Dr. Leo Galland, MD - Can Peptides Be Utilized to Combat Viruses? Transcript

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Introduction and Speaker Background

Hello. This doctor can't hold talk with another episode from the Peptide Summit. And today. Love welcoming the esteemed Doctor Leo Gallen. He is going to speak on the role of angiotensin, pathway with Covid 19 and the use of peptides in the treatment of Covid 19, and also for inflammatory bowel disease.

Doctor Gallant, received his, international recognition as a pioneer in nutritional medicine and a master clinician, a graduate of Harvard University and, also New York University School of Medicine, where he trained as internal medicine physician at New York University Bellevue Medical Center.

He has received the Linus Pauling Award for creating basic principles of functional medicine from the Institute of Functional Medicine and the C League Magnesium Award for his work in nutritional supplements for the American College of Nutrition, and at the Albert Norris Markey Lifetime Achievement Award from the Markey of Who's Who for his contribution to health care.

He has listed annual Americans, top doctors, and a leading physician in the world. And he has authored seven books and 47 scientific articles, multiple textbook chapters.

So he's he's done so much, during this 20, 20, he has been working, intensively develop innovative nutritional strategies. And, peptides for coping with the SARS, Covid, two pandemic and developing, an innovative peptide based therapeutics for the treatment of Covid 19.

So I would like to welcome, you, Doctor Gowan, and thank you so much for being part of our summit. Thanks for having me on and giving me the opportunity to share my analyzes and observations.

And yeah, you've just done some fascinating stuff. And, just your knowledge base is just incredible. And trying to figure out where where to start. You want to just maybe talk about, the role of the angiotensin, Ace2 and agents.

So one seven signaling that that peptide, in, Covid 19 and in health. Sure. I think that's really important place to start. And just by way of background, the angiotensin system in the body it has is very complicated.

It has effects on virtually every organ, and impacts on inflammation, blood flow, blood clotting, tissue repair and growth, blood pressure. And it has essentially two arms to it.

One arm raises blood pressure, promotes inflammation, promotes scarring. And has been and has been studied for decades. And there are drugs that were developed to block its effects called Ace inhibitors and angiotensin receptor blockers.

The other arm, which is what's critical as a balance to the first arm, is the one that is impaired by Covid 19. And this is this involves an enzyme called Ace two, the, which is in essence its effects are the opposite of Ace, the original enzyme that was discovered, Ace two, breaks down the most inflammatory, the most constricting, of the ends of the forms of angiotensin called angiotensin two.

It splits angiotensin two off and creates a peptide called angiotensin one seven or ang one seven and one seven has numerous effects in the body that are very positive and that have been studied.

It is neuroprotective and it improves blood flow. It, stimulates tissue repair. It supports the bone marrow. Virtually every a manifestation of Covid 19 can be traced back to deficiency of angio.

Seven of angiotensin one seven. So in order for people to understand that, I want to go, back to the way in which the virus enters cells, which is critical.

ACE2, Angiotensin, and SARS-CoV-2 Entry

And and I also want to say that the early discussion of of Ace two angiotensin converting enzyme two, in Covid 19 went off in the totally wrong direction, a direction that has since been rejected by, all aspects of the medical establishment by scientists because it it really cell to understand deeply the role of Ace2 in Covid 19, the SARS-CoV-2 virus in order to enter a cell has to attach to Ace2.

It has to fit into it like a key into a lock. Now, there are three enzymes that are essential for SARS-CoV-2 to enter your cells. Ace2 is the fulcrum.

It's the most important. But by itself, Ace2 will is not enough. First, is that the surface of the virus where there's, something that's received a lot of attention called the viral spike protein.

That surface, which is the key that fits into the lock, that has to be primed and altered. And there's an enzyme on the outside of cells called furin.

Furin that has to alter the viral spike protein to allow it to attach to Ace2. But even that's not enough. Once the viral once the viral spike protein has locked itself into Ace2, the Ace2 has to be split, and there's an enzyme on the membrane of the cell, called Tmprss2, to which splits that protein.

So it's really it's a triple whammy. You need furin, you need Ace2, and you need Tmprss2. All of those have to work together to allow the virus to enter to gain entry into the cell.

Now, why is this important? It's important for many reasons. First is, if you can block one of these enzymes, you may be able to prevent viral infection.

You do not want to block Ace2. It is a vitally important enzyme for your heart. Your blood vessels in particular. And that's that's where it's probably most important kidney function, blood flow, heart function, and the brain.

