Introduction to the Summit and Dr. Shoemaker

Good afternoon. I just an exciting day. We're going to, another edition of the Michael Thompson Chronic Illness Summit. And today I have a real a real pleasure, a chance to chat with, Doctor Richie Shoemaker.

Doctor Shoemaker has, sort of been one of the leading lights in my life. In treating chronic illness. He's. I had a patient come into me in 2001, I believe, clutching a book and a, and with a present of a visual contrast sensitivity chart and, I've been following it, admiring and enjoying Doctor Shoemaker's ideas ever since.

And, I've always been amazed at, how much he has to teach us. Today, we're going to start off with, a title that, I actually like. It's a radically different view of, mycotoxins illness.

And so, Doctor Shoemaker. Well, Doctor Gordon, you're looking younger these days. Covid, no haircuts. And I kept it up. So you asked me to talk, and I think we'll have a conversation.

I'm not talking, not a lecture. But there are some advances that have happened in the world of molecular biology that really do impact people with chronic fatigue as part of their their basic source of illness.

Now that we know that multi-system multi symptom illnesses share not only proteomic markers and we have in series we have over 20 biomarkers in proteomics.

And if we look at chronic fatigue syndrome, for example, another example of a of a chronic fatigue illness, we don't have any biomarkers at all. Not with myalgia. There's no biomarker.

So how can this be that one discipline of chronic fatigue medicine has got 20 biomarkers shown in peer reviewed literature.

Ribosomal Suppression and Mitochondrial Dysfunction

And here a much wider literature in fibromyalgia chronic fatigue syndrome. The experts having agreed on any. Yes. And they've wasted a lot of money looking well, we couldn't afford to waste any money.

We didn't have much to waste. The latest invention and, credit goes to Doctor James Ryan. Jimmy as he likes to be called. Yeah, I noticed early on when we were doing next generation sequencing and sequencing 50,000 genes, that was a whole human genome for a class about $2,000 a pop.

We didn't sell too many tests, but they the, the finding was that there was a marked consistency in ill patients of suppression of production of ribose.

So on the messenger RNA and the implication is that you don't have mRNA, for ribosomes, you don't make proteins needed for ribosomes, you don't make proteins for other spots.

And so, well, what would happen if a host had a factor that shut down ribosomal RNA in production? Well, the cell wouldn't do very well, but it probably wouldn't die along with that ribosome.

No problem. And large and small ribosomes both share the same findings. Doctor Ryan noticed that minor and ribosomes in mitochondria, mighty ribosomes also were suppressed.

Well that's interesting. And then if we look at mitochondria and as you know, there's only 37 mitochondria genes left in mitochondria themselves. The rest of my nucleus we talk about nuclear encoded mitochondrial genes.

Well, that means that in order for a mitochondria gene to have any place inside the cell, it's got to have RNA made.

Got a copy of the DNA. And if there happens to be micro RNA or long non-coding RNA or ribonucleic proteins around, that output can be suppressed.

And it is very frequently. So if we say there's a mitochondrial component to an illness, are we really just saying there's a mitochondrial gene suppression?

Well, if we look at examples of ATP synthase, which is suppressed as a bunch of genes, we know that ATP production, even if pyruvate gets into the mind right into the mitochondria, which will given that to say that ATP synthase reduction will follow.

So what if we have suppression of the electron transport chain cycle, oxygen axons, all that? What if those proteins are suppressed and the mRNA is suppressed?

What will happen then? You'll have reduced mitochondrial function. But does that mean there's a problem with the ribosome? Functionally, no. It's like the plan you've left on your desk for two weeks.

If you don't water, it probably won't do very well. You get some water. It's amazing how new growth will appear. But specifically, let's get to translocations because most people don't realize that the outer mitochondrial membrane is not just a passive barrier to flow of of one, two, or two things.

There are pores, or in some cases, voltage dependent anion channels about 20 nanometers in size, that must be open for the mitochondria to be fed. The water on the plant we're talking about on your desk.

That's how solids, ions, ADP, and pyruvate get across the outer mitochondrial membrane. And then mitochondrial proteins need the participation participation of trans low cases, trans variable location, location location, real estate.

But it also has to do with mitochondria. So if you don't translocate proteins made from RNA by ribosomes because it's not being made or they can't get through, will the mitochondrial function be normal? No.

So this whole idea of Doctor Ryan finding what an elegant mechanism. And we've extended the last four years to show we find this in heart failure, both dilated and dilated, congested.

And in this game, no different than we see in chronic renal failure. We see in college of Vascular Diseases, we see chemotherapy. We see in radiation therapy to a lesser extent.

But all of these findings are seeing is there a basic molecular mechanism to turn down energy production? Because that also turns down energy demand for the production to occur?

Or do we also turn down the demand for energy production by turning down the mechanisms that are needed to produce that energy? So if the cell is being attacked, and for sake of today's arguments, we're going to talk about revo toxins and we're going to talk about ribosome inhibitory proteins.

But we need to have a quick you know I just it to me just one second I just want to look sure that everybody understands what the ribosome is and does because, you know, is that it's where the, the, the RNA is read and made into a protein, okay?

