Introduction to Eric Gordon and Chronic Illness

Hello is Doctor Kent Holter with another episode for the Peptide Summit. And today we'll be talking with Doctor Eric Gordon. These are talk about, metabolomics, mitochondrial cell danger response.

So a new way of looking at these chronic illnesses and, you know, how they relate to health and how peptides can be used to kind of fit into the picture.

But really looking at a different way of looking at chronic illness. And I think you really, enjoy this. And I've known Eric for a long time and a great friend and see him at almost every conference.

And, it's he's been around. He's, president of, like, Gordon Medical Research Center and founder and owner of Medical associates, private medical practice in San Francisco Bay area, specializing in complex chronic illness.

In addition to his clinical practice over 40 years. Hey, he doesn't look over 30 to me, though, but, he's engaged in a lot of clinical research. He created a series of medical symposia, bring together, thought leaders in the international medical community, focusing on chronic fatigue syndrome.

M. E., Lyme disease, autoimmune disease, autism, in a collaborative mode. And he really loves collaborate and has innovative medical practice. Is he's a university researcher, which is but his lifelong dream is collaborating with, these multiple forces.

He did a amazing paper with doctor, Robert Novo on metabolomics. And we'll talk about what the heck that is, mitochondrial function, chronic inflammatory disease, and is really bringing his his dream to life with all this, amazing new stuff and new ways of looking at things.

In 2016, as I mentioned, he, coauthored, paper groundbreaking, paper with Doctor Novo, the metabolic features of chronic fatigue syndrome, published in the proceedings of National Academy of Science, or prestigious journal.

He's a medical advisor to Tech Bioscience and is the, GMA, collection site for Lyme Disease Biobank providing samples for researchers. So it's really doing the right thing and really searching for, you know, what new ways to look at things and and researching to find, you know, cures and treatments for these illnesses that people don't believe in.

And it's interesting if a doctor doesn't understand it or doesn't treat it, it doesn't exist. It's the patient's fault. And it's it's interesting. We'll get into it where they did a study that, you know, they can tell the history, chronic fatigue syndrome, fibro.

If I imagine normals, or doctors. Oh, there's no studies. Although we do our just our panel, we can pick out someone with chronic fatigue syndrome, fibromyalgia, chronic Lyme versus a normal about 70, 80% of time.

And how bad it is. So it's kind of like I think the chronic fatigue syndrome diagnosis, fibromyalgia diagnosis did a disservice to the patients, allowed doctors not to look at anything underlying.

So you got this here's a depressant. But, I'd like to welcome, Doctor Gordon, thank you for coming on our summit. We're really looking forward to, talking about all your, information, wonderful knowledge base and sharing that with other.

Thank you. Thank you again. It's a it's a pleasure. It was really nice to hear how you, you know, just how difficult it is treating people who are not respected because the doctor doesn't understand.

I mean, that that that's that's been the bane of my existence. I got into, what we used to call alternative medicine in about 1990, 92. And, it always and, you know, always amazed me how, you know, the sicker the patient, the more the automatic eyeroll of the doctor, you know, than the, you know, the positive review systems and we have so many nice ways of saying that, a patient is, you know, not sick or malingering or.

And it just broke my heart over the years to watch that. And, you know, we see these people. And the more you saw, the more you, the more you saw, you know, because in the beginning, I was kind of the same way.

It's like, can this be real? You know. Yeah. And more and more, you know, doctors. I've one patient sits in the ICU and the doctor orders a psych consult, says it's all psychological, you know, like, yeah, this I can't.

So I just had a patient, unfortunately, passed away last year. Young woman, 39. And and, you know, that was that was, you know, she had been told at least 50 times that, you know, she needed psych consults.

I mean, they, you know, in all the best places. Oh, yeah. And it's like, you know, I'm sure I'm the same as that. You know, people start crying. Just the fact that we're working them up and believe them and we're like, yes, we've seen this before.

Like, but, you know, they told me this is crazy.

Why Chronic Patients Are Dismissed

You know, that I'm crazy. And then they start wondering if they are crazy. You know. Well, that, that that and that, that is, is that is a problem. And that's what so excited me about this metabolomics.

Okay. Because, I had been looking my whole life as you for markers. What can we measure for people, you know, and, and we all, you know, we jumped down, you know, we we we always jump on, on the bandwagon of measuring this test in the test.

But at the end of the day, they still just tell us that the patient is sick. You know, we have a lot of innate immune markers now thanks to Doctor Shoemaker.

You know, Rich has been a great help. But I have a little disagreement. I just don't think there's specific markers. You know, these are just markers that you're in for treatment and a lot of a lot of things.

Yeah, yeah, yeah. You know, I mean, we started do measuring his stuff back in 2004. And you know they're are they were they are exciting. But there's so much like that out there.

You know, we have so many tests that tell us the person is sick but we don't know. This doesn't really tell us what's wrong. Just their immune system is triggered, you know.