It's also neuroprotective, and it's anti-inflammatory, which is the reason that, all of these weird and mysterious complications of Covid 19 can be traced back to the deficiency of Ace2 and Ang one seven, which is created when the virus enters cells.

Now, what's important about, furin is you can block serum, and if you block furin, you can presumably interfere with viral entry into the cells. And, the most studied and potent furin blockers are actually peptides, poly arginine polymers of the amino acid arginine can block the activity of urine.

And they've been studied in animal models of infectious diseases like anthrax, for example, where they're where they're very effective as adjuncts to treatment.

What's critically important about Tmprss2, the third member of this triad is the not all cells expressed S2, and it's the co-expression of Ace2 and Tmprss2 that's needed for the virus to enter the cell.

So there was a lot of talk back in February and March about how well Ace2 is, the way that the virus gets into cells. So you don't want to have too much Ace2.

It was totally wrong. You need it very simplistic, and you need as much Ace2 as you can get to protect yourself against the negative effects of the virus, which is destroying Ace2.

The limiting factor is not how much Ace2 there is, it's the co-expression of Ace2 and Tmprss2. And so what's interesting, for example, is that Tmprss2, production is stimulated by testosterone.

That may be the reason why men are more susceptible to getting sick with Covid 19 than women, because they have more Tmprss2. I think. I don't know, like if it's zero pregnant women or, you know, very few pregnant women have been, infected.

So also estrogen stimulates Ace2, activity. And so the Ace2 and attention molecule basically gets cleaved to this angiotensin one seven that has a kind of an opposite effect.

Right? Basically what Ace2 does is it it's it it creates a 180 degree turn around in the in hormone balance in this angiotensin system. And it takes, an essential but dangerous peptide, which is angiotensin two, a peptide that raises blood pressure, that interferes with kidney function, that promotes inflammation, that promotes fibrosis and scarring it.

It takes that promotes blood clotting. It takes that very dangerous great. And it splits it over time. It's almost like a superhero movie or something.

It flips it over to the opposite. And one seven, which is anti-inflammatory, inhibits blood clots and thrombosis, inhibits scarring and fibrosis, improves blood flow and circulation, improves kidney function.

And and that is the fulcrum on which the outcome of SARS-CoV, SARS-CoV-2 infection, swings or rests is Ace2 is so critical and, it's showing it with the angiotensin one seven and really looking into it and it does so many things like you're saying, you know, boost mitochondrial function, which associated with almost every disease of aging, you know, chronic neurodegenerative diseases, it's shown to improved memory, shown to, right, allow the heart to recover and less damage after heart attack.

And so, so many benefits to that. Right? Right. Just across the board and angiotensin one seven has been studied in animals and humans. It enhances recovery from intense exercise.

Eccentric exercise. It for women who have had breast or, or ovarian cancer, who have received chemotherapy or are getting chemotherapy, angiotensin one seven supports their bone marrow and prevents the low blood counts that are the result of chemotherapy.

And it works better than the drugs. The standard drugs in that case, in animal models, it reverses, dementia that is associated with, poor blood flow to the brain.

And it, does beneficial effect after beneficial effect. You can use it to, heal the lungs. And if it isn't angiotensin one seven that's used, it's Ace two itself in animal models of every type of lung disease.

Whether it's infectious, including sores, the original sores, or whether it's toxic, infusions of ace to help the lung to heal. Angiotensin one seven does the same thing, prevents blood clots.

One of the complications of Covid 19, is what's been called happy hypoxia. It's these people who have very low blood oxygen concentrations, but they're not exactly short of breath.

And the question is, what's happening? These are people who should not be put on a ventilator. They do need oxygen. But the reason it's happening is that the blood flow, blood flow through the lungs is all messed up.

It's not regulated properly. Angiotensin one seven restores normal blood flow through the lungs and raises you get that total mismatch. So blood going through non forget, mud blows to the areas that are not being ventilated and and not to the areas that are.

And so the oxygen goes up goes down, the carbon dioxide goes up, you get pulmonary failure. But it's not really due to lung disease. It's due to a vascular disease disease of the blood vessels.

And what's interesting is that, you know, you're naming all these studies and I've been reading all these studies and you hear these reports on TV, it's like, did doctors not read all these all literature?