And that's why it's so important because until the RNA is read, it's just a piece of protein of, of nucleic acid floating around. Okay. And mRNAs 2000 nucleotides long and micro RNA is 20 nucleotides long.

We need to account for all both of these different sized particles in our processes of my favorite thing, disease is are you ready? Okay. Lack of regulation, of lack of regulation, of lack of regulation of gene transcription for later.

Yeah. I I'm, I'm not sure about all four layers, but I agree 100% lack of regulation is the difference between health and disease. Absolutely. And and you'll teach us about the four layers if we continue on the hood two more hours, we may have time to double back and talk about that.

But okay, photons live in the nucleus, but they're not a nucleus. They don't have a membrane bound on them. And there's multiple gene functions for these ribonuclease proteins that we don't know.

There's over 300 proteins associated with nucleic acids sitting in Cahill bodies and speckles and para speckles. You know, you don't hear that on 60 minutes on the news.

But at the same time, what are these regulatory elements doing? And then if we got long non-coding RNAs, we're finding out more and more about it with what they do to regulate gene production within micron.

So yeah, I'm sorry. Just one more little definition. I'm going to keep bugging you on these because I don't want people to follow long non-coding RNAs.

And micro RNA seemed and correct me if I have more to do with regulating. They are regulators rather than things that we're busy transcribing and making into proteins.

And same thing with right with nuclear proteins as well. They all right. Or as best as I can tell there may be non regulatory functions. But at the same time if we look at the control of RNA, we look at transcription for example.

And there are some things and filaments that are involved with this. Epigenetics talks about methylation. But more importantly is acetylation. And more important is De acetylation.

And more important is demethylation. Right. So the regulatory aspects of gene transcription are remote. But imagine if you've been working hard in the fields all day making them RNA.

And you release it in the nucleus knowing that you given free passage to this drug of piece of all this 2000 nucleotides, it's going to traverse the nuclear membrane and get into the cytoplasm and you find the ribosome.

Isn't that exciting? Bercy defining event until a, you know, imbecile comes along a micro RNA and destroys that microbe, that micro RNA mRNA complex. What happened to your mRNA?

It was made normally. Regulation of DNA was normally functioning in my career. And I just shot it down. Oh, no. Oh, that wasted nucleotide.

Ribotoxins, Mycotoxins, and Endotoxin Injury

Is the nucleotide actually wasted or does it get recycled? Hopefully recycled. We can talk about autophagy another time. Yes they popped up. This is a viral infection in Covid because they're all tied together.

But there are no cell materials truly that are wasted. But anyway, I digress. But looking at record or lack of same, we're also looking at the model for a rival toxin system to disrupt protein synthesis.

Now let's take out your microscope, and I want you to look at a structure between the large and small subunit of ribosomes, both large and small ribosomes.

These subunits surround a little structure called the sarsen Reisen sloop. Now you've heard of Reisen, right. And you know it's very effective. Yeah. Killing people. Yes.

It starts in. And Reisen talk to me by the caster being. But how come it's got its name with this sauce and rice and Luke rice and exerts its negative effect on life by disrupting in this loop of nucleotides called the loop source, and rise and loop in the 15 position, there's an adenosine moiety.

You membered GCA and you're in your DNA. Well, this is the egg and it's part of a loop. And with adenosine there can be initial attachment of the three codon piece of mRNA called attachment.

There can be a sequential attachment of another three carbon three nucleotide piece called elongation. And then

there can be a stop signal that comes out another three codon nucleotide called termination.

That's how protein synthesis is supposed to work. The mRNA signal says put together 25 amino acids, 30 amino acids, whatever the code says. And you make the protein only with Reisen and source and sarcomere inhibitors, the names aren't too fancy.

They knock off the ability for that mRNA to attach, and certainly knock off the ability to elongate and severely knock off the ability to terminate. Oh my goodness, how do you get any protein made?

The answer is, if you're poisoned by rice, it very often you'll die. But what if you are poisoned by exposure to a crazy bacteria? One of the ones we know about was diphtheria.

That makes toxins, but it makes rival toxins like crazy. It's an action of icing. And lo and behold, the reason we don't talk much about mycotoxins anymore.

It's while they make rival toxins. They're crummy. You can activate very easily by glutamine, and they don't really do very much. About 7% of people with Sirs have micro toxin injury.

Compare that to action and they say it's the biggest genus by far in all. Microbiology. Yes, Pisces is of course way up there. And that collection of genetic engineering, but specifically they are 42% of Sirs patients.

And then lowly endotoxin. The thing we want to do ignore except if you're septic, is 28% of chronic illness. We know endotoxin by looking at neuro quant.

The distinctive pattern of loss of gray matter nuclei and dilated superior lateral ventricle and loss of cortical gray tells me almost a two to a two percentage point that any toxin was involved in initiation to this illness.

If you have problems with mycotoxins, you'll have a lot of inflammatory problems. If you have problems with endotoxins is brain injury. If you have access to my seats, is metabolic problems because I know mice, it's knockout complex, one with pure side ney and complex three with oligo mice and all the electron transport protein chain.

So if you want to know just just one minute, I mean, you just you have said a lot of very important things, but especially. So let me just go back for a minute.

