Chronic Inflammatory Response Syndrome (CIRS): A New Molecular Paradigm For Understanding And Treating Patients With Chronic Fatigue And Biotoxin Exposures.? Transcript

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Introduction and Guest Background

If you. Hello and welcome to another episode of the Traveler Summit. I'm your host, Doctor Joseph Rafael. Today I'm very excited to have Doctor Andrew Hayman on to talk about, chronic inflammatory response syndrome, something which I don't know as much as I probably should know about and really interested in learning more.

And he is one of the experts on it. Doctor Hayman, welcome to the show. Andrew Hayman is an internationally recognized expert in integrative and metabolic medicine.

He is currently the medical director of Integrative Medicine at the George Washington University, and is responsible for overseeing graduate education and multiple degree programs, research and clinical services, and managing an interdisciplinary faculty of health professionals.

Doctor Hayman created the first master's degree in Integrative medicine in the United States, with a major within a major academic center, and developed additional graduate curricula in nutrition, metabolic medicine and performance, men's health, bio toxin exposure and neurodegeneration, and administrative health services in wellness settings.

He holds dual board certifications in integrative medicine and anti-aging and regenerative medicine. He is also the Director of Academic Affairs, the American Academy of Anti-Aging medicine, and oversees the training of thousands of medical professionals each year on the topics of anti-aging, integrative, metabolic, and nutritional medicine.

In addition, he he has held several leadership positions in the field of integrative medicine and is the Editor in chief of the Internet Journal of Anti-Aging and Esthetic Medicine.

Welcome to the Telomere Summit. Doctor Hammond, thanks for having me. Great to be here. Thanks so. Chronic inflammatory response syndrome is, Like I said, it's something that is not something I know a whole lot about, but I think there's overlap between areas of interest, as we were talking about earlier.

But to tell me a little bit, tell our audience a little bit about what that is, and then we'll get rolling. Sure. So, first of all, thanks for having me.

It's great to be here. And and certainly, to have a platform to share a topic that I think is, an incredibly important and one that probably almost all of us are seeing in our practices every day.

We just don't necessarily know it unless we know how to get it right, how to evaluate for it. And I was no different. I mean, you know, I spent 16 years at University of Michigan, and had, you know, feet firmly planted in the world of, of both integrative as well as academic medicine and, you know, felt like I had this sort of privileged position in that regard.

Having been on faculty there for a number of years in, in our Department of Family Medicine and feeling like I had sort of been there, done that and seen everything.

And then had an opportunity to move out to the East Coast and join a small little private practice in the Virginia countryside, and was immediately overwhelmed by a type of patient profile that was incredibly sick, had lots of

symptoms, no one really knew what was going on with them, and I was perplexed.

I met, you know, I felt like my toolkit was already pretty big. And, you know, we could do the full functional medicine workup in the conventional medicine workup and all that sort of stuff.

And nothing ever really seemed to click, and nothing and no treatments ever really seemed to work, at least not very well. And being in Virginia, you think, well, maybe they all have Lyme disease.

You know, I think that's sort of where your head goes when you can't figure it out. Right? A lot of us kind of go there at some point. And then of course we get scared because we think, I don't want to deal with my patients.

Like, who wants to deal with Lyme patients, you know, but, you know, I thought, hey, it's Virginia. So, so maybe that's that's what I'm, I'm looking at.

And for a while, I, I honestly, I tried to avoid it. I said, I don't I don't want to tackle this subject. I don't feel prepared. But at some point, George Mason University had approached me and they asked me to help them develop a better line.

Test was based on new nanotechnology.

What CIRS Is and Common Symptoms

And that, to me, not only opened a doorway into the world, but from a more academic perspective that felt familiar, but also now I had a better test and I could figure out that some of these people actually did have Lyme disease, but in fact, a majority of them didn't.

And think, well, wait a minute, know? Typically they all have the same symptoms. Why is it don't you just just for a second, talk about what that constellation of symptoms is so that we can sort of keep our eyes out for it?

So, you know, by definition, the defining features fatigue. So about 80 to 90% of these patients will talk about, you know, not having enough energy to get done what they want to do during the day.

Beyond that, you know, we've identified and actually codified into a, a validated symptom scale, 37 symptoms, that group into 13 clusters and, by criteria, if a patient has one symptom in eight of 13 clusters, there's a high likelihood they have Sirs.

So I won't go over all the symptoms. But generally beyond fatigue, it's mood disorders like depression or anxiety that's very common in this patient population.

Sort of dysregulated emotions, lack of stress resiliency. But we also see cognitive issues. So word finding difficulties, word recall problems, memory loss focus and concentration issues patients will often talk about I can't do my job anymore.

I just can't function intellectually. Beyond that, they'll often talk about pain syndromes, headaches, joint pain, soft tissue pain, Naropa. Things beyond that will often see the nervous system involved.

So, paresthesia is, electric like shocks, funny sensation and, disgusting. But also just sort of no means. So it's very common for patients to have gastroparesis, gallbladder dyskinesia, motility disorders.

Hot syndrome is very common as a feature of this illness. So it's a you know, these are nerves that are on fire. So basically in the central nervous system, the autonomic nervous system, the peripheral nervous system, but also the GI tract.