It's been done. It's like they have to reinvent everything. It's like a new discovery. I'm like, yeah, that's been known for 40 years. Like the clotting and sepsis.

And you know, we're looking at like the peptide thymus and beta four for, sepsis. It's basically gone with people who have sepsis. You give it back. They don't have all those problems.

And it's the same thing. They don't look at old literature and they just come out with something and then come up with a new theory like. And so some of this is actually pretty new literature and they're not looking at it.

There are some centers that are looking at Ace two or angiotensin one seven to heal Covid 19. And there is a research group, a couple of research groups at the University of Arizona, that are looking at that or applied for ion DS for that.

There's a research group at University of Pennsylvania, and that's worked with, with a group at University of South Florida. And, and they're looking at this there is a Canadian and European, joint, academic venture looking at infusions of Ace2, in the treatment of Covid 19.

There's been some work done at Johns Hopkins on that. I mean, it's they're major, highly respected medical centers are doing it. It's just not getting the attention that it deserves because everybody's talking about a vaccine and and that these, antiviral drugs.

But it is there and it, it really it should be utilized now because people are dying now and Ace2 and angiotensin one seven can really mitigate the outcome of Covid 19.

Yeah. And the antigen one seven is orally bioavailable, which is right there isn't. There is a form that's orally available. The but it's also available.

It can be used by injection. Main problem. And it may not be a problem is that it has a very short half life. That is if if you just use pure angiotensin one seven, it hasn't been modified to expand it's half life.

It'll disappear within 30 minutes from the blood. Despite that, maybe it doesn't need to be around for a long time.

Furin, TMPRSS2, and Blocking Viral Entry

Maybe once it starts that signaling, you know, it's like a a bowling ball that knocks down all the pins. Yeah. The only takes it only takes a few seconds to get down, to initiate that.

And, there was a study in, in mice that were exposed to whole body radiation and a single dose of angiotensin one seven given once a day, or sometimes only for a couple of days after the radiation, not even before it was able to restore the bone marrow.

So it just takes a little bit to, to move things in the right direction. It's an initiator of healing responses. And I think that's true. We find that doctors, you know, don't seem to understand there's between, you know, serum half life and physiologic half life.

Right. Absolutely. Where like hormones the basic go in the cell nuclear receptor. They change protein synthesis. So they're going but they, they're they're gone out of the serum by T3.

They go, oh it's such a short half life now it's about two days physiologic half life 2 to 3 days. So yeah yeah yeah that's that's a really important point.

So yeah. So I've been pushing since March, the beginning of March of this, 20, 20 for angiotensin one seven as, to be available as a therapeutic strategy in Covid 19.

And actually, I'm, working on and I and it's, being submitted to the FDA, and I hope that by the, time that this summit goes live, that that will be available now.

So you're doing the just one seven and then also the fear in which we're going to time, right. So, so that that's

another, that's another concept that I came up with.

And that, also needs that also will need and I and which I hope will be in place and just so you know, now in a nasal mist or a nasal spray, after you've been exposed to Covid 19, will inhibit the furin in the lining of the nose.

And maybe that's the only place you need furin inhibition, because the virus will not be able to enter its incubation chamber. The nasal nasal mucosal cells.

And so it doesn't there won't be enough of the virus, replicating because it really appears as if how sick you get from Covid 19 depends on the initial viral load.

You know, in the famous episode of the Sackett County Choir in Washington, where there were 60 roughly 60 women in the choir who were singing together in a closed space for two hours, practicing social distancing.

They were six feet apart. The attack rate was 86%, which kind of indicates that there's a lot of aerosols of this virus. It hangs out in the air. You're just not just getting it directly from someone else's breath, or coughing or sneezing.

One person in that choir had a cold. So they thought, thought that person had Covid 19. 86% of the people in that choir got sick. Two of them died. That's an infection fatality rate of 3.7%, which is very high.

It's likely that the high viral load that people, were infected with initially had something to do with the with a very bad outcome in that group. So what the current thinking is, is that there are very few Ace2 Tmprss2 molecules in the lungs.

There are some, but not nearly as many as in the nose. And that the way that pneumonia occurs, it's not directly from the virus that gets that. You inhale from the air, it goes into your nose, the virus multiplies in the lining of the nose and reaches a critical amount that really raises your own personal viral load to the point where the virus can you can inhale it into your lungs, from your own nose, from your own respiratory tract, which happens, of course, all the time that it can get into your brain, following the olfactory nerve from the nose up into the brain, and that it can get into your blood vessels.