And, and that's been the problem with getting our patients respect without a test, we're kind of left to that. Well, we know that these people are sick.

There's there are very few diseases. I mean, Horowitz is very good at pointing out to a very few diseases that give you transitory or through allergies, which just doesn't happen.

That's why. Right. You know, I mean, they'll basically say a standard, actually start now. They don't have Lyme, but they've lost the power of basically exam and, you know, review of systems in history.

Like what else does that. Well nothing else. Well now your test is negative you know. So what's explaining it I don't know. It must be psychological. Yeah.

So you know, what the heck is metabolomics okay. So but I mean, you can say it any which way you want. I don't know the metabolome a lot. I don't know myself, but I, I guess it comes out of me as metabolomics.

And then you did a George Bush there. Yeah. Yeah. Okay. Nuclear. Yeah. Okay. This is looking at the small molecules in the body. These, they they call them small.

They weigh about less than a thousand Daltons, which make them, you know, like the amino acids, glucose, the small, the smaller, small proteins. But most and, and the fats, you know, and these are we always looked at those as you're basically your raw materials for the body, but they're also the end products of metabolism.

So metabolomics measures, you know, the raw materials for metabolism and most of the end products, you know, if you think of it, the genes are the blueprints, okay.

The enzymes tend to be the machines and the and the metabolites, because that's what we're measuring with metabolomics is the metabolites are the raw materials and the end products.

And what has been so interesting about looking at metabolomics is you start to see that, they are what is controlling a lot of the information. The this is the mechanism that allows for epigenetics.

Okay. We talk a lot about epigenetics, but epigenetics basically is the, you know, whether you methylated or assimilate or methylated to stimulate the histone.

So you begin to expose different parts of DNA, which then will, give you, another, it just so the viewers like with epigenetics, it's how the environment affects the genes.

So if you have a gene for like diabetes, let's say that, if you someone, two people on the same gene, one person eats great know. But the person's not so much, that person will get it, but someone who doesn't have a gene.

But they eat terrible. They'll still get it. So the environment affects the genes that are, produced. But, yeah. So data and metabolomics is the way that this is, is the intervening step.

Okay. Like, because, say, if you have too much glucose, you begin to like, like, like light, because glycosylation of, of, proteins. And you also, change how much, insulin is made.

You know, that happens because you're turning genes on and off. And so the doctor Inovio, got into this because he initially was doing a lot of work with, important areas of metabolism.

That's that's usually lipid disorders in kids, things that will often cause death in the early in the first ten years of life. And if, of course, you're doing a lot of genetic work, that's what he's mostly doing.

But as the more he did, the more he saw that genetics doesn't predict much. Okay? Because even two kids with the same gene defect have vastly different prognosis.

And we see that all the time. Take, you know, thalassemia or any, you know, anything. You know, you have the gene, but some people have symptoms and many people are asymptomatic.

It depends on whatever else is happening in the environment. And that's where the metabolome directly talks to the genes, turns them on and off. And and when you're looking at the this, metabolome, the metabolomics, are you looking at like it's basic taking all these things, measure the amount of certain things or you look at specific markers because there's so many, how do you interpret that?

Well, that that's the thing is that, I mean, whether there's 2000, 3000 or 5000, they're still arguing. What? Generally what can be measured is somewhere between 500 and 1000 relatively easily.

What Metabolomics Measures

You can measure and know what they are because in metabolomics, you can do, metabolomics is done using these days using mass spectrometry, mass specs.

And you know, it's the kind of device that you can get a ton of information, but you just know something is there. You don't know what it is. But when you measure chemicals and identify them, usually you're measuring about 4 or 500 and you can really identify what the chemical is.

And what we've been looking at. And that will take the chronic fatigue as an example, is, at that point and I just want to make it very clear that paper, which is a brilliant paper was, is, I supplied to patients.

Doctor Nabi supplied the brains. It's your I know you did more than that. So you're very basically, you know, I mean, I just want that quick, it's, a he had.

He is an amazing thinker. I really think that if you if you actually look at his website and some of his ideas, I just really, really, I think groundbreaking because he's got a very eclectic background.

He was originally a is an MD who was a PhD in virology and then went into inborn errors of metabolism and has a great interest in evolutionary biology as well.

So he all of this together very broad and and I'll do some art. His recent articles that on the cell danger response and on the healing cycle I think are really worth looking at to get a broad view and we'll come back to that.

But, just, just so patient, people out there can get a fine copy of those, articles. Look up cell danger response. Yeah. Do you. It's nab I a you x you know and he press it novio if you just stick that in and cell range or just even Cd-R your paper will show up.

It's there. I mean the same thing. Chronic fatigue and chronic fatigue and Nabi. Oh, the paper will show up. He's also done a lot of work with autism.