So we'll see Sibo and Gerd and bloating and constipation. And you know, all of the attendant you know, symptoms related to that. There's also a profound impact on the cardiovascular circuit.

So patients will often describe air hunger, shortness of breath loss, loss of exercise tolerance in addition to endocrine apathy. So they'll be describing a propensity for weight gain.

You know these patients will gain 3040 5,060 pounds in, several months. And they won't know why. So, you know, so my criteria is if you have a patient that comes to your practice and they say, well, I have fatigue and depression and I can't sleep at night, and I have aches and pains and I've gained a ton of weight.

And I tried diet and I tried exercise and I tried supplements and hormones and exosomes and peptides and this and that, and I nothing is working. It starts to sound like surgery.

And yet if you think about it, these are, these are common, you know, very common. But family right. What family medicine practice doesn't see this. Now from a functional medicine perspective, these people get the label of adrenal fatigue and hormonal imbalances and neurotransmitter issues.

And oh, you must have heavy metals or, you know, leaky gut or whatever. They get all the same labels too, and they typically don't get better through those usual strategies as well.

So I think that there is some overlap with traditional sort of chronic fatigue syndrome. And so I would imagine there probably is. Yeah, yeah. Under most circumstances this sort of chronic fatigue fibromyalgia patient is worse.

Okay. Oh okay. Yeah. Yeah. It's worse until proven otherwise. Covid long hauler is sirs until proven otherwise. Basically this is a group of people that have had a bio toxin exposure to the virus, and a portion of them just never recover.

So that's the difference. Now, we know in the past we didn't have necessarily, you know, a lot of geologic agent for chronic fatigue. So they talked about, you know, herpes virus, herpes virus six various other things.

Right. Right. And now what you're saying is that there this is probably from most assuredly, I guess, in your case, from exposure to a bio toxin. And I know, you know, you want to you'd like to make the distinction between a bio toxin and a toxin.

We're talking about those before. And so it's it's it's tell us about that. That makes it. That makes it. Yeah. That's so that's you know, I think one of the core features of the illness is that, by definition, it it's these biologic processes.

The pathophysiology is instigated by what I would term a bio toxin, meaning an organism, or a fragment of an organism. So we've not linked it, for example, to chemicals or VOCs, heavy metals, food stress, you know, other things that certainly make people sick.

But this is a very specific and unique process originating within the innate immune system. So it's inflammatory in origin. Now it becomes more complicated than that.

You get these sort of secondary tertiary metabolic consequences as a result. But it begins with a dysregulated immune system that's instigated from a bio toxin 80% of the time.

Most of the time, it's from what we would term amplified microbial growth from a water damage building. So basically, microbes that are present in our living space that's instigating this inflammatory response.

Now remember I walk through the lime door first. Lime can do it to Covid can do it. The actual the origination story of this, started in the Chesapeake Bay with actually algae overgrowth, that there was an outbreak of the story in the late 1990s, and no one knew it was making all these people sick in the back Bay.

Well, it turns out they were swimming in the water and encountering hysteria. And they had all the symptoms that I mentioned know when it was going on.

And that set off the journey of trying to figure out, well, what is this thing that how can these people have so many

different symptoms? How can they be so sick?

It took our research group, quite frankly, a long time to first find the part of the immune system that was being triggered or turned on. It was not in the areas that we typically or classically measure.

So what to account doesn't go up? Said rate in CRP does not go up, and egg and autoimmune processes do not, you know, are not really features of this illness that in fact, it was a dysfunction within the innate part of the immune system, which is sort of those early responders that should really kind of turn on and turn off.

But what's happening is in this patient population that's vulnerable, the innate immune system turns on and it stays on and it doesn't, transfer it to the adaptive side of the immune system.

Technically speaking, what's happening is it's about 20% of the population. They're born with alleles, within the HLA and chromosome six that if present, there's about nine of these genes floating around in the human genome.

If any one of these nine genes are present, it means that their antigen presenting cells are relatively inefficient. So the innate immune system turns on these antigen presenting cells should find that antigen and snip in epitope and present it to a T cell.

But by definition, if those genetic variants are present, the antigen presenting cells are not very good at their job. So the innate immune system turns on.

But you don't see a very efficient transition to T cells and B cells. So you don't see a big antibody production. So what happens is you have this sort of perpetual cytokine storm.

And over time it's just so okay. So the cytokine storm is not the usual il6 TNF alpha. That or C-reactive protein. Which cytokines is it. Was oh so go ahead.

Basically neurotoxic factors that begin in the brain. So basically we're turning on micro glial cells. And those micro glial cells are turning on a cascade of cytokines that in particular begin to affect and quite frankly, injure the posterior hypothalamus.

So we begin to see changes in leptin receptors. And then ultimately, production of melanocytes, stimulating hormone, basal activities and peptide production and arginine vasopressin as features of production of the anterior hypothalamus.

So the cytokines are in the brain itself. And that begins to create first functional and then structural changes in the brain. And then you get these secondary and tertiary consequences throughout the physiology.

