting to catch up to what we're seeing and observing here clinically. And if you're ever pulled into any of these legal battles, you know, it could be very challenging.

And making sure you document things linearly and objectively is is really important when one taking on this kind of issue clinically. Yeah. So I really think for people listening that do feel like they have a lawsuit involved that so important to find a physician that's familiar with how the law works because it's a whole different approach than if you're just going to somebody to get well.

I think the efforts behind it to be successful in a lawsuit, I think somebody like you who can navigate the legal system and know what's expected is important.

Yeah, there's a lot of moving parts and variables with it. Yeah, that's going to be changing, you know, as our science gets better with the micro text and testing, a lot more studies are done in that area too, because not everybody who's sick or old has the full service diagnosis.

You know, it can show up a lot of different ways, but that certainly if you have have a service and, you know, diagnosis and there's something legal going on, you know, that you want the right support for it.

And yeah, yeah, yeah, because just getting better. But it's a lot. It's definitely a lot. And I think, you know, you definitely hit the nail on the head because sometimes it's not a full blown sirs, but you know, maybe there's historical exposure that's leading to that, you know, immune system priming that never got resolved.

You know, So at the heart of this is, you know, a dysregulated innate immune response that didn't have the ability to kind of put out and extinguish that fire.

So to speak.

Cell Danger Response, Metabolism, and Weight Gain

And as you may know, there are more than 30 different entities that make and trigger sirs, you know, including beta glucans, man ns virus, cyclic dry and dry amines LPs actin hateful fragments, cell wall fragments and Botox ins and boxes, mycotoxins, immolations, all sorts of things that could be found that kind of, you know, drive this immune response.

Which makes sense are so many things in our bodies and in our environments that the immune system is trying to work on and then creates this collection of symptoms and biology.

Correct? Yeah, it's very fascinating. So let's dig into some of those other aspects of biology. I know, especially with the gene tests, you can get a much better indication in the whole type of metabolism that's happening.

The cell danger response and physiology. So let's let's talk a little bit about what those things are and why they matter. All very fancy words in our space.

You know, it's very interesting stuff because at the heart of this, you know, if you're ever go through a gene test, you know, basically sections one and two is looking at metabolism and insulin signaling, which is very important.

At the heart of the sirs is this issue. It's mitochondrial insult. Right. And stemming from cell danger response type two. So, you know, cell danger response, as you may know, as the cells perceive a danger and it responds and it responds by taking this defensive posturing so that it could conserve energy.

And, you know, there are these various, you know, bio toxins that are ultimately what's ribose toxic. Right. And these ribose they're toxic to the ribosomes of your cell.

And, you know, within the heart of all of this is basically, you know, the cell's going to take this defensive posturing and it's going to try to conserve energy and you're going to see that ultimately there's a downregulation or a suppression of the ribosome and the might of chromosomal RNA.

And when we see that, we could see that both on a large and small subunit level of ribosomes, which means there's decreased protein production, decreased mitochondria like electron transport chain, and this gives rise to a lot of the symptomatic presentation in regards to energy production.

This could also be further quantified when we see this, you know, with an increased anion gap and metabolic acidosis that could be determined in a a comprehensive metabolic profile.

And at the heart of this is, you know, when we have this kind of molecular or hypo metabolism, which was coined by Dr. Jeremy Ryan, you know, with his research in the transcriptomic field, you know, basically what we see is that, you know, we're kind of getting funneled into the next phase of this, which is, you know, identifying whether or not there is the Warburg effect or Arabic glycolysis taking place.

And this approach to glycolysis actually sets up the microenvironment for what's known as proliferative physiology, which our cells are dead for diverting a lot of its resources to making cells rapidly.

And I use that term the Warburg effect, because you may know that term as it relates to cancer biology, as it sets up that low oxygen environment. So at the heart of this is aerobic glycolysis and a robot aerobic glycolysis just basically means that we're not manufacturing energy appropriately.

Actually, at most we're generating two atpase and two pyruvate versus when we have oxidative phosphorylation, we're able to generate 36 ATP. So that's that's the fatigue right there.