So when you're seeing endotoxins, that's why you would be thinking more about those with the people with more neurologic presentations. Is that has that lined up clinically?

No. They have a 37 symptom program like all those patients do. So those three symptoms. But you can't distinguish the metabolic source of injury from the endotoxin mechanism of injury based on symptoms.

Symptoms remain. Some people have argued with me other than the HHS meetings for Lyme disease. I said symptoms are probably the worst mechanism to use for delineation services there is.

And someone else said, well, my line patients, they're the very best. And I said, your group of symptoms are no different than my representatives, and I know they are good.

Okay. Well, that's the thing. I mean, because we've been flying on the symptom train for a long time, you know, because I said in the past I always found the, the lab tests to be nonspecific for, for causation.

And, you know, you're right. And everybody's I always laughed like one man's bartonella symptoms is another man's the BCA center. So we can't be too, attached to that.

That's come on. All patients going. We're just exactly the same as when we shut up. Absolutely, absolutely. I mean, that, but there's flavors. I mean, at least we think there are flavors.

And the. It's all the same. You stir up ice cream, you got chocolate and strawberry. They all turn out to brown. What are you going to do? Yeah. Well, okay.

Well but but just going back. So. But you so what. You're just what you're saying is that even though, the get, you find that the endotoxin, predominant person will have more likely a neural quite a specific pattern on neural quant.

You really not directly relating that to symptomatology, but symptomatology remains the study of symptoms. And we're not really talking about that. I understand that symptoms need to be corrected for people to regain perception of health, but I also maintain that correction of objective biomarkers, molecular mechanisms is necess is necessary to eliminate disease.

I love it. Keep going I just I okay so perception of disease mechanism of disease which do we want. We choose the chronic fatigue syndrome world chased after symptom rosters for years.

Oh yeah ridiculous. Absolutely. You're right. There's no point in arguing that you know let's get something else. So the mechanisms then get to be one that we can say does one protocol fit all.

And if you have molecular hyper metabolism, the beauty of the one protocol that I've used since 1997 is that it corrects molecular hyper metabolism, it corrects proliferative physiology.

And if you don't know that you have that, you're going to see I feel pretty good. My symptom is saying proliferative physiology is not hurting me. That's right.

When a cells dividing making plenty of building blocks, there's no shortage of building blocks. And things feel pretty good. Cells aren't having a pop ptosis.

They're not dying all the time. But meanwhile the brains being rotted. Meanwhile, pulmonary hypertension has been published because of those exact same mechanism.

And Otto Warburg while he left at glutamine in his assessment. You didn't know where the ammonia was coming through in the cell culture, correct? It has to do with with glutamatergic pathways.

And we know that the synthetic pathways in cells. But the key issue is that you get a false sense security when you take an antifungal. You say, I feel pretty good and you're taking it.

You're consol that blocks the VDA. See, nothing gets through. So you say, well, I'm feeling pretty good. Meanwhile, your brain is dying. Meanwhile, your pulmonary pressure pressure's getting better.

By the time you leave the doctor, you go to a new one. He says, don't use antifungals. That's where we find pulmonary hypertension to be bad. That's when we find that the European is showing the complications from endotoxins or from antennas, which also block the DSC.

Well, you know, this is perception mechanism. I'm with you. We like to treat the mechanism when we can. It's as I said, well, we each the thing about medicine is that we see, each other's failures and we see where things go.

Symptoms vs Mechanisms in Chronic Illness

But you are learning and learning, and that's what's so exciting about the gene is, I think that you're getting more and more of these pieces, and I'm just very excited that you're putting this puzzle together for us.

So, and have you ever worked up somebody for a Tia? Yeah. Yeah, sure. And what do we say? Translate this schematic attack. Okay. It was translate. It didn't last very long.

A scheme, it was like a blood flow. But where was the attack? It doesn't show on Cat scan. It doesn't show an MRI with three Tesla coil, but it does show on a nine Tesla coil and Tias are now capillary hypo perfusion.

They go on to micro clots and micro bleeds. And in the areas of capillary beds there will be the presence of amyloid beta protein hooking on to coagulation compounds, creating an inability to have a clot in a microvascular circulation.

And with no blood flow going beyond the clot. Guess what? You've got distal hypoxia, which serves as a stimulus for what phosphor at all. So are we looking at a mechanism?

The vascular hypothesis, a neuronal injury? I wish I could say it was my idea. Sydney Strickland from University of Rockefeller, New York, is one has written more than that.

That is absolutely fantastic. But what was the vascular basis of the clot initiation? Is it a clotting factor that we can measure? Heaven forbid there would be a laboratory that would do all the clotting factors.

We can get compared if we look at the genes for clotting abnormalities. Oh my goodness. If the integrin deficiency BP six GP six GP nine where did all these things come from?

Where's factor five. Where's factor 12. Well there are two. There are two there probably 5 to 10% of what we want to, Yeah. Right now life life happens, you know, I mean, that has always been the frustration.

Is that the clotting, the clotting systems that we can look at, through, you know, regular blood tests are clearly only a tiny component because we see lots of people.