Biotoxin Exposure, Genetics, and Immune Dysfunction

So are these we also know are these the these is these these genetic tests of are available to see whether or not they are I mean you can order them. I ordered them every day from LabCorp.

So they're called HLA. And you know it's the DRB one Dxb 12345. And we can assemble this, the snips into the aleo, essentially, and we can tell if they're in the 20% and if they're in the 20%, it means they're predisposed.

Are they labeled within LabCorp as being for cars? No. So you have to now you need to know which one down. Okay. You have to know which ones they are.

Yeah. So there's a what we call a Rosetta Stone. And it's actually on a website called Surviving mold.com. So there's an exercise that you have to go through where once you get the HLA report from Lab Core, you can go to the survival mode website to the Rosetta Stone and use that to interpret and put the ADL together to know if you have one of these alleles that's predisposing.

Oh, okay. Very interesting. Yeah, yeah, yeah. And interestingly, just recently just to kind of confirm our model, there are some separate research groups that showed that they found the HLA that predispose to Covid long haulers.

Well, it was all the same HLA that we found for Sirs in general. And it may have been very gratifying for you. It was very gratifying to see that. Yeah. Yeah, yeah.

So now the physiology of it, you know, these the antigen presenting cells are not working correctly. And so and then the glial cells start producing cytokines that then cause all these other Endocrinol.

But these, and, and this is can somebody get this that doesn't have those, genetic mutations? Yes. It's a great question. So if you have the HLA, it means a few things.

Number one, a small dose or a small exposure can ignite a large fire. It also means that once that fire starts, the body has a hard time turning it off.

So there's a real vulnerability if you're in the 20%, if you're in the 80%, the dose response relationship is held. But you could be someone who, let's say, lives in a really moldy environment and it'll turn on, sirs, even if you don't have the way.

The other thing that we've learned is that while the HLA helps to really get things going in that vulnerable population, those inflammatory compounds that are released not just in the brain in terms of cytokines, but in particular, we're turning on, THC 17 cells in the periphery.

So there's often very high TGF beta one and diminishment of T regulatory cells. So there's discordance there that we see as a consequence of that. There's overproduction of inflammatory compounds.

We kind of know what they are. They're they're small. They're lipid soluble. They're about 1.4 angstroms in size. They're produced throughout the tissues.

Those inflammatory compounds will traverse the cell membrane, traverse the nuclear envelope and they'll start acting as transcription factors and begin to alter patterns of gene expression.

So they'll literally change the instruction set of the body and how different genes are turning on and off. So we have a research lab in Massachusetts where we do what we call transcriptomic research.

We actually, you know, we we're we're experts in mRNA, not the vaccine, but, but we measure mRNA because that's how we know which genes are being expressed and which are not being expressed.

So in 2015, we started asking the question, are gene patterns altered in this illness, and if so, in what way? It took us many years to identify the patterns that are hallmarks.

And Sirs. But there are patterns and there are hallmarks. Most of them are actually expressed within in the mitochondrial DNA. And so this is really a disorder of the mitochondria and changes that occur within the either overexpression or under expression.

Mitochondrial DNA. And so we can now tell based on doing that sort of evaluation, does this patient have Sirs based on their pattern of gene expression.

In addition to that, we also found that genes that turn on from old exposure, UCLA and Johns Hopkins found the genes that turn on for Lyme disease. There are other genes that turn on for chronic infections outside of line.

And so this started to act as a roadmap for us. One of the findings that was surprising was that over time, the genes that are really negatively influenced are not inflammatory genes.

They are metabolomic genes. And, and and what's happening is when the cell is threatened, it will shift from

oxidative phosphorylation into a robust glycolysis.

There's a downregulation of ATP production. So now you're only producing two ATP percent of the Krebs cycle. So this is the hallmark of the fatigue. And the cells are becoming dependent on glucose as their substrate of choice as opposed to oxygen.

You might say well why are they doing this. Well, you know, why would the cell change how it burns energy. And it's doing this so it can upregulate it's defensive posturing that it changes other pathways that are adaptive for cellular threats.

But it can only do that at the expense of turning down oxidative phosphorylation. So we often talk about our cells are going from energy plants to battleships.

And this is we did not know the degree to which the metabolism is deeply affected. Now we know why people tend to gain weight and they develop insulin resistance.

And interestingly, the degree of that metabolic shift genomically also parents directly with the degree of injury to the brain. And this links in that notion of type three, Alzheimer's.

Right. This this idea that the brain is becoming injured by glucose. Well guess what? That's this illness. So when the cells are becoming more glucose dependent, the brain does not like that.

And it accelerates injury to the brain that we see greater areas of nuclear atrophy. The further down the pathway that person has gone in terms of the change of their gene expression, metabolically speaking.

So one of the consequences said differently is not only is the person now becoming more dependent on sugar and gaining weight, it's injuring their brain.

And the mechanism of injury is metabolic as opposed to inflammatory. So this is one of our recent insights, which we think is fascinating, that they have brought us to just talked about toxins and as being, you know, increase in, risk of Alzheimer's to a certain degree mean some of it was our work.