And I'll touch on that at the end if we have a minute. But what we what we did in, chronic fatigue, he looked at between 4 and 500 chemicals in patients, 20 males and 20 females.

And that's one of the important thing about metabolomics is that sex, age matter, okay? And it's not just the sex hormones. Men and women are significantly different, even subtracting out the sex hormones.

And it's just like they say, women are a little more complicated. I think if I remember correctly, if we, if we, we can hit a very good differentiation of chronic fatigue patients from healthy patients in men looking at about eight chemicals in women, it took at least 12.

And even then we probably because they're a lot more complicated than we are. But that was the beauties. What we saw is that, you know, we found basically about 40 chemicals that were two standard deviations from the mean from normal in people with chronic fatigue.

The other thing that's very interesting about and he's done this in, PTSD, using, people from, you know, in the Navy, before and after this, you know, in Afghanistan and Iraq.

He did it. He's done it in depression with a large group from from Denmark. He's done it in Gulf War syndrome, you know, chronic fatigue. And he's now working on a Lyme cohort.

Yeah. And, you know, when we look at our panel, you know, they're you're looking at 400 things and we do like 35. You think we're crazy? We do. You know, so many things.

But it's we we see that, Lyme patients, autistic patients, Gulf War syndrome patients, they look the same, you know that. I mean, that just, that, you know, everything just like, wow.

With this, they have this common pathophysiology. What we'll talk about. Yeah, the common is, is, is inflammation in different the inflammation stuck in different parts of path in different pathways.

Because one of the things I mean this is just an aside, it's that we're seeing is that we did to chronic fatigue studies which we oh we one of them has been published so far.

The other one is kind of in the can waiting to come out. And one of them was done almost all with my patients. I shouldn't take my patients. Your patients from my clinic.

And the other one was predominantly patients of Paul Cheney. I don't know if, you know, a lot of people don't know about Paul anymore because he's kind of gotten quiet.

But Paul was one of the original people who, you know, Paul Cheney and, and Peterson, you know, basically woke everybody up that this was happen. It. Yeah. He got a lot of arrows, too.

Yeah, yeah, yeah. But, but the thing but what's interesting is that Paul doesn't quite believe as much in chronic Lyme as we do. I think you're saying. Yeah.

You know, and one of the things is that I, we get a lot of the people you know, in the chronic fatigue group started off with Lyme disease years ago. We treated their line and their joint pains went away there a lot of their symptomatology went away with.

They were very we were very careful to exclude anyone who had typical Lyme symptoms. What they were left with was, to me, the hallmark of chronic fatigue, you know, post exertional malaise.

They couldn't their lives were limited if they did, you know, whatever it was, if they did two hours, four hours, whatever, if they passed that they crash the next day or late at night, that when they did that.

There's so many of the academic articles on chronic fatigue are not talking about chronic fatigue. These guys, the the people in academia do not understand chronic fatigue.

Yeah. Yeah. Yeah, I wholeheartedly agree with that. Yeah. They they confuse I mean, there's no way you can confuse a depressed person, okay. With someone with chronic fatigue.

Yes. Some people with chronic fatigue get depressed. But you know, when your family abandons you, when you don't, you can't function. You can't. And you wouldn't be depressed.

You may get depressed, but that's not what's wrong with you. And you know, and anyway, this is so getting back to so what in our panels. What was so interesting is that, what we saw was these group of chemicals called single lipid, single mylan's and Sarah mites, things that I had never heard of.

I mean, I had heard of in medical school, basically, actually, when I was in medical. I don't know if you can talk about that. I know Christine been in fire.

Yeah. Okay. Right.

Metabolomics Findings in Chronic Fatigue

Pretty much I mean, you know, I always left the, the, Oh, what is it? See, I'm getting told can't remember things. Anyways, it doesn't matter, but I would just say they were.

They were. The library was not as big. And in the 1970s, it really wasn't a prostate gland. It's. There was there was maybe three papers in volumes on prostaglandins when I was in medical school.

Okay. Now you could fill a library with things on prostaglandins. That's how far we have come with information. We haven't learned that much, but we got a lot.

Yeah. So the thing is, is that these lipid, these, glycol lipids, okay, are a lot which have a lot to do with exosomes,

interestingly enough. You know, the exosomes are made up of these glycol lipid envelopes a little bit a few proteins inside.

Well these are what's abnormal, significantly abnormal in people with chronic illness because they're the they are the signals that there's chronic inflammation in the bloodstream.

So because I just one of the problems in metabolomics is we're looking again at the blood when many times the dysfunction is in an organ. And so we're still looking at just the drainage from the system.

And that's why a lot of signals get lost. Because if it's your liver, you know, or your kidneys that aren't doing so well, but the rest of the body is doing okay if you're, you know, obviously, if you're measuring the end product of what the liver does, especially with the kidney especially, you can discriminate, but sometimes you can't tell when it's just a little dysfunctional.