Everything's normal, and they still have hyper, you know, clotting events or micro, micro thrown by. Yeah, all the time. So I mean, again, so and with geni, you can see the growth, the, the, the genes that are upregulated and turned on or, or suppressed that can, that will create these clotting abnormalities.

Absolutely can. One of the exciting things was to correlate neurocognitive abnormalities with the vascular hypothesis. And the role injury. But what we can see is that the mean number of gray matter, nuclear atrophy, together with white matter and gray matter together, two areas is 6.15 out of eight.

If you've got four or more coding genes upregulated, just coagulation genes up in association with brain injury. The best part is when you fix the gene, we can heal the brain injury.

Okay. Now that's exciting okay. Yeah that was that in 2017 is on the survival website. Take a look at it okay. In this VIP. But we now know that VIP who actually coagulation abnormalities.

So what were we doing with this gray matter. Nuclear atrophy. Likely we're fixing the vascular source of that at all. Yeah okay. Yeah. And if you take away the, what I call your two, what you call the, the, that probe that, I forgot your word for the, proliferative, Yeah.

Proliferative metabolism. Yeah. You can let things actually heal. So scar already? Er, this is vitally important. But I'm not rushing. No, I did what you spoke Russian.

I know you did answer that, but proliferative physiology is a word that was part of your vocabulary. And I want it to be part of everybody's vocabulary.

If we were having proliferative physiology, we either are pumping glucose into the biosynthetic pathway to make new cells, or we're in a situation where we won't have pyruvate transported across the outer mitochondrial membrane to get into the mitochondria preferred energy production.

So that is lactic acid. If you've got lactic acid locally, there will be metabolic acidosis. Is that method of metabolic acidosis going to be stimulating phosphate all?

I haven't heard anybody explain that. But hypoxia and lack of energy goes together with lactic acidosis. So I think it will be a factor. But having said all that, as we go further down, what a geni report will show.

We looked at cytokines and that was very exciting because so many people will get a alumina X panel and get 15 cytokines, not knowing on average 30% of those are going to be wrong.

Yeah, yeah. Those are my most disappointing test. I, I do them rarely. And then every time I do, I'm usually sorry. Even more sorry are the people that use elicit testing for cytokine abnormalities.

There are so many similar structures or epitopes that those are not really very useful. So the question is how really are we going to assess protein modification, of which there's 300 different kinds that will affect proteins after the mRNA has done its thing and after the insertion right has done its thing.

You've made the protein and now it gets changed. That's where the diversity of the human organism comes in is protein diversity by modification. So it get all very complicated.

And how can you measure approaching accurately if it's been modified. Yeah with difficulty. So there we go back to gene analysis. And now in order to do gene analysis guess what you need Eric.

Wani a control group. And guess what's missing? Most of the time in people writing events and exciting findings, exciting discoveries is the null hypothesis.

Says everything you said is wrong.

Gene Testing, Clotting, and Brain Injury

And that's unfortunate. Science has that null hypothesis. But it's also often the case that, you know, I did this 50 times and it happened. You know, it's like flipping a coin 49 times.

It came up, you know, tails one time it came up heads. So it's 49 to 1 that's significant in that. Well, maybe it is, but the statistics aren't getting to the point that you can't prove it. Statistically.

It probably is not going to satisfy the null hypothesis. And gene transcription needs a huge control group. And I can remember Jimmy Ryan still to this day saying I need more controls, I need more controls, I need more controls.

So every advance that we make in science, usually there's a step backwards. And right, you get the idea. You try it and you find out where it didn't quite hold.

And then you can either adapt your your protocol or adapt your module, or ignore the fact that there was a failure and the data don't lie. And that's one of the things that really matters.

And so if we start looking at gene data, we can get those very nicely in any number of different places. We'll give you a Z-score comparison and point of five significance of the difference of a gene from controls.

And that comes vitally important when we start looking at some breakthroughs. I don't know if we've talked to you about in multiple chemical sensitivity.

Can I use another another great quandary? Yes, I'd love to hear yes, patients coming in because I knew they were suffering and I felt their pain, but I couldn't do much anything at work.

What I didn't have was a case definition. What is mix two? You might be mix to mean, maybe, but maybe not. You know, there is. Well, this is it was a preme court justice who said, I know pornography when I see it.

Well, I used to say, I know mix when I see it. And sometimes I just saw autism as I did. It's a very humbling disease. But specifically, if we look at an anti-inflammatory group of zinc finger proteins called licorice, there's three of these genes.

We add up their z scores compared to controls. And if we give a positive number, then the comparison to VIP

receptor one. Yes, that's the VIP receptor itself.

Who cares what VIP levels are? I want to know what the receptor shows. But if the receptor is negative an increase is positive. That mismatch correlates 85% with people with environmental sensitivity.

And environmental sensitivity can be a mobile chemical sensitivity, food sensitivity, or drug sensitivity. Fascinating a nonspecific finding. And like you said, it doesn't say what chemicals a person reacts to, just says they do pay attention.

Conversely, we had a case today. A great case person's been doing beautifully. Hyper metabolism has been fixed. Thank you. Tullio is doing a gorgeous job.