Oh, is that right? Yeah. He smartly is is taking from whatever he needs today to to. Yeah. Well that's that's his that's his move and something. But you know look he talks about measuring some of the markers that we use and uncover and the injury to the brain and all that.

Hey, you know what? It gets the word out. So, you know, good for him. But yes, the transcriptomics has been an enormous breakthrough for us because it allows us to do the deep dive and ask the question, how have the genes changed in terms of their pattern of expression?

And the way I describe this is we've moved well beyond, early molecular biology of genetics and asking what snips are present. And now I can ask the question, all right, that's fine, but what are your genes actually doing.

And said differently, it's it's, it's it's almost like saying, well, you know, you have this whole symphony orchestra of genes that you were given that make up who you are.

Genetics is asking the question, are each of those instruments built properly? Does your does the slide on your trombone stick? Do you have all the keys on your clarinet in the right order?

Are those strings on the cello tuned properly? That's genetics. And the assumption is if you have an instrument that wasn't built well, the sound that it makes is probably not what it should be.

Genomics and transcriptomics is asking, okay, that's fine, but what are whole sections of your orchestra doing? Are they playing too loudly? Are they playing too softly?

Are they playing at the right time? Or is there some sort of discord? Now, what's fascinating to me is you could have a whole section, you could have a whole brass section of your instruments that they're all built normally, but they're not playing at the right time or at the right level.

So you can get fooled by snip testing to set up this whole section of the genome is fine. Well, guess what? If it's not doing what it's supposed to do functionally, you still have disease cure. Yeah.

So we were talking earlier about, before getting on camera about, what role epigenetic epigenetics might play in that. But, you know, most of the epigenetics that is available now is to, you know, DNA methylation.

And you're saying it's other transcript, transcription factors that are causing this. So you wouldn't necessarily pick this up in an epigenetics, stream.

That's right. Because we know methylation doesn't play a large role in in influencing expression patterns in this illness. Now, don't forget there's really three signaling pathways.

There's acetylation, phosphorylation and methylation. All three act as sort of those epigenetic signal. Sure. But but but other things also influence genetic expression.

So the disease process itself the inflammatory story is over time what actually changes, you know, gene expression in this patient population. And and I'll tell you how powerful the insight has been.

So so we're starting to build a model, right. We're saying, okay, people have an exposure. They get really inflamed. They get really sick. And now we're starting to figure out genomically, you know, from what.

Right. So I can tell the genes that turn on from old and I can tell the genes that turn on for a long time. And what a powerful tool. What's fascinating is, in our last data set of a thousand research subjects, when we looked at their gene expression patterns, only 7% of them were sick from molt.

And we said, well, that's odd because most people in this category are actually sick from water damage. Building. So if it's not from mold, what's making these people sick?

So we started looking at other genes. And sure enough, there was this pattern of expression that kept coming up over and over and over, and we said, okay, these are people reacting to their environment, but it's not so much similar, but not the same.

So what is it? It turns out that as we went to some research out of Denmark and Finland and Austria, there are research groups that demonstrated when you have microbial growth in a living space, much of that microbial growth is actually not mold.

It's back. It's a bacteria. It's actually bacteria. What kinds. So it's a it's it's a it's a, it's a it's a gram negative staph. And the most common is called actin a mite since it's a mouthful, actually, to my seats, there's 6000 species.

Some are soil based. But the ones that we have found and this is really the I mean, this talk about Alice in Wonderland going down the rabbit hole. So first off, we found that 42% of that research population were sick from academic genomics, compared to the 7%.

It's not even close, right? No. Most people who are sick from their living space, they're not sick from mold, does not more sick from if they're sick.

That's not mold. It's actually a mixture of bacteria. And and and this this further sort of debunks you know, a lot of practitioners are doing urine testing for mold.

And that test has actually never been validated. It's not been shown to correlate with environmental exposure to mold. It only actually correlates to food that people have eaten with a little bit of mold.

Transcriptomics, Metabolism, and Brain Injury

We know that based on 150 studies, it's not a great way of assessing. Now we know for certain because the real culprit, the real criminal, it's not mold, it's actually my seeds.

What's even more fascinating is there are different forms or types of actos. Some are soil based, but the one that we figured out that makes people really sick colonize is the skin.

So you can get this so touching. Not you can get it by touching and you take your critters with you. Oh. Right. Contagious. And so we're finding that, in fact, if you have the skin based form, you're likely seeding and being reseeded by your living space.

So the bed, the bedding, the bathroom, the closet, the bedroom, the places where you spend most of your time in your home. That's where antennas will concentrate.

Wow. And so now the remediation strategies that we're developing are really focused on two things. You know, good hygiene of the home but also good personal hygiene.

You gotta really scrub the skin to get better. If you have the skin form of nose, you've got to be clean and your living space has to be clean. And they're very specific ways of doing that.

We are finally cracking the code. We finally are solving the Rubik's Cube of what really drives this illness. And most people, it wasn't Lyme, it wasn't mold.

It's emerging to be this bacteria that's ubiquitous. And, and, and, and is, is very good at living on all sorts of surfaces. Interestingly, two, you know, when you talk about it inhabiting a living space, it doesn't need a lot of water to grow.