Or maybe 20% of your liver is not working. Well, that's where the money is. Someday we'll figure out how to really check that. But yeah yeah, yeah. On the cell signaling on the on the cell surface.

How do you get blood to check that. You know. Yeah. Yeah. We're we're we're checking the whole system. But we can see patterns. And that's the important thing.

The pattern of inflammation. And what's interesting is the patterns. So the patterns are similar enough amongst different symptom groups like the Gulf War syndrome are significantly different from the blind people, from the chronic fatigue people.

Okay. But they're all in that same ballpark. The single lipids are off, but they're off sometimes in slightly different ways. Yeah. What makes it even more interesting, okay, is that amongst people, as I said, we had about 40 metabolites that were abnormal, you know, in the chronic fatigue group and about, you know, 25% were had to do with having chronic fatigue were shared, but 75% of the abnormalities were individual.

But based on that person's biochemistry, genetics and environmental exposures, like all the other things that they have exact. Now, can someone order these tests or is this all well and not yet.

Yeah. I mean, there are people selling small panels, but not yet. I mean, there's a company called metabolome which you can order your, you know, your metabolomic panel from, but they're giving you I don't think you get really much information like that's the thing.

How do you interpret it without looking at the rest, comparing it to the right. So at this point below, mixes is still in the experimental field. I mean, we had the tech bio that you mentioned, we started Tech Bio with the idea that we were going to measure, we were going to be selling Metabolomic testing.

This was back in 2000 and 16 when we first and the first paper came out with, oh my God, we're going to be doing this. And then we turned around and we discovered something is that the information really didn't give us in chronically ill people a lot of treatment help.

Yeah. What what do you do? Thanks. Yeah. I'm sick. I know I'm sick. Exactly. Yeah. Would have been. We would have had to charge $1,500 for the test, and it would have just told you you have chronic fatigue.

Now, if we had a big enough sample size and I could have made an FDA diagnosis, you know, then maybe still a lot of functionality, but that's exactly.

But it because what would what I had hoped. Okay. Just like I'm sure you is that if you give me enough numbers, enough chemicals, I'm going to make, I'm going to change things and and this goes back to the big lesson, that has a lot to do with the cell danger response.

But the big teaching for me is that what we saw is that just because something was high or something was low,

correcting that didn't change the system, sort of like we talked about in the beginning, the right supplement doesn't always change the system when somebody is chronically ill, because what we're dealing with is compensation.

Yeah. And it's not a cause. It's a result, you know. Yeah, exactly. And, you know, being a doctor, we got so used to if, you know, if you're low in blood, we give you blood, you know?

Right. The blood pressure can be lower and everything's a vicious cycle. And you know where what caused what the. You know, the genes. Great days. And then they affect the genes.

So what? You know, where where do you go? And everything, every system seems to be affected. These patients and and where do you start. You know, one causes the other causes the other.

And doesn't matter what the initial thing is now it's this big vicious cycle. But with like the cell, dangerous monsters we'll talk about. But mitochondria are key, right?

Actually, let me just a one of one of, another another doctor Bob aphorism, is, you know, like the path to health is is not the reverse is not reversing the path to disease.

You know, again, because that is the way we often approach medicine, because we are medicine is based on a, a trauma, a the story of trauma. You know, we really good if you broken traumatically, we put you back together because the instigating event was the trigger and we just have to repair it, you know, just remove it and let the body heal.

Unfortunately, with chronic illness, that doesn't happen because the instigate, the thing that's making you sick is your body's response to the trauma.

Just like we see with our Lyme patients. You know, there's a I don't know, a significant number of people. You kill the bug there better. But there's even more that we get stuck with.

You kill the bug. Yeah. There's no there. There's still there. So anyway, so they'll show you the mitochondria of how to think differently about disease.

And it's the mitochondria is, mitochondria have different forms. I mean, obviously we know you have like, different mitochondria in your heart than in your fibroblast.

And let me just mention that, you know, mitochondria in the cell to early medical school, they're basically an evolutionary organism that was probably separate and came into the cells, but they were taught they make the energy for the cell.

That's kind of what they do. That's what we're taught in medical school. And that's what you asked, you know, 99% doctors. That's what they're going to say.

So I just wanted to explain what that is. Yeah. Right.

Cell Danger Response and Mitochondrial States

And that type of mitochondria that form is Bob has labeled M2 and you can ignore the, the numbering. Yeah. I usually get them confused anyway. But he did.

So but this mitochondria, this is the mitochondria that's functioning in normally in your body. And its job is to produce ATP efficiently. Okay. Energy.

Right. Energy, energy. And, and it makes some of the raw materials that allow you that your body needs for, for growth. But its main function is to take the energy that's produced in by burning glucose and make that efficiently into a lot of energy.

Okay. By passing the electrons down the electron transport chain. Now that's the M2 form. Now what happens is that if that if that mitochondria starts to sense that it's not getting its share or its normal amount of raw materials, it browns out.