That's then, you know, the the neurological symptoms and the decreased cardiopulmonary endurance and so on

and so forth that could be associated with, you know, this environmental threat, the cell danger response and the shifting of that metabolism of the cell towards aerobic glycolysis and setting it up for proliferative physiology.

So it's very interesting. And, you know, offline, you know, we're talking about, you know, the role of of certain things exacerbating that, you know, so what makes this happen?

And I'm sorry if I'm getting too technical again, I'm a molecular biologist by heart, so I love all the granular detail, but. I'm loving how you're tying this together just for our audience.

Because when you can tie together a root cause with what's going on at this detailed biochemistry physiology level to, you know, the symptoms that people are seeing it, you know, people that are like wondering, is this really is this what's causing my illness?

Is this can I trust this? That if I get to the bottom of this, it's going to help me to get better? I think it just gives that ease like, okay, we're really starting to understand some of these detailed mechanisms, so I'm loving it.

I've got good DNA. And it's, you know, it's interesting, too, because, you know, this is going to set us up for insulin resistance and weight gain and all the other symptoms that could be tied into it as well.

I mean, it really is fascinating how many people gain weight when they're exposed to any kind of environmental toxins. So this explains part of why that happens.

Yeah. So think it has to do with the body trying to protect the mitochondria and the cell membranes in the organs from the toxins and trying to make the adipose tissue to store the toxins in.

But this certainly makes a lot of sense of why people's metabolism stuck and they can't lose weight for the likes of them. Yet metabolism is short, you know, aerobic glycolysis taking place where fermenting bread, you know, essentially, you know, we're in a state of fermentation.

You know, I always tell patients, hey, you're either rusting or rotting, you know, depending on the micro environment and what's what's taking place. And unfortunately, you know, you know, these these different into bio toxins and mycotoxins are, you know, at the heart of this, they're they're essentially sap or phytic organisms, meaning they're going to feed off, you know, dead, decaying or living matter.

So it's really a very interesting, you know, dynamic microbe ecology that's behind all of this. The cellular environment that's behind all of this. And furthermore, looking at the role of leptin resistance, you know, so leptin is one of those neuropeptides that binds to the market nucleus and the hypothalamus and signals satiety are feeling full.

And what ends up happening with a lot of chronic inflammation, you know, sirs or otherwise, is that we start developing leptin resistance which further worsens, you know, some of the weight gain and difficulty to shed some of those pounds.

So there are so many different factors here, you know, that are influencing things, you know, so so we went over some of the molecular hypo metabolism, we went over some of the proliferative physiology.

And this has to do with something called Iris two signaling, which is basically this kinase that activates a one in four channels and you know, it works with map kinase mtor fatty acid, beta oxidation and all sorts of stuff.

And then we have the closing of these feedback channels, these voltage dependent anion channels.

Treatment Protocols, Coagulation, Histamine, and PTSD

And when we have the closing of those voltage gated channels. Right, that's not good. And that leads to a worsening of cellular injury. It leads to accelerated degeneration because at the heart of this, you know, if we get a,

you know, a neuro quant MRI, we'll see very specific patterns of nuclear atrophy, cortical gray lateral ventricles that could be associated with this chronic inflammatory issue.

And, you know, so using the Gini could really provide some very great detail in regards to, you know, these patterns of nuclear atrophy and the proliferative physiology.

And, you know, we spoke offline about, you know, the role of antifungals. And, you know, whenever we see this iris, too, or the, you know, the proliferative physiology signature, we got to be careful with the antifungals because, you know, the ASAL is more specifically worse in that damage.

And there's there's some studies on that in regards to closing it. So this is where you know, I know it's I know it's a little bit controversial, but again, from a mechanistic standpoint, you know, the results worsen the neuronal injury from the closing of those VDC channels and just worsening the iris too, and the proliferative physiology.

So it's very interesting. So, you know. Are a rock and a hard place because you need to get one thing fixed so another can get fixed. So it's at least good to have the data to know where you are and to your progress.