And so if we view the nose as the incubation chamber blocking furin or Tmprss2 in the nose, may really prevent serious infection. And just just for clarification of furin is an enzyme, protease.

Correct. So it. Right leaving. Right. Furin is a protease. It's a category of enzyme called proprio Tene converts. It takes one a protein, one protein and converts it into another protein.

Basically, and it plays an important role in infectious diseases and in cancer. And so there's been a lot of interest in finding ways to block furin, to treat, cancers and, and also in certain infections.

Now, furin, most of the furin in your body is inside cells. We don't really care about the intracellular furin because that's not where it's important for viral transmission.

A small amount of it travels through the cell to the membrane and goes through the membrane. And it's kind of coding and hanging around in the in the space around the outside of the membrane.

That's the furin that we want to target. That's the furin that has the prime, the viral spike protein for attaching to Ace2, which is embedded in the membrane.

And, doing that is not likely to have any adverse effects intracellular, because it's whatever you're doing is not getting into the cell where most of the furin is just dealing with this thin coating outside the cells.

So the poly arginine peptides do that very effectively and irreversibly and have been tested in, animal models, as I mentioned, of anthrax. For anthrax to make you sick, it has to oh, there's a toxin that it's produced and that toxin gets activated by, by furin.

So with animals who are infected with anthrax, if you give them, the poly arginine, you can interfere with the effects of anthrax toxin. And there's another model, that was used in which, mice had their eyes infected with Pseudomonas and pretty nasty bacteria.

And Pseudomonas produces a toxin that requires furin for its activation. And you can treat the infection by using eye drops of poly arginine to boxing up your.

I think the furin is also responsible for species different species transmission and also the flu virus needs fear and correct. Yeah. There are a lot of viruses that use furin, for modification.

Actually, the Merz virus used furin to b to alter itself to bind to its receptor, which is not Ace2. Now and here. This is really, I think, one of the things that makes it clear that furin is really important, the original SARS virus, we'll call it sars-cov-1, that did not require furin.

That virus was very deadly. It's probably on an individual basis. It was possibly ten times more deadly than SARS-CoV-2, but it was not readily transmitted.

That whole epidemic of SARS back, 15 years ago that killed about 800 people and infected about 7000 people in the world. I mean, this, SARS-CoV-2 is a thousand times more lethal, not because it's more deadly on an individual basis, but because it is so much more readily transmit it, it from person to person.

Now, the biologist who has looked at the, at the genetic, code in SARS and various SARS viruses, the whole clade of SARS related viruses and SARS-CoV-2 have found that the distinctive difference that allowed this pandemic to occur was related to genetic changes at what they call the furin cleavage site.

In other words, the scent. The increasing sensitivity of this virus to the effects of furin is what created this pandemic. Wow. And if you look at most of the and so they've looked at continuing, mutations in the virus over time since it started.

And the most common place where mutations of this virus are occurring is at the furin cleavage site. So that may or may not be making the the virus more or less contagious or deadly.

But it is it certainly is the genetic change that underlies the Covid 19 pandemic. And it makes sense then, that if you can find a way to block furin, you may be able to block the transmission of the virus.

So I'll put you on the spot a little bit here. Does that seem to be a natural mutation or a manmade mutation? Of course I'm I'm not an I'm not an expert in that.

But the scientists that have looked at this have all of them concluded that it is a series of natural mutations, that there's no lab in the world that would have thought about this, because it's a very awkward and imperfect mutation, and it would have been hard to interpret, because if we go back to the idea of a lock and key, it's not as if the key of the viral spike protein that's, primed by purine is a perfect fit into the lock.

Angiotensin 1-7 as a COVID-19 Therapy

It actually is a very imperfect fit. It gets into the lock, but then it doesn't turn the lock and you can't pull it out. So if you have a key, that's kind of the wrong key for a lock, but it sort of looks like it could be the right key.

You'd really screw up the lock. The lock, you know, you put the key in and now you can't get it out, but you can't turn it. And so these scientists have said, you know, they just don't know of any lab that would have designed this on purpose.

And it really looks like it was a series of, of multiple mutations, and that it's very likely that this virus has been around for a long time. But wasn't really suited and maybe even caused occasional illness in humans, but it wasn't suited for, rapid transmission from person to person.

At the end of the day, I'm not sure that it matters from a treatment perspective whether this is some accident that was created in a laboratory or not.