It doesn't need some sort of water intrusion or high humidity or a broken pipe or, you know, something that we think about with, with mold, actually, the early research shows it will happily grow in a home with just dust and certain building material, and therefore even a new build that was built properly and dry.

You can still have a tennis present. Oh, I was so happy that I have. I live in a new building, but I guess there goes that you never know. Now you're probably in the 80%, right?

You're probably in the 80%, but there's a bunch of miserable 20 percenters out there. And and now with the power of transcriptomics and looking at patterns of gene expression, we can identify that patient population. So, you know, kind of tying it into at least aging more broadly, if not telomeres.

You know, we now know this illness reaches into insulin resistance and diabetes because of those metabolomic changes and those mitochondrial genes. We also have shown that, there are a set, there's nine of them.

There's a set of coagulation genes that can also turn on in this illness. As a consequence of the exposure. And we've shown that if three or more of these coagulation genes activate, there's an accelerated risk for vascular dementia that micro clots start to form in the small capillaries feeding the brain.

So now we have a model that links exposure to vascular dementia, exposure to diabetes and insulin resistance and weight gain. Interestingly, we also found in 40% of our population for those sort of cell activation junkies, there are two histamine genes that can also turn on in this illness.

It means every nucleated cell in the body will be overproducing histamine, not just mast cell. So I'd love to have Doctor Afrin about sort of raising awareness on histamine.

And I do think there's a, you know, a small patient population that truly have national issues, but I think a much,

actually turned on their histamine genes.

So we've even linked it to that too. There's all sorts of tendrils that this illness has. And it's, you know, it's like that old allegory of the it's a three blind men touching the elephant.

And you think that's one thing or another. Well, it can be any one of those. These things are a mixture of, of, you know, chronic illnesses and deep symptom findings.

And, you know, and these patients are very resistant to standard therapy. They will not get better with just standard nutrition and hormones and and even the fancy stuff, even the stem cells and exosomes and peptides and all the regenerative therapies, until you figure out what that exposure is and did it and deal with it, these patients won't get better now.

They're going to remain sick. They're going to remain sick. So you go about doing that with, I think we have diagnosis. You have a whole checklist of things that do to the criteria that you meet.

And then is this transcriptomic analysis available? Is that. Yeah, just research it is. No, no, no, not at all. So, there's no real berry in terms of getting the test.

So it's it's actually listed on a website. It's called Pro Gene Rd project. So you can go and you can order that the gene test kit. Now it says on the top a health care provider must order this.

That's not true. Anyone could go out and get get the test. The challenge is once the test kit shows up at your house like it does with all of my patients.

Now what? Because it's a blood draw. In addition to that, because it's transcriptomics and it's a bit fancy, the sample needs to be frozen overnight and shipped on dry ice the next day. So.

So there's a little bit of a rigamarole, doctor. Should have it done through a doctor's office. Yeah. You should. Yeah, well, and also the the results, the report is, you know, sort of nearly inscrutable, unless you understand how to read it.

And it's, it's not a user friendly report. I would have to say. With that being said, it's an incredibly powerful one because of the depth of insight that it that it can give the practitioner.

It's an amazing tool. It's an amazing tool in that regard. And that's, that's that's name again is the lab is approaching and approaching the website Pro Gene X.

Yeah. Dot com. And it's the gene test. It's funny that the telomere testing the company that is it's the follow up. Yeah. Yeah. So, so, so, so okay, so then you have the, the diagnosis and then you have protocols for attacking this situation.

Obviously removal of the X and A bases is, is is important for treating the Lyme or stop swimming in the algae or get over of Covid or, you know, whatever it is.

But is it a situation where once this sort of set of inflammatory, genes have been turned on, even if you remove the offending agent or inciting agents, that it's hard to turn it off, or is there some therapies for turning it off or there's just removal of the, of the, of the exposure, you know, help to remediate?

You are it's a great question. You're smarter than the average bear, as they say. So the answer is you can't we we have figured out how to turn the genes off, or let's say restore normal gene, behavior.

We have one quiver or one one arrow in that quiver, which is one treat, one treatment, which we know works. There are probably others out there that do it, but there's also a sequence of, of, therapies that we have to go through in a certain order to get to that last step where the magic happens and we we turn the genes back to

normal and, and sort of said differently.

And this is fascinating. When you look at our transcriptomic reports, we can now tell based on the patterns, if someone is a new diagnosis and really hasn't received any effective therapy, we call that stage one.

It's a pretty classic set of findings. Stage two certain genes start to turn off because of the initiation of proper treatment. And that means typically a binder of some sort.

Usually it's a bile acid sequestering. We also know, for example, that, okra and beets, can act as a proper binder. We're firm believers that charcoal and clay and Chlorella do not work as a binder, even though lots of, you know, I get back to back tears or try that, but.

But in our data set, we either need to use color star. I mean, well, call okra beets, sort of somewhere in that mixture. Those really work to get people moving in terms of draining the body of inflammation.

After that, we start dealing with sort of the metabolic disturbances, whether that's decreasing inflammation in the gut or balancing or hormones or so on and so forth.