Yeah, and oh man, there's just so much out there as well because like, you know, some of the uncharted territory that I would love to explore is going to be looking at some of like for example, a German ISO path like remedies where they actually have, you know, these cell wall structures from Aspergillus mucosa and Candida and so forth that has used an ISO path form to kind of help a weakened immune system energy.

So I don't know if you're familiar with the term immune energy, but it's basically when your immune system's inundated with all sorts of crap, it just says, Hey, you know what, I give up, man.

I can't I can't handle this load anymore. So it just starts ignoring things, right? And there are different ways to kind of go in there from various levels of, you know, therapies to to reawaken the immune system to alleviate that burden.

So there's so many different things that I think, you know, this this subset of medicine and this pathophysiological process has to evolve to including some of those additional remedies, which I haven't seen in the science or research on that as yet.

But I know it's out there and it's kind of hard to get here in the U. S., But, you know, it's another form of, I think, addressing this issue. So really immunotherapy, which is becoming so big and yeah, cancer treatment and all kinds of things.

So I'm I look forward to hearing more about that, too. Let's get a little bit more into treatment and what kinds of things you see how patients get better.

Yeah, you know, I think treatment there is a very set roadmap with everything here, you know, And at the heart of the shoemaker protocol is essentially going through all the different steps and phases and ensuring that you've kind of completed those steps before graduating to the next phase and then ultimately getting to VIP.

And you know, that protocol is pretty well established and laid out and very specific, you know, in regards to basically using the BCS test as kind of a gatekeeper of assessing that neuroinflammation on that optic nerve and then starting a binder.

And as you know, the binders that's typically endorsed by the Shoemaker camp is going to be your well call course styrene. And then, you know, but step number one in this entire process is ensuring that you're removing yourself from ongoing exposure.

And if that means remediation to your current living situation, that it's done in a medically sound environment. And typically we recommend Safe Start with Dr.

Larry Schwartz. Safe Start, I take you out of Illinois, and Larry is a sweetheart. He's one of the best guys ever, and he's the one writing the protocols and training a lot of these IEPs and or environmental professionals on how to address and remediate some of these issues.

So we typically love Larry, and I've done a home inspection with Larry before historically, and it's like he's a doctor for your home, He's an engineer, but he's also going in there and like he's he's like a Ghostbuster, you know, he's looking at, you know, thermal signatures, heat indexes, earmuffs and, you know, all all these different areas of infrastructural vulnerability, flashbacks, kickbacks, you know, that could be, you know, not typically observed, you know, by a certain routine kind of assessment by these different IEPs.

So, you know, step number one, ensuring you're in a medically safe and sound environment, that you're not reacting to step number two, get passed up excess with a binder.

Step number three, you know, once you pass a X test, address the mark ons, you know, which is going to be, you know, their various therapies for from Arkansas, you know, and that's Dr.

Joe Musto from Microbiome Detox, Microbiology Detox, where, you know, you essentially go on EDTA silver nasal spray and or bag spray or Formula One NSP.

And then once you clear the mark on your kind of cleaning up the gut in the process as well and avoiding those anti ugly out and antibodies, and then you make it into the hormones, you create the hormones and then, you know, so there's this very stepwise therapeutic protocol.

And if like the TGF beta is not coming down, they're entertaining low dose losartan potentially for this issue. And you know, so it just really depends on the individual.

Some of the things that we also see on a transcriptomic level is, you know, upregulation if one a hypoxic inducible factor one a which, you know, these individuals, you know, are kind of predisposed for the potential for pulmonary hypertension.

But when we see that, you know, we really need to work up the, you know, cardio pulmonary circuit, whether it's getting an echocardiogram to rule out premature hypertension or, you know, doing a sleep study to make sure that, you know, there's nothing festering there.

And, you know, these are the individuals that have, you know, decreased exercise tolerance. These are the individuals that are like, oh, man, I just went up a flight of stairs and I'm short of breath.

My goodness, what happened? So, you know, we see that signature with then the gene test. And another very important thing that needs to be addressed is the Kovacs.