My analogy is that it's like the old days when you knew, you know, the vandals were coming, the, you know, the barbarians were invading. You would, like, burn the fields, retreat into the castle and seal the walls.

And so hopefully they would they would they wouldn't be able to survive long enough to lay siege. And they move on. And so we do the same thing that mitochondria, when it senses that it's not getting enough energy, it stops making as much ATP.

And by doing that it increases the mitochondria when you make ATP is when you use oxygen. That's when your body's using oxygen. Okay. You can burn you can burn sugar without using oxygen.

But and we call that anaerobic glycolysis. But, what we when the mitochondria are involved, it's aerobic. It's using oxygen. So when that stops oxygen concentration goes up in the cell.

And that creates a very oxidative, rich environment. And so you have to your nucleus then starts to pour out your antioxidants as a response. Okay. So but the other thing that's happening at that time is the mitochondria also start to take some of the ATP that it's still making and put it onto the cell surface.

And it becomes a signaling molecule and ATP, adenosine triphosphate and and all of its derivatives, especially things like things that we think about like adenosine, are cell signaling molecules.

These work like, paralleling hormones. They're giving information to the immune system and this nearby cells that there's danger here. There's a problem if this happens on an ongoing basis.

I mean, this is kind of how we learn when our cells actually put a little more ATP on the outside. If it's a nerve cell, you learn faster, okay? Because you focus.

I mean, you can't focus on every every input that comes into your body. So you have to know which ones to focus and games. Your focus is you at all levels.

Because this is a very conserved, this has been happening since we were amoebas. Okay. This is the first thing. And what happened and it creates what we call sickness behavior.

You're fatigued, you withdraw socially, you lose your appetite. You know? I mean, all these things are happening because you're making less energy. Yeah.

And it's like how people get the flu. It's not the virus causes something. It's your body's response. Exactly. And what happens is that the mitochondria are directly involved in creating what they eat.

The increasing interference into, yeah, interfering one group which is made intracellular and, you know, starts to make the inflammasome. And the thing that makes you feel sick.

Okay. So that's the that's the normal. Well, that, that's that's what happens when. So you have the the normal mitochondria are making energy. We compute.

Then it goes to this M1 which is like this battleship form of the mitochondria okay. And it also causes changes in the cell membrane. The cell membrane thickens.

It starts getting less permeable because it's defending itself. It's less if you stop communicating when the cell is really injured like an injury, it stops communicating to the cells next to it because it's got to heal.

So this is this is what's happening, when it's healing. And then the next state is what we call the M0 stage is and

this is this is what happens when you're making energy by burning sugar.

But the mitochondria have turned on again and are now using oxygen. So there's no longer oxidative stress. But you are now using that ATP to make, you know, you're not making ATP.

You're using the electron transport chain to make intermediate chemicals, things that make all your pyrimidines, which is part of your, DNA and RNA, you know, and also chemicals that will help make hemoglobin and, acetyl CoA to help burn your fat.

So you're making all kinds of things that help the cell grow. This is what happens in a cancer cell okay. But this is also what's happening in your normal cells when they are rebuilding after injury.

Okay. And at that point they're not communicating well with each other. And this is this is one of the things if cells and then if the next step is they go back and the mitochondria are now operating normally.

But now you're in what we call the third stage of the cell danger response. And the cells are just beginning to begin to communicate with each other. And if they don't fully go back to normal communication, we now have senescent cells, which is one of the dangers of aging.

It's not too dangerous. That's probably how we do a lot of this aging process of slowly falling apart is that we have more and more, cells that haven't been able to come fully back online.

They're working. They just don't have enough energy, and they're not communicating. You see, this is the thing is communicate, you know, remember that.

Cool. You you're old enough cool hand Luke. You know, what we have here is a failure to communicate. I mean, and it's interesting and, you know, with the mitochondria, I think, yes, almost every person is chronically ill.

What's their body temperature? You know, it's low. We'll check everyone's basal metabolic rate. They come in the sick patients. They're usually 25% lower, you know.

So they're burning less oxygen making less heat. You know, you're making energy or creating heat. And they're all low and, they don't eat anything and kind of, you know, no gain weight unless they have, you know, terrible gut issues or can't eat anything.

But, it's it's interesting. So, yeah, we see that commonly, I think more and more, even on a subtle level where people, they don't feel horrible, but very low metabolism, where they're kind of that early phase.

Yeah. It's it's it's where you get stuck, you know, that's the thing. Different diseases get you stuck in different places. And, and it can be happening in different.

See, what's confusing is that we always want to make blanket statements, but it can be happen. You can have cells in your body that are stuck in the CD1, you know, and then you might have a lot of scarring and hypertrophy of tissue, or especially if it's clear terms are CD2, you'll get a lot of scarring, you know, and when you have a lot of inflammation, because it's usually then you're still signaling the neutrophils to come cause problems in CD1 because that's, that's that, that, that, that acute phase of inflammation that in some people doesn't tone down.

