Can Peptides Be Utilized to Combat Viruses? Transcript

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Introduction to ACE2 and the Angiotensin System

What ACE2 does is it creates 180 degree turnaround in hormone balance in this angiotensin system. And it takes an essential but dangerous peptide, which is angiotensin 2, a peptide that raises blood pressure, that interferes with kidney function, that promotes inflammation, that promotes fibrosis and scarring, it takes that promotes blood clotting.

It takes that very dangerous ombre and it flips it over. I mean, it's almost like a superhero movie or something. It flips it over to the opposite. This is Doctor Talks, real talk from real doctors on the issues that matter to you most.

Hello, this is Dr. Kenton Holthorpe with another episode and today I'd love welcoming the esteemed Dr. Leo Gallin. 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.

Dr. Gallin received his international recognition as a pioneer in nutritional medicine and a master clinician. A graduate of Harvard University and also of 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 Sea League magnesium award for his work in nutritional supplements for the American College of Nutrition and at the Albert Norris Marquis Lifetime Achievement Award.

from the marquee of Who's Who for his contribution to healthcare. He has listed annual American's top doctors, then a leading physician in the world. And he has authored seven books and 47 scientific articles, multiple textbook chapters.

So he's done so much. During this 2020, he has been working intensively to develop innovative nutritional strategies, and peptides for coping with the SARS-CoV-2 pandemic and developing an innovative peptide-based therapeutics for the treatment of COVID-19.

So I would like to welcome you, Dr. Gallin, and thank you so much for being part of our summit. Thanks for having me on and giving me the opportunity to share my analyses 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 to start. You want to just maybe talk about the role of the angiotensin ACE2 and angiotensin 1,7 signaling that peptide in COVID-19 and in health?

Sure. I think that's a really important place to start. And just by way of background, the angiotensin system in the body 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 studied for decades.

Dr. Gallinu2019s Background and COVID-19 Research

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 involves an enzyme called ACE2, which is, in essence, its effects are the opposite of ACE, the original

enzyme that was discovered. ACE2 breaks down the most inflammatory, the most constricting of the forms of angiotensin, called angiotensin II, it splits angiotensin II off and creates a peptide called angiotensin 1,7 or Ang 1,7. Ang 1,7 has numerous effects in the body that are very positive and that have been studied.

It is neuroprotective, it improves blood flow, it stimulates tissue repair, it supports the bone marrow, virtually every manifestation of COVID-19 can be traced back to deficiency of angiotensin 1,7. 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.

And I also want to say that The early discussion of ACE2 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 really failed 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 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, F-U-R-I-N, that has to alter the viral spike protein to allow it to attach to ACE2. But even that's not enough.

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, 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 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 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 ANG1-7, which is created when the virus enters cells.

Now, what's important about furin is you can block furin, 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, polyarginines.

How SARS-CoV-2 Enters Cells

Polymers of the amino acid arginine can block the activity of furin. And they've been studied in animal models of infectious diseases like anthrax, for example, where they're very effective as adjuncts to treatment.

What's critically important about TMPRSS2, the third member of this triad, is that not all cells express TMPRSS2, 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 wanna have too much ACE2.

It was totally wrong. You need as much, 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 TMP-RSS2. I think, I don't know if it's seropregnant women or very few pregnant women have been infected.

Also estrogen stimulates ACE2 activity. And so the ACE2 angiotensin molecule basically gets cleaved to this angiotensin 1,7 that has a kind of an opposite effect.

Right, right. What ACE2 does is it creates 180 degree turnaround in hormone balance in this angiotensin system. And it takes an essential but dangerous peptide, which is angiotensin II, a peptide that raises blood pressure, that interferes with kidney function, that promotes inflammation, that promotes fibrosis and scarring, that promotes blood clotting.

It takes that very dangerous array and it flips it over. I mean, it's almost like a superhero movie or something. It flips it over to the opposite, Ang 1-7, which is anti-inflammatory, inhibits blood clots and thrombosis, inhibits scarring and fibrosis, improves blood flow and circulation, improves kidney function.

And that is the fulcrum on which the outcome of SARS-CoV-2 infection swings or rests is ACE2 is so critical. All right, so you're diving deep into peptides and longevity on this podcast and we're right there with you.