Right. And this goes beyond running a d-dimer attack complex. Is your Von Willebrand's your factor aids and so forth. But when we see, you know, there's this upregulation in the coagulation biomarkers within, you know, SIRS system that has to be addressed because that is going to lead to the vascular or, you know, vascular hypothesis for neuronal degeneration.

So what ends up happening, you know, is that essentially you these these cells, these these neuro the neurons are basically they're these little micro bleeds and there's distal hypoxia that could lead to a transformation of or tell tale body formation, which are ultimately phosphorylated and could accelerate the neurodegenerative process.

So when we see the co AGs and the tubule and genes upregulated, that tells us, hey, you know, we need to pay attention to our circulatory system. That coupled with the hip one day, you know, we really need to pay attention to the circulatory system and prior to prioritize that therapeutic intervention for that person and make sure we're not missing, you know, other factors that could be, you know, influencing their clinical presentation.

And we need to be very aggressive with that because time is tissue, you know, just like, you know, in the hospital,

time is tissue. And, you know, the longer that these neurons are exposed to these insults and the hypoxic microenvironment, the more they're going to it's going to lead to irreversible nuclear atrophy of the brain pattern tissues.

So all that's very important, you know, so very common in what we see. Another really cool thing with the, you know, the gene test that I love and if you go back to the literature is looking at the f kp, f k, BP five gene, which is associated with PTSD.

And oftentimes, you know, if we're not addressing the kind of the psycho emotional component of things that could be driving a dysregulated, autonomic response, locking us into a sympathetic dominant state so we can actually see, hey, man, you got like PTSD and, you know, that needs to be addressed.

Well, I'll just tell you an interesting story. I ran this test on myself, I think maybe about five years ago. I'd gone through another mild exposure and I was in it.

You know, the test is finally available. I had been waiting for it for a few years while it was in development. And so I did it and I actually had the capability, PTSD, gene upregulated, and I was like, What?

I don't know of any trauma that I have that I don't have PTSD. And and then after like settling into it, I realized, oh, I got electrocuted when I was too pretty badly burned.

Pretty badly, actually. I had a fairly traumatic brain injury and that was that was probably so I probably had that gene upbringing in my whole life. And it was really helpful to know that because then I could go to the work or that.

Yeah, and you know, we can not neglect the work around that because as you know, you know how many people are stuck in a state of sympathetic dominance associated with some sort of level of trauma.

And what does that do for us long term with that high static, sympathetic load? You know, it it decreases our cardiac coherence, which is essentially, you know, the heart rate variability.

And, you know, that will we will never allow ourselves to heal or unlock the healing mechanism if we're locked in that state of fight or flight. You know, so it's I think, one of the most fascinating associations with this test is looking at that RFK, PB five Gene.

And then also, you know, there's we see some T cell B cell dysregulation. And when we see like T cell receptor DOWNREGULATED with antigen presenting cells, again, that's furthering the, you know, the immune dysfunction not being able to have the warm handoff from, you know, the unsophisticated form of the immune system to the sophisticated form of the immune system to put out this fire and tap the brakes on things.

So it's very interesting. And then another really cool set of genes that we look at is going to have to do with the histamines, right? So I know that there's a lot of opinion about, oh is it m gas, is it histamine, What is it?

And it's like I 60% of people that are dealing with sirs have histamine dysregulation and on a transcriptomic level, when we see that we're not just dealing with mast cells, we're dealing with all nucleated cells that have the ability to produce histamine.

So this goes between and beyond impasse in regards to, you know, the profound impact that histamine might have on this entire process. So, you know, it's just really dis elevated.

How do you think about it with your patients? You know, so I like to connect the molecular dots.

VIP, Co-Infections, and Closing Thoughts

I like to typically run full genome profiles on everybody, not just HLA haplotypes. I like to say, hey, is there like, you know, HCC, AOC one gene polymorphisms that could be associated with an inability to dampen that histamine

response, maybe deficient, you know, determining oxidase kind of activity.