She's in New Hampshire. Everything's looking fine. Is the guy still has a brain injury and he has some anxiety that's not fixed? Well, he didn't have a mismatch of positive accuracy and negative VIP one.

He had the reverse negative accuracy and positive VIP one, which is associated with accentuated CNS injury. He had exposure to endotoxins. Guess what he had superior lateral ventricle dilation, cortical gray atrophy and five out of six gray matter nuclear atrophy.

So he had gray matter problems up the wazoo. And he's going to need VIP for a year to fix the cortical gray and spill out of ventricle. Well, we now know we can fix that.

Back when you and I talked to. Where was that conference, anyway? Before Covid, we didn't. Oh, right. Right. We were in Texas. Houston. That's right. Houston. Right. Right, right.

Anyway, we I didn't know about the accentuated CNS injury back then. Didn't know about MSCs back then. So it's a matter if you wait long enough. Sometimes you stumble out your door somewhere.

Yeah. That okay. So this is I mean because yeah mix is is just one of those things that yeah, yeah. We're looking to turn down the reactivity, but it's different in I mean, the reason it's hard to define it is because, as I say, for me, most of the people I see the diseases, it's their individual response.

That's what's got the problem. Lies. You know, it's not like it's not the lime. It's not the mycotoxins or the water damage building toxic. It's how their body responds.

Because, you know, ten other people have the same exposures in pure fine. So it's all about the individual but the mix or even a more refined group of that, because one of the more humbling elements in medicine is that if you find that you can identify a objective parameter with mix, you're part way.

There you go. Yeah. With the illness, you validate the illness in in skeptics eyes, insurance companies eyes saying, I can show you the gene basis or why I react to air freshener or cigaret smoke or any of the diesel fumes, and the list goes on and on and on.

But every one of those patients we saw and didn't know, we try to. Yes, we did, but they were telling us, I have a mechanism that's gone awry. That was all of my illness.

I may react differently than my friend and my neighbor, but I've got a mechanism this is bound to be one you can find exactly that has happened. And as we go further, looking in metabolic and maladies, beta turbulence to better attribute is a for a baby one.

There's a whole series of them are associated with the ability of cells to divide proliferate. And she looks at cell division. What is that microtubule is not able to attach to the centromere.

That section of the chromosome that has a kinetochore protein. That's where the microtubule attaches. Microtubules should pull one chromosome to one side and the other chromosome to another side of the dividing

cell divides down the middle and boom, you get two identical cells.

But if the centromere is not functional, the kinetic protein is not functional because the turbulence are blocking the, voltage dependent. An ion channel.

Will we have cell death of the birth of reproductive cha string? Yes. So this is a bad actor. So when I said literally, physiology gives you a false sense of security, you're also creating an element of death of the offspring.

Well, okay. And do those guys died peacefully or do they fall apart? I mean, are they really going through apoptosis or are they going through other kinds of, cell death?

And so what mechanisms are active for free pop ptosis for many that have viruses, you especially you'll see point activation of BCL two in our post Covid syndrome.

Our paper was published yesterday and I sent your copy of it to you. Yes, I read it sitting here. They are. Yeah. But the issue is that if we look at this viral evasion of apoptosis, able to this is the mechanism kill the cell and kill the virus.

It's in it viruses. I don't want to be killed. So it sets off blockers of apoptosis. You blocked the classical pathway. The, cell membrane never forms around the guts in like it's supposed to the cell contents, and the cell doesn't die.

But what if you have defective apoptosis, a gene? All right, A1 turns on the right console. Yep okay. One R. I. P three I learned three that there's there's a whole bunch of we don't just here but this is a mechanism to kill the cell without going through.

They have ptosis. So you release free DNA. You release ribosomes. You release very inflammatory. Yes boy talk about a cytokine storm. Now, I use that term in 2001 talking about Lyme disease when people got close to Army and then they felt horrible.

And believe it or not, cytokine storm is still in our lexicon thanks to the effect of Covid. It's not you. It was described 20 years ago. Yes. Yep. You know we saw it then.

So going, going back I said just just actually just just one thing for the accurate the Icarus. What what what do those what are they, coding for? Icarus.

They are group of anti inflammatory genes. When you have the Icarus turned on inflammatory genes get turned off. The best example is ApoE e4. And all of this genes that it's acted on is blocked by Icarus.

So if you want to have an ApoE4 you'd rather have Abo E2. You're not likely as a party to ask your event, but if you have problems with 17 T Reagan balance, you're going to have a cardiac event.

Whether you've got a belly for another, we can tell you that was with Jeannie as well. Okay. With that is. Yeah. You know, it's it's what's what's so exciting is how many pieces are coming together from different I mean, you know, you're sitting there, you know, like, are you say like, describing, you know, dementias, you know, and, you know, it just is so many different types of, you know, chronic inflammatory because, you know, basically, my worldview, chronic disease is chronic.

Environmental Sensitivity and Immune Reactivity

It's just dysregulated inflammation. And you're sitting there, kind of telling me that you've got a way of looking at the control panel for a lot of that.

When you add metabolism, you're not wrong. You were like, I told you, you take you. We go as far as science will let us go. And when inflammation is all we can measure, that's what we thought the problem was.