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It's time to elevate your journey, starting now. With angiotensin 1-7, been really looking into it and it does so many things, like you're saying, boost mitochondrial function, which associated with almost every disease of aging, chronic neurodegenerative diseases, it's shown to improve memory, it's shown to allow the heart to recover and significantly less damage after a heart attack.

And so, so many benefits to that. Right, right. Just across the board. And angiotensin 1,7 has been studied in animals and humans. It enhances recovery from intense exercise, eccentric exercise.

For women who have had breast or ovarian cancer, who have received chemotherapy or are getting chemotherapy, angiotensin 1-7 supports their bone marrow and prevents the low blood counts that are the result of chemotherapy.

And it works better than the standard drugs in that case. In animal models, it reverses dementia that is associated with poor blood flow to the brain, and just beneficial effect after beneficial effect.

You can use it to heal the lungs, and if it is an angiotensin 1,7 that's used, it's ACE2 itself. In animal models of every type of lung disease, whether it's infectious, including SARS, the original SARS, or whether it's toxic.

Infusions of ACE2 help the lung to heal. Angiotensin 1,7 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 through the lungs is all messed up. It's not regulated properly. Angiotensin 1-7 restores normal blood flow through the lungs and raises.

You get that total mismatch, so blood going through non-perfume. Yeah, blood flows to the areas that are not being ventilated. 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, do doctors not read all these old 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-4 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. Well, some of this is actually pretty new literature and they're not looking at it.

There are some centers that are looking at ACE2 or angiotensin 1,7 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 have applied for INDs for that.

There's a research group at the University of Pennsylvania that's worked with a group at University of South Florida 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 there. 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 at these antiviral drugs.

But it is there and it really it should be utilized now because people are dying now. And ACE2 and angiotensin 1,7 can really mitigate the outcome of COVID-19.

ACE2, Angiotensin 1-7, and COVID-19 Protection

And the angiotensin 1,7 is orally bioavailable, which is not true. Right. There is a form that's orally available, 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 you just use pure angiotensin-17 that hasn't been modified to expand its 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. Maybe once it starts that signaling, it's like a bowling ball that knocks down all the pins.

It only takes a few seconds to get down, to initiate that. And there was a study in mice that were exposed to whole body radiation. And a single dose of angiotensin 1,7 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 move things in the right direction. It's an initiator of healing responses. And I think that's true. We find that doctors don't seem to understand that there's between serum half-life and physiologic half-life.

Right, absolutely. Where like hormones, they basically go in the cell nuclear receptor, they change. protein synthesis, so they're going, but hey, they're gone out of the serum, like T3.

They go, oh, it's such a short half-life. No, it's about two days physiologic half-life, two to three days. So yeah, yeah.

Yeah, that's a really important point.

So yeah, so I've been pushing since March, the beginning of March of 2020, for angiotensin 1,7, as to be available as a therapeutic strategy in COVID-19.

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

So you're doing the edges of 1-7, and then also the fear in which we're going to talk. Right. So that's another concept. that I came up with, and that also will need an IND, which I hope will be in place.

Arginine 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 mucosal cells.

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 in the famous episode of the Saget County Choir in Washington.

where there were 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 aerosolization of this virus. It hangs out in the air. You're 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. 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 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 the way that pneumonia occurs, it's not directly from the virus 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 for clarification, so furin is an enzyme protease, correct?

So it's leaving. Right, furin is a protease. It's a category of enzyme called proprotein convertases. 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 also in certain infections.

Now, 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 is kind of coding and hanging around in the space around the outside of the membrane.

That's the furin that we want to target. That's the furin that has to 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 intracellularly, because whatever you're doing is not getting into the cell where most of the furin is.

It's 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, there's a toxin that it's produced and that toxin gets activated by furin. So with animals who are infected with anthrax, if you give them the polyorgenines, 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, a pretty nasty bacteria, and Pseudomonas produces a toxin.

that requires furin for its activation. And you can treat the eye infection by using eye drops of polyarginine to block furin.

Furin Blockade and Nasal Prevention Strategies

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

Actually, the MERS virus used furin to be to alter itself to bind to its receptor, which is not ACE2. Now, and here, this is real, 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 was probably on an individual basis, it was possibly 10 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 7,000 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 transmitted from person to person.

Now, the biologists who have looked 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 furan cleavage site.