There's a whole protocol we go through. So we we deal with the exposure. We drain the body of inflammation, we correct those metabolic disturbance. But that gets us to stage three, that even after we do what I would think of as many functional interventions, there's still this is irreducible, nugget of genes that will remain abnormal.

And it's the reason why, you know, you can give these patients all sorts of fancy therapies, but those genes just won't go back to normal. And the risk is still present for dementia, for diabetes, that some of those really important genes that we want to turn off, they're still on.

So stage you see that in the transcript. If you did the trend, we can see it in our data sets. And it's it's very predictable. It's amazing that we can tell what are the genes that typically turn off and what order that the body has some sort of internal intelligence in that regard, that some genes are great at turning off because of our nutritional interventions and, you know, support of metabolism and so on and so forth.

And that gets us to stage three. And stage three indicates the person is ready for the magic sauce, essentially, that we've turned off all the genes we're going to turn off through lack of a better term, a functional approach.

But there's still that irreducible nugget. And so we give a special neuropeptide called vaso active intestinal peptide, or VIP for short. And VIP acts as a transcription factor.

And it will turn off those residual genes and restore normal health and basically induce cure. We don't we do not have a substitute for that treatment.

It's the only one that we have found that once you get to stage three, we'll get we'll push people over the finish line and get to stage four, which is cure.

So we we can tell as the person is healing, you know, as we do their genomics where they are, what stage are they in, are they still being exposed. You know, so sometimes, for example, like earlier today I was treating a patient and I thought she was further along than she was because we thought she had fully remediated her home.

But, you know, but there were still some symptoms that were residual. And we were wondering, are you ready for VIP? Well, we did a genomic test. We did Jeannie, and sure enough, it showed she was still being exposed, that that roadblock was still present.

So we have to go back. We deal with the house, do further remediation, and then she'll probably be ready for VIP it. We can, you know, if you give VIP too early in the process, it won't work.

Nothing bad will happen. There's no sort of side effects or adverse events. It just you won't see the magic, you

won't get people to cure, you know? And, How.

I mean, I'm just curious, but how does VIP work in this, in this circumstance? My, my my memory about what in fact, VIP actually does is is a little bit, so it's interesting is.

Yeah. Well, you know, first of all, it was actually brought to market decades ago as a drug, as an antihypertensive, and especially for pulmonary hypertension.

Right. I remember that. Yeah. Right. I mean, this has been around for a long time, but it fell out of favor because it was expensive and you had to inject it.

Right. But there's lots of human studies on, on VIP. So it's endogenous, right? Our bodies make it. And we have two different VIP receptors. They're scattered throughout the tissues.

You can even look up VIP receptor maps. They concentrate in the blood vessels and in certain organs and tissues. And so you can begin to guess sort of what, you know, effects that VIP might have in the body.

Diagnosis, Remediation, and Treatment Staging

One of the most immediate, of course, is dilating blood vessels, which I'll tell you, is incredibly important in this illness because one of the consequences of Sirs is actually constriction of the capillaries.

So it's that's what really contributes to the low VO2 max and loss of exercise tolerance and the cardiopulmonary impairment. So if we do CPR testing in these patients, we'll see their VO2 max tends to be 20, 2122.

They're like heart failure patients. Do they have any dysfunction as well. They do. Oh yeah. They've got endothelial dysfunction. They've got ballooning of the right side of the heart.

They've got stretching of the trail. No, they got the real deal. They typically have a mild pulmonary hypertension and ethereal dysfunction on top of the micro clotting.

Right. So we can see stroke and heart disease and the rest in this patient population. So when we give the IP it works quickly. And boom those blood vessels were dilate.

And the person for the first time in years would go I can breathe again. Wow. I can't believe I just took a breath and I feel like I'm finally oxygenating oxygenating my my tissues.

And of course, it's opening the capillaries to the brain, right? So the brain is finally getting oxygenated to that happens right away. VIP has some anti-inflammatory qualities as well, which of course is good in this illness too.

But the magic, how is it getting those genes back to normal? We have no idea that it's working as a transcription factor. It's clearly restoring those genes back to normal.

We have tons of data to show that, but quite frankly, how does it know to do that at the level of the mitochondria so predictably and reliably no one knows.

And it's, the daily subcutaneous injection? Or how long do you have to do it? Actually, it's a nasal spray, but it is. That's right. It's a it's a nasal spray.

It's a nasal spray. But initial injection, you said. Well that's right. Initially for blood pressure 60. It was an injection. Now we haven't made it. There's only two pharmacies in the country that know how to make the ship properly as a nasal spray.

And it takes time. You know, this is basically there's a build up phase, but on average, it takes our patients about six months. Oh, really? Not the magic dust.

Yep. At the main dose to to to induce cure. Now that's average. So we have some patients that you know take a lot longer than that. And others are overachievers.

And they get there in 3 or 4 months and they go I can't believe it. I feel like my old self. I haven't felt this good in 20 years kind of thing. That's an amazing thing, especially in this patient population that is so unbelievably miserable and so sick in so many ways.

And, and they they're so grateful and thankful when they finally feel like their health is fully restored, it it's an amazing, thing to bear witness to.