But people ism is shown out to be a problem with, you know, hypo metabolism, especially on a molecular basis. That's also saying that is added to inflammation and the metabolic abnormalities create inflammatory processes.

Yeah. Well I guess, you know, again, it's how you cut the circle. To in my, in my stories, the inflammation is often what's causing the hypo metabolic state.

Right. It doesn't really matter. I mean, because at the end of the day, it's getting the system back to self regulation and modulating. This is going to get us to the next to it.

Back to health. I think there's an expression to the rounder. We go the faster we get. We're chasing inflammation and chasing metabolism with metabolism and inflammation respectively.

The rounder you go, the faster you get. The answer is if we stop the right Botox and initiation of the protein abnormalities, good things will happen.

You get off the boat and you start using for that of words. Dear to my heart, I never use them much in the past they call cure. When you fix metabolism, you fix inflammation.

And you show that the steady state has been for six months means they're out of re exposure. Life's good. Very good. Yeah, yeah. No, this is this is excellent.

So, I, I as we're running through this, the, the fields, the fields of geni, you know, as. So how are you? You know, right now you're doing how many, you you're measuring about 100.

And you said 180, 188. Transcriptome or ribose, you know, actually. And then he showed that we had 2000 nearby genes that were upregulated or downregulated in our patients, compared to 50,000 all at all.

But in the question is, all right, what flavor of candy bar would you like? Johnny? You're in the candy store. You only get him. Yeah. So what Jimmy did was pick out 188 with the highest signal to noise ratio.

So the most bang for the buck. So the low signal ratio, like interferon, we don't include because it's just not good enough to separate out a room if we get a hit on interferon.

Is that signal for the noise. Right. And if you asked me to define how Jimmy figured out signal to noise, I'll refer you to the methods that he wrote there in our last paper, because I didn't write it, and I can't tell you that.

Okay. Yeah, it's just so. But you came up with the about 188 and, How are you? You know, right now, you're, you know, like, when you look at, like I said, you took the post line people, line patients, chronic fatigue, the the, water damage building, components, as you say, and the mycotoxins people, you know, how much crossover are you seeing or how how much are you able to really delineate the groups?

I used to talk in the lecture about peeling the onion. I think you might have given a similar talk yourself to one thing at a time. Yeah. Which out of the kernel or inside?

We have to do multiple things at a given time, because multiple changes of drug intervention of a given protocol will be occurring. It's not any onion at all.

It's many onions, all being peeled. Some are being peeled faster than others, but each has to be peeled to get to the final kernel of all of them. That's where the cure of this mistake that I made was thinking one intervention, one change.

It was one intervention and multiple changes. I didn't know about happening with the one I did know about it. Yeah. So that that's when you work with the body.

Yeah. I always I mean, that's kind of what we depend on when we work at the body. You know, that's what healing

is about. We do one thing and then the body goes off it goes.

A lot of stuff. You know, we said it was easier to fix cars. You know, actually, it's easier to fix people sometimes because, cars don't heal themselves.

People do. So, you know, get with the idea that you don't have to be right all the time. If you start the process, sometimes the body will continue it.

But getting back, I want to get back to the genie test because I'm really I said I, I, I was interested in it, but it sounds like you've taken it to a new level now.

It's something I really want to understand. So are you able to delineate just, you know, I mean, I know you feel you can delineate treatments, but you can delineate the, the major drivers, if you will, or you just.

Are you predominantly looking? You look, you say at the mechanism and, you know, and again, very appropriately, I think, not worrying about what necessarily triggered it, but what's going on in the individual today.

Well, if you read the paper we published in March, we've been publishing a lot lately, you know, seeing for the first time depiction of precision specific causation.

We're looking here. People are exposed to actual lysates. We have an environmental sample we're in. Does we find more than 16, 16 or more species? We also find immune reactivity with Map kinases, low level reaction, mycotoxins, very high level reactive.

But we also then have the third component that if you got exposure confirmed and you've got napkin is nonspecific. But stimulators a whole series of downstream anabolic and catabolic events, both reactive and you have TGF beta one receptor activity.

We have got specific causation. Scott McMann one of the word precise come in there. He has medical legal reasons for wanting to include that. But specifically for the first time, we can tease out antennas out of the myriad, the vortex of amorphous items that 32 different categories of things that make people sick known in water damage buildings. We can also, maybe daughters having a tweetstorm now also looking at endotoxins with specific causation for them.

CD 14 into for our specific random toxin. So in answer of can we separate lime out. Yes. We use the work that came out of San Francisco by the way. Yeah. Yeah.

Okay. And Charles Chou from UCSF and John Ackerman from Hopkins, Maryland's got to get their name in there somewhere. But he looked at people with acute Lyme manifests by a bite and and rash and an illness.

And so he had a rash in there so that if you didn't have any rash, you maybe weren't covered by a study. But he treated people, for three weeks with doxy and then didn't give any more antibiotics.

Of course, it's Hopkins. And then look at them. Finally, six months later, what changed was still abnormal with a whole slew of them, of course, because they were just Asians from line that were not being treated.

But we can sort out those genes, and that's one of the elements we have undiagnosed Lyme disease or line, that's been treated within six months. So that's made a big, big progress.