If anybody decided that they were going to do this to screw up the world, they were pretty stupid because it's not that deadly a virus. And if, and shut down economies and.

Yeah, you know, a lot of the reason that's been done, a lot of the reason that that's happened is because of stupid decisions that have been made and a misunderstanding, I think of the of the nature of the virus and how it works and how it makes people sick.

This did not have to happen. Now I, I agree. Oh, and of course one of the reasons and but it happened in so many places, including places that are not allies politically, that it's very unlikely that this is some, gargantuan conspiracy.

You know, they're just too many ridiculous moving parts to it to have been manmade. You know, my one of my favorite authors, Raymond. So, Raymond Chandler, who created the Philip marlowe character, in all of the books, when Marlowe was going through a detective story, he would say this had the book.

The expression was something like, this had the simplicity of a lie or this did not have the simplicity of a lie, and this does not have the simplicity of something planned.

Yeah, it would have to be a lot of people involved, but, yeah, I don't know. It's the world's crazy now. It's like, I don't know. Well, it's it's it's created chaos.

It's created chaos. And one of the s one of the characteristics of chaos is that the initiating conditions, when chaos is created, have a huge impact on the outcome.

And so we had a small window of to get it right. We didn't get it right. And that is what happens with chaos. We may have another window now. Yeah, I think it's true.

Everyone just panics and so many things are political. And, what's the perception they're doing something or, you know, so it's just it's crazy, you know, it's like.

But, well, we change gears and I know you use, a different peptide. You use a lot of peptides. And I've talked about the ones that that you that you love to use, but I know I've, which is a, tri peptide, meaning just three amino acids of alpha monocytes.

See, when in hormone. If you, want to talk about that a little bit short. Well, I treat, before Covid 19 consumed all of my time and attention outside of treating patients, my research interests were primarily in the GI tract.

The impact of the gut microbiome on health, intestinal permeability, so called leaky gut. And I've been working with those concepts clinically, writing and lecturing about them for over 30 years before there was a term.

Wow. You're 28 years ahead of your time. Well, you know, was it, yeah, quite the, there was a time when I would talk about leaky gut to a patient and they'd go to their gastroenterologist and say, there's no such thing as a leaky gut.

Now, you see the term in the New England Journal of Medicine? Yeah. There's been a lot. You turn around in, in the understanding there. And so I in my practice, I have over the years dealt with many patients with bowel problems and have had a special interest in inflammatory bowel disease, Crohn's disease and ulcerative colitis and have actually written three textbook chapters on nutritional approaches to inflammatory bowel disease.

And so I'm always embarrassed to say I have not read those. I have had to go to find those. So, right. Well, I mean, they all become I mean, first of all, they're not they're not in mainstream textbooks, but they are in, well, they are in mainstream textbooks, just not I don't know if anybody reads them, you know, metabolic medicine and surgery.

Oh, those are the ones that. Yeah, integrative gastroenterology. The only problem is they're like 400 bucks, but, yeah. Right. So, the, so finding innovative approaches to treating inflammatory bowel disease has been one of the things that, has been a, a focus of my practice.

And, and aside from nutrition, their specific supplements, and natural products, specific probiotics, that I use, in my view, on the use of nutrition in medical treatment is that nutritional therapies should be used with the same attention to detail that you would use if you were prescribing a drug, or the doctors should use when prescribing drugs.

That is, there's no one size fits all. This is not like, well, put everybody on a statin to prevent heart disease. And it's not like, oh, here, this is this is the diet.

This is what everybody's diet should be like. This is the the bread. People will ask me what's the best probiotic. There is no such thing. So in the course of looking for individualized treatments, I have gotten very interested in two peptide therapies.

One is cGRP, the, the, the melena fragment, which is anti-inflammatory and which has been tested in a clinical trial in ulcerative colitis. And KPC is something that has become part of my, treatment protocol for patients with inflammatory bowel disease.

I don't start with CPGs. I start with diet and nutrition, and decisions about probiotics and prebiotics. That's stage one. Stage two, if people are not fully controlled is KPC.

I really like it. It's been very helpful and a lot of my patients have benefited from it. And then there's BPC 157 a totally different peptide normally produced in the stomach and under stress conditions.

And that's, that's, I think 17 amino acids.