And so I like to connect the molecular dots again that's going upstream to the genomics. We're looking at the transcriptomics and then proteomics. You could measure this through chroma grant and trip days, histamine and so forth.

And if that is a culprit, we definitely have to work on it in regards to stabilizing the H1 H2 receptors and then also maybe providing some Dow enzyme activity and maybe decreasing that the filling of the bucket of that histamine burden with, you know, being conscious of a low histamine diet.

So, you know, that all plays a role into things. So I it's definitely a part of the equation. And you know, with follow up, there's just so many so many different rabbit holes to go down, you know, the coagulation, the histamine, the PTSD, the molecular hypo metabolism with proliferative physiology.

And so on and so forth. It's just it's quite overwhelming sometimes. And in regards to the therapeutic order, you know, we just we can't neglect the histamine component, we cannot neglect the PTSD component.

So all of those need to be addressed respectfully. Well, and it's so great to have these tools now because, you know, a lot of people really do it very well, just getting in a clean environment, helping their detox pathways work better, get on a low inflammation diet, work on their mindset and and they just help very quickly.

And then but then to have these tools for the people where, oh, there's another of the puzzle there that maybe we need to look at some of our genomics, we need to look at some other transcriptomics some of these other infections.

It's just such an amazing experience to have these tools and to be able to fill in these puzzle pieces with the clues. And you hit the nail on the head.

And you know what we've seen longitudinally with the different stages of cirs, with ultimately a chain and gene expression with the trait on transcriptomic level is the role of VIP at undoing some of that accumulated damage to the DNA.

So, you know, as you work through the protocol, you know, the goal is as to basically get you to a point where you could tolerate the vaso intestinal peptide to undo some of the, you know, the the damage accumulation over time.

And then you also mentioned, you know, the infectious roles. And I think the genie has a segment there for, you know, Lyme disease and co-infections. And, you know, it's very interesting because sometimes what we would see is what's called the Granzyme defense ends up regulated and these are a serial, a series of different proteins that influence and triggered by various bacterial and viral insults.

And when we see that need to kind of shift gears a little bit and address this potential for a significant viral burden that could be dampening the immune system and being, you know, needs to be prioritized in this equation.

So, you know, when we see grand aims and defense upregulated, that kind of shifts the narrative. You know we'll work on the back end with all the bio toxin stuff with cleaning up the environment and everything else.

But then you know in the meantime we have to prioritize, you know, addressing this infectious burden and, you know, what's really mediating it. And there's very specific protocols for that.

Well, just we could probably hope we could go on for days digging into each one of these areas. Then I think this has been super helpful, especially for people that are a little stuck like they you know, they need to fill in some of those puzzle pieces and to to be thinking about what else could be going on and and helping them to heal.

Yeah. Yeah. It's it's there's so much to to look at but it's you know what what's great is if you kind of just follow this protocol and this, you know, and you don't skip the steps, you don't really jump around and you kind of just have

faith.

The results are just incredible. You know, It really is. Now, is this the Holy Grail? And as is CIRS, you know, everything it plays a very strong role.

But I also think that, you know, there could be other factors influencing health expression as well that shouldn't be neglected, you know, hormone optimization and, you know, even neurotransmitter optimization and so forth.

All those things cannot be neglected. But generally speaking, when attracts and it's very like longitudinal in regards to connecting the dots and you follow through with the system and the protocol, the results are quite remarkable.

Yeah, but if and this is what this summit is all about, it's really turning health. And even when you think you're not able to recover that and there are there is a path.

You know. Yes, absolutely. Well, thanks so much for bringing your brilliance and your curiosity a lot. Love how your mind works, to be able to hold so many pieces of information all together at the same time and get clarity is really quite impressive.

Thank you for sharing that with our audience, and I look forward to more conversations as we get to explore new territories. On improving the healing path.

Well, thank you so much for raising awareness about this issue. And I you know, I got to say, what you're doing right now is much needed. You know, with over 40 million people potentially suffering from this undiagnosed kind of seemingly very prevalent issue.

So thank you for your all the work that you're doing. I look further. I look forward to further conversation with you. Thanks so much. Thank you.