If we've got something we worry about Bartonella, we look at defenses because other bacteria, other viral infections are there. Bartonella lights up like a Christmas tree.

So it's really there's no one says like a Bartonella. And they went to galaxy and maybe they had a test. Maybe they didn't, you know, as in ice where it had.

We were both giving a talk at the American Society, Tropical Medicine Hygiene years and years ago, and the elevator got stuck between the first floor and the in the lecture hall.

So we had 45 minutes to talk in the dark about Bartonella and Ed talk. Most of the whole time there's 28 different species of Bartonella. He'd identify.

They were pathogenic in dogs, right? How many were pathogenic people have Bartonella and I know it. And I came along in 2003. We had a fellow who actually is, you know, it's 2003 for the first time.

He published a silver at that, I mean, stain of Bartonella in the lymph node. And that was exciting. So we took a lymph node out of a patient with Bartonella who had cat scratch fever, as we call it back then, erosional layer.

And, I probably pronounce it wrong, but we sent it off to him. He grew it out of our lymph node. The first in the world. Oh, wow. But he's again, you can get calculus, Cassie, from lots of things. We.

A guy with a monkey scratch was a cat. He was in a zoo. We got a cat scratch, and there was an upper trochlear node positive for Bartonella. Unbelievable.

Plumbers working underneath cross braces and all that is a problem. Yeah, yeah. No, no. Bartonella is is is a nasty. So the defenses definitely pop up.

You know, I mean, the other thing that, you know, we're looking at some T-cell reactivity these days to get a better idea of, you know, when, when the bug is actually active.

But. Yeah, well, tell me that would be interesting. Correlations. Tell me about your T-cell assay, because if you've got CD, 3D deficiency, that is the CD marker for the interaction between an antigen presenting cell in a naive T lymphocyte.

If that gene is downregulated, you won't have that interaction. You won't have T-cell reactivity at all. Well, that could explain some of the maybe some of the negative tests and people who we think have, Bartonella.

But, you know, you know, it's that is always the problem with, with every test when we're looking downstream, like we'd like generally we do in medicine, you know, I mean, this has always been the issue.

If the immune system isn't working the way we want, that's what we've always been able to look at is just immune, you know, like ECGs. And now and you know, and also T-cell reactivity.

But yeah, if the communication isn't working, it's not going to show up. If we go back to Covid for a minute. Defective antigen presentation is of concern and vaccination.

I get the question every day in here about should I get vaccinated if my my CD 3D is downregulated? Well, chances are that you run a risk of having defective antigen presentation.

And the negative test is an antibody based test. The antibody is dependent on having normal antigen presentation all the way through, and there's no guarantee that's going to be the case.

So that's why physiologic elements make more difference to me that antibody tests yeah. Yeah. You know and I maybe there but the yeah you've got it. Yeah. That's right.

You poor T-cell. Yeah T cells I mean this is but one of the innate. Well every area is so dense. I mean you're immunology. See what.

Post-COVID Physiology and Treatment Strategy

You know, far better than I think that, you know, we just keep finding more, more, more different cell types and

more different ways that the cells communicate with each other.

You know, I mean, years ago, people thought that B cells and T cells, you know, they didn't realize how B cells have huge amount of regulatory, effects on T cells.

I mean, it's a everything's in communication. The body is one big soup. There's an editorial in nature Reviews Immunology years ago just talking about the different phenotypes for natural killer cells.

You said there's 28,000. Okay. Let's say you're wrong. There's not 28,000. There's 228,000, right? Right, right. I mean, this this is. Yeah, we we we have to, split and lump in order to talk about things.

We just have to remember that's often not what's really happening. But it's what we need to do. And you've done so just as an overview. So with the gene test now, how are you using it clinically?

I use it to look for let's just start with post Covid people. TGF beta one signaling is gigantic. We've all known about pulmonary fibrosis, and interstitial lung disease was dependent on, endothelial from this angle transformation or EMT run by Smed turned on by TGF beta one receptors.

Well guess what. That's a huge player in post-Covid syndrome for people that have Covid and have recovered. Some people have symptoms that affect activities of daily life.

Other people don't. If you're still sick six weeks later with impairment of activities of daily life, I can almost guarantee you the 64% of your of the people like you will have endotoxin reactivity and 56% will have, actual mice's activity.

This is incredible. The Covid patients are released to go as a deformed host, as effective host, effective antigen presentation host into an environment that was safe before.

But now, because these organisms live in homes hugely throughout the US, they are now exposed and now going to pay the price. So is the gradual accumulation of new symptoms a result of post-viral and I had an expansion of of the transcriptome, or is it new susceptibility, new priming events to old pathogens?

We could even fight off before? I think that we cannot determine that until we treat or something like, well, one cortical star, I mean two, three weeks before jumping in with the IP, which will fix TGF beta one receptor.

So now that we've got specific layers of treatment, we can conclude specific layers of injury. Yeah. Well this sounds similar to you know I think you said this I don't it's been a long time I, I, I always liked it though was that, you know, we did see that many people did not become quote unquote.

You know what. Well water damage building sensitive or mycotoxins sensitive. Until after they had, you know, an infection like line and then later became, you know, much more sensitive to their, their environment.