Peptide Approaches for IBD and Gut Healing

The and BPC 157 has been helpful for inflammatory bowel disease, especially in conjunction with KPC. But I've also used it a lot, for other kinds of bowel problems. And most of the research on it, which has been done in animals, has shown benefits in conditions like short bowel syndrome, because it stimulates healing in, esophagitis and gastritis.

And I treat a lot of patients with, with gastric with Gerd, and esophagitis, it's very common. I mean, there are tens of millions of people in this country who have that.

I really think that the standard medical approach to treating esophagitis is so, physiological. It's amazing to me. I mean, esophagitis is not due to an excess of acid production.

So blocking acid just converts acid reflux to non acid reflux. It may relieve symptoms. It creates a whole host of complications, especially on stream side effects and intense.

Yeah. Like the body pressure compound 157. It tightens the lower, you know, or the, the sphincter makes the more, you know, they get all dysregulated.

Yeah. Right. Yeah. Actually physiologic. Bring them back to normal. Right? I mean, Gerd and esophagitis are basically motility disorders in which there's a deficit in the functioning of the lower esophageal sphincter.

And so treatment should be based upon trying to improve the function of that sphincter, not reducing the amount of acid that the stomach produces. So those are destined to get dysbiosis, which now we're finding huge problem, you know, which is that brain access to everything, gut everything axis.

Right. And there are studies, I mean, over the years, the studies on the impact of of the negative effects of these,

proton pump inhibitors and acid suppressing drugs has accumulated.

Still, the majority of gastroenterologists, will tell patients, oh, you can this is safe. You can take it forever. There no problems. It says short term, 14 days on the on the package.

Right? Right. Even in 14 days, it increases your risk of pneumonia. But, at least the proton pump inhibitors do that long term. The dysbiosis produced by these drugs has been very well documented.

And they are they contribute to C difficile, Clostridium difficile colitis. Now there was one study which looked at the impact of the routine use of acid suppression in hospitalized patients on the likelihood of in-hospital pneumonia and C difficile colitis, and concluded that these drugs in hospitals may be killing as many as 30,000 people in the US every year, just based on the frequency of in-hospital pneumonia in patients getting these drugs and the mortality rate of in-hospital pneumonia.

A one bad place to be is the hospital. And yeah. Yeah. And taking these standard meds, you know, it's and it's not doesn't make any news because it's expected you know right.

Yeah I know yeah. Actually I think hospital medication effects are the third or fourth leading cause of death in the US. Yeah, yeah. Doctors are a big leading cause of death.

Unfortunately, that's the case. And that's preventable. As. But, can you, talk about, so a body protection compound? Yeah. We'd love it for, you know, gut issues also work systemically reduces inflammation.

And, you know, really, with this gut brain axis, probably the best thing for, for leaky gut. We also have the thymus and, beta four, which is a frag which the tight junctions, will help, basically tighten those up.

So I have been really impressed. Yeah. I'm sorry. I've been really impressed with thymus and beta for, and for its, for its neuroprotective effects. Originally I got interested in it as an adjunct to thymus, an alpha one for immune modulation.

But it became, pretty clear that there were other effects. And when I started looking at the literature, the thing I love about the peptides is therapeutic tools is that there is for all of the peptides, there's a pretty extensive scientific literature.

Some of it is from the US. A lot of it is. And so like with BPC 157, the research there has been done mostly in Croatia and China, for example. You know, there'll be countries in which certain researchers will get interested and they'll carry these the torch for these peptides, thymus and beta for what I found to be really important was its benefits in head trauma and head injury.

I mean, so if I have a patient who's just had a head injury, I will get them on thymus and beta four right away. And I'm really seeing an even in people who have neurologic problems that have been longstanding, I'm seeing beneficial effects from thymus and beta for yeah, I, I happen to love that one.

And and I agree, you know, all the issues with traumatic brain injury now and, you know, thymus and beta for massive amounts of studies on it. And the problem is has to be injectable.

But so the what we have out is the A fragment which basically has all the effects of them. It's in beta four, but it takes out the part that cements mass cells.

But also BBC show which radical brain injury. I mean, I you can argue that all these football players, whatever kinship, you know, playing sport should be on it.

Preventively. Yeah. Right. So and BPC has this other really interesting effect. It's a mass cell stabilizer. And I mean, that opens up a whole other area in medicine that interacts with every other, complex, condition that we're looking at is and it's becoming very much recognized now.

And whereas it had been virtually unknown a few years ago, which is mast cell activation syndrome, you could you could of course, devote hours to discussing.