So it helps us think a little bit about this. What I'm hoping is going to do eventually is help us treat people, you know, as we as we can measure metabolomics and enough people and eventually even be predictive.

I mean, to me, the really hot thing is, if I can measure your metabolomics at age 30 and see where you're headed, especially through the lens of the genetics, you know, we've all been frustrated by the genetics because, remember, 20 years ago we thought, oh boy, do we're going to open that book of genetics and get all the answers.

And lo and behold, we opened the book of genetics and we mostly get confusion. You know, you know, and that is

that is a problem. And I've got all these genetic tests going out, which I do like them is that.

But I'm really correlating them with, you know, with metabolomics and, you know, or if you're seeing the results like I people have, you know, myself, I have like every gene for that's terrible.

I should have high homocysteine. But I don't, you know, and so it's yeah, it's interesting I think. Yeah. Using it and correlating it with what's, what's happened with what's happening because we, we yeah.

Genetics. Genetics is, is a mess because we didn't realize how much is, is variable how you, we, we thought it was a binary system on off and it's not at all putting it.

Yeah, yeah. And and but but if we combined it with the metabolomics and the history of what we see, we can see where things are important and then treat appropriately instead of putting people on 35 supplement, you know it my, my, my whole thing in life because I said I went through too many years with the with the shopping bags people would literally walk in with, with two shopping bags full of supplements.

Yeah, yeah. And I hate that, you know, I made a lot of money with that for a while. But I do like it. And it kind of goes into like treating a healthy person versus a chronically ill where, let's say treating the mitochondria, that you can give them all these things CoQ10 and but if it's not working, it's not going to help or healthy person may get a little boost if they're low.

Right. But the chronically ill you gotta look at a little differently. Absolutely. You know, if you some of the things we were talking about there, there are you know, there are some of those peptides that seem to work just in a very, benign manner.

They always help almost everybody or they don't hurt. And there are some of the peptides that seem to have some double edged sword to them.

Using Peptides to Support Healing

And that's because I think they're the ones that will work, powerfully in that CD2 mode, when the body is in that place where it is acting actually like a cancer cell.

But we want, you know, that's a time when using some of the, some of the interventions that will help the cell not kill itself is really good, because you don't want to lose a lot of brain cells or a lot of heart cells, you know, until you're waiting for the, for the for the vasculature to, to rebuild, you know, you'd rather not lose them because they, they grow back, but not really.

Well, yeah. So so you would like to prolong their life. And so we there are some peptides that will help do that which is really great. If you have a stroke or a heart attack.

But if you've got a tumor it might feed the tumor because it's that same metabolism. The tumor is in that same metabolic phase of burning sugar to grow, and using the mitochondria only to make raw materials, not using it for energy production because it's so it's that that's the important thing of, of of where I think understanding metabolome and this was understanding metabolomics but thinking about the cell danger response, thinking about the cycles of what's happening in the body.

Now, it's not easy because, it's just we're just we're making it up, learning it all at the same time, you know, getting that thought process going of like, am I dealing with a person who is going through just a, a normal healing process?

Okay. After an injury? You know, there's that six month, it could be a normal healing process. Or am I dealing with somebody who is stuck in the early phase of inflammation and we can't get them out of there, you know, and that would be that would be a time.

You know, I think something like the BPC 157 is because I think that's what it helps do is post, mobilize and get you to continue the healing phase. Right? Right.

Yeah. And it, yeah, there's so many, things that it does, it helps you to basically, heal and rejuvenate. Lower inflammation. Yes. What what are the peptides that do you like?

Well, at the BP, BPC 157 is one of my favorites because, I a lot of the, a lot of the patients, I don't see a lot of patients anymore. Okay. And the patients, a lot of the ones that I have are people who have failed, not just what everybody else does, but also what I do.

I send them out, they come back and, you know, and we tried everything. Yeah, we we get counseling mentally, you know, 10% better here, 20% better there.

I, I don't live in the world of home runs anymore. I miss that. But, the but anyways, with those folks often so many things are inflammatory, you know, so the BPC 157 I've been amazed that even people with major mast cell issues have been able to tolerate it.

Yeah, and that's more and more becoming a big issue with these patient pots. But really, I think it has to do with so many of these chronic infections.

You know, basic mast cells are the allergic cell. But they we've learned that they basically secrete tons of inflammatory products, not necessarily just histamine, an allergic reaction.

So yeah, calming those down actually, part a lot of Marcel Marceau groups that are still stuck on directly inhibiting mast cell, but really looking upstream and affecting the immune system, which affects mast cells, I think is much more, affected me.

That's. Yeah. I'm I'm with you. I mean, that was something, when Doctor Afrin first started writing about it, and I, I, I had dinner with him in the first.