Is that something you think something similar is happening or exactly the same? I use the term a cytokine priming event. I need to be a cytokine storm.

And it was really having one person that then my take place repeatedly in Connecticut never had a problem until I had to get a package by the handful stinging me at 6 or 7 stings, and shortly thereafter they started getting Lyme disease, which was something was changed with HLA.

Something was changed with the molecular control mechanisms. Why? That's not a quite a bit. I don't know what it is in Covid. I wish I could say it's RPC one if this is something not just RPC one, but that's an intensely inflammatory element.

So we use it for post-Covid syndrome. We use it for people who don't feel well. We use it for people that know they've been in a water damage building.

We use it for people especially. They think there might be toxins. They're still sick. They take antifungals and it doesn't fix the mechanism. But if you go after the physiology, antifungals have their own presentation right there.

We can see it basically a shut down back in tubulin. That was it. Okay. And how are you changing your your your you know, your program. I mean, as far as the steps in your program based on on on the gene or is it just making feel that you better know what you know?

Oh, yeah. So I mean, what are you doing differently based with with Jeanie haven't changed this order of events in the Or the steps at all. I've been using a low dose VIP program for people with environmental sensitivities, as well as a run up to using any medications.

That's. That's been one change because we can show correction of CD3 d with that one. But specifically when I jumped in with Jeannie, found that by skipping steps in my protocol, I actually was not helping anybody at all.

And we still say remove from exposure a month of binders with minimum, continue with binders or eradicate mark ons. We did eliminate did be able to show that if mark ons is present, but my auto ribosomes are normal.

You don't need to worry about the mark hunts, but if mighty ribosomes or abnormal there the polycyclic ether toxins were made by mark ons. That's been the real bugaboo, is that we were able to show in multiple mastic studies that these unu unknown polycyclic ethers were made by mark ons, but they're not in any library.

The fragmentation. You and I were going to run a business, right? I know we wanted to do something with that, so I wasn't able to come through on that.

But because this is huge, because that's always been the issue is, you know, who's more cons to treat? Okay. You're right. You're fine. You don't have to treat. Take it for me.

What was that again. Does your mind. Ribosomes are normal. If the miner. Okay. Well, the I mean, right there is a is a good reason to do the geni because I mean that that that there's a lot of mark times and, you know, you just hate to, to be, you know, treating what's not going to, especially since so many times the more cons is, you know, you treat it and, you know, it's one of those things that if the immune system is able to get rid of it, you know, six months later, it's back again.

It's there. And, you know, it's just a it's colonizing you. And it could be in many people. It is just a commensal. Obviously, we had to retreat away from bug spray because, yeah, we switched over to Silver Nathaniel.

Silver things, shiny silver mixed up with EDTA that did fairly well, which is what preparation came out. And guess what? Every attempt to kill and eradicate by killing didn't work.

If we suppress biofilm formation, the normal immune responses in nasal mucus, which are fascinating. You know, there's 400 organisms at a given time, and each milliliter of mucus that people make when you blow your nose is for organisms, for a million organisms, for any organism sitting, sitting on your Kleenex.

What are they all doing? They're fighting for their own place. You've got fungi. I ten of my CS bacteria fab. And every mucous membrane known. You got fab. You find one out of proportion.

Yeast. Vegemite is well, what really is dropping out there. Actos regulate anymore excel for class example daughter fossilize suppressed. So if you go out of C out of the world of mark ons and you've got too much bacteria, you bring that back into play with plain EDTA for six months.

It's inexpensive, it works. You wait until the normal micro biologic environment reestablishes, but all you do is you put things back into pecking order.

I like that, I like that it does it. It does make it. It's clinically it's what we've seen also is that focusing on the on the biofilms, yeah. Is getting good results.

Yeah. That that's you know it's amazing how many people's brains clear just with that, if you don't have polycyclic ether, you got a chance to start clearing your brain.

Although as much as other people like to say spontaneous healing of neuron one has not occurred yet, we have a 5000 neuro quads in our data and I haven't seen it yet.

You need to use medication, the app usually to fix brains. Yeah. No, no. Absolutely. Absolutely. If you missed. But in terms of mechanism, are we looking at correction of gray matter nuclear atrophy by self-healing. No.

Well turn off the inflammation. And you know what? I just I don't want to digress for because we want to talk a little bit more about the, you know, work with the water damage buildings, but just a little bit, in fact, you know, what I'm going to do?

I think I'm going to just wrap up this one because I want to keep it to an hour and then we're going to come back because I, I want to talk a little bit about Covid and hyper coagulant.

It's just one of my little, favorite subjects. I just want to hit that one. Maybe we'll do that just before we end. And you, you can tell me what you're what you're feeling is been because when I, I didn't, I hadn't seen a lot of Covid patients, just by the nature of, I live in a county where they locked down hard and people didn't have to go to work.

Those who I see. So, I mean, it's really it's amazing how, a socioeconomic disease this has been, you know, on some levels, you know? Okay, well, when when we come back, we'll start with the coagulation and engine controls cases peaks negative and positive.

And the p value. Very good. Just one check in here. So.