Yes, yes. And it's syndrome and it's huge. And I really think a lot of these things like weren't talked about. I don't think they weren't issues as much 20 years ago, 30 years ago, you know, I think it's I have the theory ends and toxins and stress and.

Yeah. Yeah. And I have a theory about the mast cell activation issue because of some facets research that I discovered that was done at the Karolinska Institute in Sweden about 20 years ago.

I mean, there is no higher prestige place in the world. The Karolinska Institute hands out the Nobel Prize. So this is not, you know, this is not some sleepy university.

They were, they took a bunch of healthy people and they put them in front of the TV monitor for two hours, and they did skin biopsies before and after, and they looked at the mast cells in the skin.

And the exposure to the TV monitor caused deep granulation of the mast cells, which is activation mess, or these hundreds of 200 chemicals in these granules in about 15% of the patients.

So it's possible that one of the reasons we're seeing that high exposure to electromagnetic fields is one of the things that is kind of it's it's rate raising the floor, lowering the ceiling and mass cell activation, making the barrel smaller so that symptoms are more likely to occur.

And I think for these, for the many people that I've seen who are very sensitive to emfs, the mechanism of that sensitivity is mast cell activation. Yeah.

I think it's a common denominator in a lot of the, you know, parts especially, you know, yeah, it's all this time patients and the increasing frequency of parts that we're seeing, I think has to do with.

Yeah. And then also the immunologist whenever they do tilt table they go that's your problem. Like they well what's causing that. It's a symptom, you know. Sure.

That's that's just yeah, that's just, a symptom. It's it's like, thanks for telling me I'm dizzy when I stand up, you know? Right. This is the. This is the mechanism, but it's not the reason.

Yeah. And what do you think's that happened with this 5G? Oh, like, you know, probably. Look, I don't know. I haven't actually looked at the data on that.

The, the only thing that I can say is what is certain is that there's increasing EMF exposure globally everywhere. And that that we are seeing the blossoming of conditions that were not really they could have been recognized 20 or 30 years ago, but weren't.

That is the technology was there to do it. It's not as if, oh, now we can, you know, oh, now we can measure this. No, we can't measure it any better. It's just that, it wasn't happening as much then as it is now.

And I do. These patients are discounted as, oh, it's psychological, you know, and if a doctor doesn't know how to treat it, it doesn't exist. And it's the patient's fault.

It's crazy patient, you know, and it's interesting. Was digging down on us, you know, looking at all these just tiny, you know, like two amino acid peptides by peptides.

Five peptides have like, huge effects and they can't find the receptor. And I found one study looked that they feel it's harmonic or harmonic. They vibrate a certain frequency and activate.

And I'm like think wow. So all these emfs, you know, messing that up, it made me really think, you know, that's real.

That's really interesting. And it it it makes sense because molecules vibrate and there are harmonics created.

And, it would be very surprising if the emfs don't impact on that.

Thymosin Beta-4, Mast Cells, and Clinical Insights

And understanding how that affects the function of cells that can have that can really help to, just to explain some aspects of chronic disease. Yeah, good and tough to study, though.

Yeah. Yeah it is. Yeah. And yeah, just, let's talk just a little bit as we're getting close to finishing up here. When your clinical experience with, you know, BPC, Cfpb, other your other peptides with, you know, something about disease or anything else, just some, what, what you basically thing, a thing, people should know about to for conditions or to stay healthy.

Just with all your extensive knowledge about so many things. Well, first of all, I, as I said before about my attitude towards nutrition, I don't believe there's one size fits all.

I don't believe that everyone should be taking this treatment because this is the way that you're going to stay healthy. I really think the treatment, that prevention and treatment both need to be individualized.

Knowledge and understanding is important. And, the main way that I've been viewing peptides at present is from a treatment perspective, an individualized treatment perspective.

The and as one of the Russian researchers in this area said, this is these are potentially the most effective categories of drugs that there are peptides.

I am not yet what using peptides, for general prevention. That may change. I mean we'll see. Yeah. And it will it will always be individualized will be based on the characteristics of this person.

Yeah. And what's history? I did a genetic test and, my thinking back yesterday were, using that significantly now for, you know, looking at how to treat patients.

And, you know, one showed I have a terrible step for IGF one, you know, that I don't make IGF one. And so it's like kind of we think of, well, okay, let's just kind of replace that.