He's he has changed. The first dinner I had with him, he he didn't quite get how important it was to deal with the infections, but now he does. It's really I mean, I'm really impressed with that man because not many people were okay.

Yeah. And you know, especially when they when when they think they have the answer, you know, and he, you know he really. Yeah I post about on on that group but I said you can kick me out of the group.

But this is what I found that works. You know, it's against. But even these, you know, and these guys are considered real smart, dedicated people in there too.

And I've seen you post a lot too. Yeah. Yeah. It's it's just it's nice to see but it would. But the mast cell thing is one thing that, you know, Bob pointed out to me, the mast cell is the original immune cell, the first in the mold.

And when you have a multi organ system, you know, system, the first immune cell that popped up long before T cells and B cells and macrophages was the mast cell.

So that's why it is. So it seems to be everywhere and do everything to our patients because it's primitive. But it makes like 200 different chemicals. And you know it and it's it's so good and treating it I love your idea.

You have to go a level 1 or 2 levels up to really help. So fix the immune system and probably the most important thing, the hardest. But sometimes the hardest thing is, is get that sympathetic nervous system off the roof, because that's what, you know, when you, when you stay ill, you can't help it.

You're, you're you're activated. You're you're, you know, you're. Yeah. Then it gets turned down. The same thing is. But if we can turn that down, we can also really ameliorate the mast cell issue as well.

And and a little perlite, lecture on is that one major stimulator to mast cells is quite a bit releasing hormone. So you know, basically the sympathetic nervous system you're going to stimulate cortisol which part of releasing hormone stimulates activates that secretes cortisol.

So these patients will have even high normal cortisol. But their HGH is very high. So they're just, you know, in fight or flight or just give a little more cortisol lower that activates that lowers that CRH all the time to just get better.

Yes. And that's the wise use of low dose cortisol is it is a true art. And yeah, you got up in some people. Well, it's, Yeah. That. Yeah, it totally, you know, in a way, when you think of it, that really is, you know, it's the, you know, you know, cortisol is really not an amino acid peptide therapy, but say my arm, I get all my arms in high places.

Yeah. And and actually BPC will actually lower that CRH or lower the active. You can't check CRH. And when they're high cortisol you know I don't want to give them cortisol but their ACH is even higher.

BPC will actually, lower that. Thank you. That that's a really interesting pearl. Yeah. No, I like that. I, you know, like many people, I started off using BPC, you know, focused on the gut.

Yeah. And and has the gut is very you know, we're finding more and more that, you know, gut brain, gut everything axis. And it's really again that epigenetics, you know, goes along with that.

And you just, kind of getting close to ending here, like the famous ends. Oh, I love. Yeah. The, the the time and again in my population, I want to start off with this time is in beta for before I'll use the alpha.

You know, I just find that, you know, it's. Yeah, it's often in people who are really have very reactive systems. I need to mellow the immune response a bit, you know, and then just kind of for everyone watching that.

So if you, if you look at this oversimplification and you have kind of one, which is the intercellular, immunity in to which it goes to 817 but, and usually everyone's out of balance.

So this is kind of the inflammatory autoimmunity, VPC lowers this tb4 kind of is, modulator. Both thymus and alpha one really increases, for what's good.

But it also is to really kill infections. And you can have more reaction. To that. So it's often good to start with the BPC that that lowers that, that to and lowers that, that inflammation that people can tolerate.

And it's a little side note to find myelin, probably it's much more anti-inflammatory than all the other famous ones. And for hearts is that we use that.

So just a little side note, you know that that that is always important because it's it's it's balance. I mean, and you know, when, when somebody is staying ill for a long time, you know, okay, you know, it's there's no rules, you know, always want to make rules.

But there aren't any. You do because you think it's the same thing with like, the L 37 stuff. You know, it's a really nice immune just goes out there and like gives information immune system, you know, kind of kill.

But you know in a lot of people health it's it's it's really easy. But in in my patients I go very very, very, very gently. And unfortunately I don't think we can get that anymore.

Yeah, but you can. But it's hard. It's getting harder and harder. But electrons are just softeners. It's, antimicrobial peptide. So it's very broad spectrum viruses, bacteria, yeast.

But also like BPC, you don't think of it as an antimicrobial peptide, but it kills viruses. Candida. And, you know, one study showed that it, killed the herpes virus at 1/100 the dose of a cycle.

Beer.

Mast Cells, Fascia, and Clinical Integration

We will have BPC with k pv, which is alpha monocyte stimulating hormone, which is like Milana Tan makes you tan, lose weight, a Barbie doll, peptide. It's a small fragment that which doesn't cause the pigmentation, but amazingly anti-inflammatory and anti-microbial.

I would love that, because, you see, in the old days we, we used and I still occasionally used, you know, when we could get me. For now, we have to use the melatonin, but used to get MSH, you know, along with the IP, those are the I mean this is another contribution of shoemakers, you know, I mean like those again they really help reset the imbalanced immune response.