And, and even though this is incredibly complicated and yes, these patients are very trying. The other side of it is really, you know, gratifying. No, you know. Yeah, absolutely.

Yeah. So getting back to what I'm curious about in terms of figuring out when somebody's sitting across from you with a constellation of symptoms, is this typically, a fairly abrupt onset?

Because, you know, if you look back, they might have been, you know, dealing with for years, but is it an abrupt onset where there's that first exposure and then you see this happen?

Unlike there's something else that's a more sort of gradual degenerative thing. You usually pick that up. Yeah. You know, sometimes in the moment when the person, let's say, moves into the apartment or the dorm room or whatever, and they, they start to, you know, start to feel tired and depressed and gaining weight, you know, it's it's often easy to sort of ascribe it to the person circumstances.

They go, oh, well, I'm in residency or I'm stressed from school or I just got married, you know, whatever. You know, my job is difficult or it's or that's just life sort of thing.

But most people looking back when they finally connect all the dots, they can pretty much they can pretty much determine when this thing started. You know, it might not be abrupt per se, really obvious, but there is a starting point where people say, my health changed.

My health definitely changed when I was living. You know, wherever X, you know, and sometimes it is dramatic. Sometimes it's, you know, they went on vacation to the Caribbean, they ate a fish with sick with Tara, you know, they had a biological exposure and they came back and the wheels fell off.

You know, it was clear there was sort of this before and after. But there is a start point most patients can identify, either when it's happening or soon after they look back and say, you know, six months ago something happened to me.

I don't know what it was, but I'm not I'm not myself. And you can you can reassure them, saying, look, first of all, it took us 20 years to figure a lot of this out.

There's a reason why your typical labs are normal. There's a reason why no one can sort of really figure out what's going on with you. But guess what?

Your body has changed all the way down to your instruction set, all the way down to your DNA. No wonder you don't feel like yourself. No wonder you feel so sick.

You have every right to feel the way you do. This is not in your head. It's not psychiatric. You know, we've got to go deeper than that. And there's a consequence, you know, there's some damage.

Your brain, if left unchecked, you know, this is a serious illness. This is not we're not messing around with just this functional stuff of, you have some magnesium deficiency.

Like, No, this leads to dementia. You know, we I mean, we we take this seriously. So what, in terms of, I don't know

if you have these figures, but epidemiologically, what was the prevalence of this this disorder, you think?

I mean, I'm sure there's some undiagnosed, but. Yeah. Is it how do you know? I well, if you if you imagine 20% of the US population is vulnerable because they carry those genes, 50% of US buildings have water damage.

So those numbers. Right. So we're talking 40 million people or who are at risk just from that now. Is it all. No, of course not. I mean I plenty of patients who they didn't get sick from mold until they were 50 and you know, they had the gene and of course they encountered mold.

But but why age 50. We don't know. We don't know why the gene might fully express itself later in life. There's probably some loss of resiliency. Maybe it's telomere shortening.

You know, maybe there's just kind of this accumulated effect of, you know, loss of metabolic resiliency and, and sort of biological aging that contributes to that final expression of the illness.

It doesn't happen, you know, right away with a lot of people, it happens later in life. Later. Those genes are is there, a reference paper for those particular genes so we can sort of look them up and see what they are?

Sure. So, I mean, I would say even though it's a compendium, it's probably the best single source of all of this information is our textbook. It's called the Art and Science of Sirs.

And it's, you know, it's it's, you know, it has everything that I'm talking about. It has chapters on diagnosis, it has chapters on, you know, what, what's the symptoms scale, what are the lab findings?

What's the genomics, what's the brain scan? What are the treatments. And it's peppered with cases. So it's also very practical in terms of okay fine I you know diagnosis stuff.

But what do I do. Well that's all laid out as well. So it's it's all in the texture and, you know, and it's cheap. I mean, it's, you know, it's, it's a, it's an e-book.

So it's, it's really inexpensive. But it's got everything I'm talking about in there, you know. So the art and science of sirs are great. I, I think it's, amazing work you're doing.

And I'm still trying to think about the overlaps because I think about things, in terms of telomere biology, because they're pretty high up on the list.

But the innate immune system being the starting place for this. You don't see, I mean, maybe you haven't looked at. I wonder whether you see an increase in senescent cells.

If you look for them, and maybe so, you know, particularly with Covid, probably shorter telomeres. But there are a couple of papers that show shorter resilience to predispose to more severe disease because they don't have the resources to a in results in lymphopenia.

And also there's more cytokine storm because there's more senescent cells producing. But you're not talking about that same kind of cytokine storm. I wonder, but just also lastly, I'm sure it's in the textbook, but the final common pathway is the VIP for the treatment that no matter what the area lesion logic is, ultimately, yeah.

Because as I said, there's always that irreducible negative genes that just don't seem to go back to normal until we give the IP. Now, you know, are we eventually going to substitute and find other therapies that that might work as well or better?

I certainly hope so. I don't like having only one treatment. We know this one works, but it's expensive and it takes time, you know? Right. Exactly. Doesn't work quite as fast as you might like it to.