But you know, so many other things I think is that gets more sophisticated and I think genetics played a big part. I think sometimes we're ahead of ourselves with, you know, some of these 23 to me.

And it's and it's much more complex than one gene causing this and that. Oh so almost. Yeah. Yeah, yeah. I mean polygenic in terms of inherited conditions, it's almost always polygenic genetics.

That's important. But epigenetics is probably even more important. Right. The gene where you could have the genes for diabetes. But if one person eats great exercises they're going to get it.

But another person who could eat normally but they have the same gene, they're going to get diabetes. Yeah. Right. I I'm constantly yeah, I constantly see patients who have had genetic testing and they have, genes that, you know, they'll have a gene for high blood pressure or a cluster of genes for high blood pressure.

The blood pressure is great. Yeah. It's and of course, the other thing is, if we go to the microbiome, it's one of my I mean, I could talk for hours about that, you know, have you back for that?

Because I think that's, that's there about there are about 25,000 human genes in the body. That means that we have fewer genes than rice has a grain of rice has 40,000 functioning genetic units.

We have 25,000. How is that possible? Well, there are about 4 million bacterial genes in our gut microbiome, and

they have a tremendous impact in shaping the way that our own genes are expressed.

And so, I do think that I mean, I think peptides are a hugely important therapeutic tool and will help to shape the future of medicine. Understanding the microbiome is going to be foundational for the future of medicine.

And and what do you think the viral biome. Oh, let's you know right. That's going to be the levels of complexity become really hard for the human mind to comprehend.

For one thing, most of the viruses there are, hundreds, thousands of viruses in your gut. Most of them are what are called bacteriophages. They are eaten by bacteria.

And so they inhibit. They enter the bacteria and they alter the function of the bacteria. So now you've got, you know, 4 million, bacterial genes whose activity is being altered by thousands of viral genes, in expressed in bacteriophages.

And to take it a step further, food that you eat and substances you take may activate or inactivate the activity of these fighters. So for example, stevia, you know, this common sugar substitute that is one of the most potent products at silencing bacteriophages.

Not just specific bacteriophages, multiple bacteriophages. Oregano does the opposite. So how do you even begin to comprehend what stevia or oregano is doing when you put it in your body?

Aside from from from the known effects, there are all these indirect effects, that nobody has ever thought of before the past year or two. And most people, most researchers on the microbiome aren't even thinking about because the level of complexity is way beyond the tools that we have.

And it's interesting. And even like I was reading a study on, bacteria secreting butyrate, which is good, but if it gets a certain level, it stimulates the bacteriophage to kill it so it doesn't get to us.

So it's just like, wow. Like we've evolved with, with these things for, you know, millions of years. But I think that's going to be the next big thing is the virus, the viral biome.

And by the way, we will be, coming out with some bacteriophage, treatments, so you'll be the first person we'll talk to about them and, yeah, just really quick, like VPC, k pv combination.

What what do you think of that for patients? Well, I, I mean, I love it for, inflammatory bowel disease and, and, and ulcer healing as well. I use BPC a lot for upper GI problems, not just lower GI problems.

I haven't looked at kp v in the upper GI tract, but from what I've seen in the physiology of it, it makes sense to combine them in it. Not just to use an inflammatory bowel disease, but in diseases of the stomach and the esophagus.

Yeah. Because I, I think everyone now, I think it's the foods and you know, all the additives and, and these things to have, you know, inflammation of the gut, leaky gut.

It's like you do food sensitivity tests, things like they're not allergic but sensitive to everything, you know, which means their gut is let these big proteins in causing the inflammation.

Now they have all these, you know, you know, symptoms that no one can figure out. And it's everything's a vicious cycle, you know? Yeah. Right. Oh, it's the leaky gut.

Is definitely creates a vicious cycle. In fact, first paper that I wrote on it was called, Leaky Gut Breaking the Vicious Cycle. That was published. I don't know what you're saying is published about 30 years ago.

Oh, my gosh, that that's so impressive. You know, it's like gastroenterologists are just now discovering probiotics,

but, it's amazing. It's just been a pleasure speaking with you.

And, just a wealth of knowledge. And I think everyone's going to find this a fascinating interview. So, I thank you. Yeah, well. Thank you. Thanks a lot for giving me the opportunity to share the work that I'm passionate about.

Now, I am, it's it's incredible. So, keep up the great work. And thank you again. And thanks for being on.