You know, I was very, very, very impressed with those. So I think that's the those are really those are really important ones. Yeah. It's nice for that.

That should be out probably the next month. But yeah great studies on plan for bowel disease and autoimmune. So yeah, a lot of literature on it, which is not.

Yeah. Yeah. No, no, that, that is, that is, is fascinating. It's fascinating stuff. And you know, the microbiome just to say is that that's the other thing.

We can see a reflection in the microbiome. We can there's about 30 probably more, chemicals that we can measure in the, in the, in the metabolome, in metabolomics, in your blood that are clearly from your body doesn't make them.

It was it was the bugs in your gut and a lot of them. And it's interesting with this gut brain access that it goes both ways that the gut, you know, based on fluids, the brain, the rest of the body, but also rest of the brain influence the gut.

So it's like you give all these probiotics, but then all of a sudden they're it's back to the way it was, you know, because the influence, the other way of what's going on in the system.

So it's it's interesting. Yeah. Everything's a chicken or the everything, you know, that my, my, my favorite thing to say is, yeah. Two favorite quotes. One of them is everything works.

Sometimes I've used your line. You get it actually lecture thing that I just have to say, I mean like, I, I love I, I've worked with some brilliant practitioners over the years who, who have, much more patience than I do and are able to, like, stick to one therapy for a while, and you really get to see the full beauty of what it can do.

I, on the other hand, tend to be a, you know, I'm I'm like, yeah, I'm adding I'm moving on. Yeah, I'm moving on. Okay. That didn't work. You know, I mean, and which is a shame because I have learned thankfully I had these people around me.

So I watch and I learn through them that many times if you wait three months, it does work. Yeah. That's not the max I. Yeah, but and that's the problem too.

It gets very complicated. If you do treatment A it works. It doesn't work. The treatment doesn't work and it does work, but they do it together. It works, you know.

And so there's so much to it. Yeah. So yeah, I was going to go to my point I was going to make is that when once when people are stuck I just it's not the, I just I really believe that so much of chronic disease can be ameliorated, maybe not cured, but ameliorated and helped if people pay more attention to the fascia and the connective tissue in the musculoskeletal system, because that is often where you're getting that, where the glitch is in the feedback from the central nervous system to the organ, and vice versa, is because those nerves are often very sensitive to, facial tension.

And to be honest, just like, you know, the acupuncture meridians are probably flowing through the fascia. And if that's all tight, that's when you acupuncture just, you know, it doesn't do much.

Yeah. And it's interesting. I know Doctor Neil has talked a lot about the fascia and go along with that with just, practical. We have, a stem cell doctor and he comes let's say you have rotator cuff right now.

We want to check in there right next to the stem cells, right into where the, you know, where all the problem is. He does does it sub kind of into the fascia?

Right. And you're like, how is that going to work? And ten minutes later, the people have full range motion. It's it's incredible where they go like, okay, this this is, this is ridiculous.

But, you know, it's sort of like, you know, I mean, basically neural therapy, you know, is where we we put information right below the skin that feeds into the sympathetics and they're going right to the brain.

I mean, it's this body is just so amazing. I mean, like, you know, doesn't matter. I mean, that's like my dream is to have is to have the team of of, of people who can dance, who can treat different ways, but who could actually work together.

One of the difficult things where you have seen over the many years that you've had your clinic is how hard it is to get the a team willing to work together.

That's, yes, herding cats. Yes yes yes yes yes. Oh yeah. You're good at doing that. We were just good. So I think we're, running out of time here. Okay.

And just want to say it's just, Yeah, I was, I hope I, I hope people learned something. I, you know, it's often hard to convey new, complicated ideas, in, Yeah, in a short time.

But I think the main message is that there are more forms of mitochondria than we knew, than we've thought about before. And, a one. Yeah. And they do actually change shape in the mitochondria while they're changing the when they're in the normal form, the M2, they're long strings.

They kind of connect to each other. When you go into that danger zone, they break apart. They stop connecting. And yeah, I think we're studying very old study where they did muscle biopsies and chronic fatigue syndrome patients and their mitochondria were very different.

Oh yeah, about 80% of another conversation one day that isn't the only way to peptide is, a riff on a recent study of showing that serum from chronic fatigue patients actually has some interesting effects on normal people.

But anyways, there's so much to talk about. Can. You are so cute. You make them sick. Yeah, and we actually make them healthy. Not healthy. You make them resistant to viral infection.

Interesting. So yeah, it's all set up to fight. Yeah. Yeah. It's at a price. At a price? Yeah, yeah. Nothing's free. Well, always wonderful to to talk to you.

Always, new things. So we really appreciate you being on with us. And, thanks so much. Okay. My pleasure. Thank you. Well. Bye bye.