Right. But who knows? I mean, with that level of disarray and damage, it's going to maybe take anything a while

So then they get back to full exercise tolerance and and all these other things.

Turn around. Right. Yeah. Look, can they do to looking at that textbook? Well, it's so not all metal. Not all mold is mold. Okay, so that's one. That's right. That's, That's right.

And most of the time it might well be a bacteria and affected devices. And so and probably most of what we in the past, thought of as the, you know, diagnosable, untreatable chronic fatigue, syndrome, fibromyalgia is this year.

You will. Yeah, I would say probably, under many circum maybe not all, but but certainly a portion of those patients that that's it. This is it. This is what that and and that's a that's a fairly large number of patients.

So yeah. Absolutely. And there's a genetic predisposition which, which makes sense.

VIP Therapy, Recovery, and Future Directions

And then the change in gene transcription genes load the gun lifestyle or exposures, the exposure, is what pulls the trigger and then. That's right. But to put the bullet back in the barrel, with, with the things that you're doing, what's been fascinating.

Talk to me about this. Is there anything else you'd like to share with our audience about, you know, where you're going with your next steps with this? And, you know what the future is for this, I would say.

Yeah, I think it's a great question. I would say, a couple of things. Number one, if you're in this space as a practitioner and, you know, your, your, you know, seeing patients on a regular basis, I think this is, a tool that you need in your toolkit that, that, you know, most practitioners, unless, you know, they're pretty narrow in their focus, they're likely seeing these patients every day.

And they, you know, they go under those labels, right, of of leaky gut and adrenal fatigue and metabolic syndrome and hormonal imbalance and psychiatric issues and neurodegenerative.

And, you know, the list is really long and it's common. Most of us who are in this field of integrative functional anti-aging, you're seeing these patients, you might not have recognized them as such.

But even if it's not something you want to take on treatment wise, I would urge people to learn about, had at least quickly identifying who these people are.

There's a few simple things that you can do to sort of pick up on them. One is the symptom scale. So again, you know, if they have one symptom in eight of 13 clusters, in addition to that, there's a visual test that we have people do online.

It's a contrast study. And we know that contrast is lost or impaired as a feature of this illness. Oh, so that in extreme sensitivity testing, that's it.

Yeah. I see, you know, like this, a while ago, because it was a sort of a biomarker of aging to a certain extent. It just sounds like an accelerated aging syndrome.

Anyhow. It is. And we know, for example, that if a person fails the visual contrast test and they have those, they meet criteria for symptoms. We've got a 99.6% accuracy rate of making the diagnosis of Sirs.

So if you're just kind of attuned to the symptoms and you suggest the patient take the visual tests and they fail, you can be pretty much assured you've got a serious patient.

Now, you might not want to take it on clinically, but you'll do that person a service by finally giving them a label and hopefully packaging them up and sending them off to someone who you know, knows how to deal with this and

So that would be, I think the first thing you just kind of a take away of, hey, this is really common stuff, and it's certainly very common, I think, in our patient population, because they've usually already failed conventional medicine and they're looking for some sort of, you know, new insight into why do they feel so terrible.

The second piece is as we sort of move forward in our, in our research and our work, is that, you know, common things are common. And, you know, while there's been this great insight to say, oh my gosh, you know, it's not Lyme and it's not mold, it's this weirdo bacteria that seems to be everywhere.

And then you step back from that idea and you realize if most of these people are sick from their environment, I think the great insight is, why aren't we using our living spaces as a way of, measuring another vital sign in our patient population?

Very interesting thought. Yeah. You know, that of so many people are so affected by the quality and health of their living environment and their living space in their workspace, we just need to take that more seriously, that this is, to me, a failure of primary care.

It's a failure of public health. And it to some degree, it's a failure of our community to that. We're just not up to speed recognizing, you know, how ubiquitous this is.

And, you know, it's not always a nutritional deficiency and a hormonal imbalance and a feature of aging. And, you know, that we have to see as a whole people in whole environments and even in our community, I think we've sort of missed the role that our living spaces will play that that they play.

Yeah, it's in our patients health and well-being, you know, system type thing. I mean, hooked up with the well building people. We, we we've had some interaction with them in the Environmental working group.

And, you know, there are groups out there, but all the I mean, I'll tell you our research and I, you know, my sister's a year old, two years old. I mean, this is I mean, really cutting edge stubborn.

This is we're still learning as we go, you know, so so, you know, we're starting to gather a community of like minded researchers and scientists and professionals.

But it's still early days. It is, even though we're 20 years into it, it's we're still early days. So. But yes, we've been in contact with, you know, as, as as many groups as possible to sort of spread the word.

Well, fascinating work and, gratifying, I'm sure when you, you make the diagnosis and see the, the, the final stage come to fruition. And, I really appreciate your sharing, your expertise and, and, your knowledge on this, and look forward to continuing the conversation at some point, maybe in a forum or or, you know, the next time we I know we crossed paths, there's a surprising we haven't, as we do often.

Yeah. Again, thank you very much, Doctor Hammond. Thanks for having me. Appreciate it. You are. You are welcome. Let me stop.