In other words, The sent the increasing sensitivity of this virus to the effects of urine 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 furan cleavage site.

So that may or may not be making the virus more or less contagious or deadly, but 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 man-made mutation? Well, of course, 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 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.

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 can really screw up the lock.

You know, you put the key in and now you can't get it out, but you can turn it. And so these scientists have said, 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 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, but 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 Of the nature of the virus and how it works and how it makes people sick.

This did not have to happen. No, I agree. And of course, one of the reasons, 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.

There are just too many ridiculous moving parts to it to have been manmade. You know, one of my favorite authors, Raymond Chandler, who created the Philip Marlowe character.

In all of the books, when Marlowe was going through a detective story, he would say, 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. The world's crazy now. I don't know. Well, it's created chaos. It's created chaos.

And 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 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 when we change gears and I know you use a different peptide you use a lot of peptides and we'll talk about the ones that that you that you love to use, but I know KPV, which is a tripeptide, meaning just three amino acids of alpha monocyte simulant hormone.

If you want to talk about that a little bit. Sure. 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, it was, it, yeah. There was a time when I would talk about leaky gut to a patient and they'd go to their gastroenterologist, you'd 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 huge turnaround in the understanding there. And so 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 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.

Peptides for Gut Health and Inflammatory Bowel Disease

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

And aside from nutrition, there are specific supplements and natural products, specific probiotics that I use. And 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 the diet.

This is what everybody's diet should be like. 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 KPV. the melanocortin fragment, which is anti-inflammatory and which has been tested in a clinical trial in ulcerative colitis. And KPV is something that has become part of my treatment protocol for patients with inflammatory bowel disease.

I don't start with KPV, 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 KPV.

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, I think, 17 amino acids. And BPC 157 has been helpful for inflammatory bowel disease. especially in conjunction with KPV, 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 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 unphysiological. 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 with side effects unintended.

Yeah, like the body pressure compound 157, it tightens the lower, the sphincters makes them more, they get all dysregulated. Yeah, right. You actually physiologically 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.

And so I, those are dysbiosis, which now we're finding huge problem, you know, which got brain access to everything, got everything access. Right. And there are studies, I mean, over the years, the studies on the impact of the negative effects of these proton pump inhibitors and acid suppressing drugs has accumulated.

Still, the majority of gastroenterologists We'll tell patients, oh, this is safe. You can take it forever. There are no

problems. It says short term, 14 days on the package.

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

And they contribute to C. difficile, clasperidium 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.

Hey, one bad place to be is the hospital and taking these standard meds. And it 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, doctors are a big leading cause of death.

Unfortunately, that's the case. And that's preventable. Yes, yes. 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 access, probably the best thing for leaky gut. We also have the thymus in beta four, which has 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 in beta four and for its neuroprotective effects. Originally, I got interested in it as an adjunct to thymus in 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 isn't. 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 the torch for these peptides.

Thymus and beta-4, 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-4 right away.

And I'm really seeing, and even in people who have neurologic problems that have been longstanding, I'm seeing beneficial effects from thymus and beta-4.

Yeah, I happen to love that one. And I agree, you know, all the issues with traumatic brain injury now and, you know, thymus and beta-4, massive amounts of studies on it.

And the problem is it has to be injectable. But so what we have out is the fragment, which basically has all the effects of thymus and beta-4, but it takes out the part that simulates mast cells.

But also BPC showing with traumatic brain injury, I mean, you can argue that all these football players, whatever, kids should, you know, playing sports should be on it preventively.

Right, right. So, and BPC has this other really interesting effect. It's a mast 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, of course, devote hours to discussing mast cell activation 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?

Thymosin Beta-4, Mast Cells, and Neurologic Injury

I think it's… I have the theory… And toxins and stress and yeah. Yeah. I have a theory about the mast cell activation issue because of some fascinating 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 a 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 degranulation of the mast cells, which is activation, mass for 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 the high exposure to electromagnetic fields is one of the things that is kind of it's raising the floor, lowering the ceiling on 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 things, you know, POTS especially, you know.

Yeah, it's patients. And the increasing frequency of POTS that we're seeing I think has to do with. Yeah. And then they go to the neurologist, whatever, they do a tilt table.

They go, that's your problem. Like they don't, 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 like, thanks for showing 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 should happen with this 5G?

Oh, like, you know, Probably, look, I don't know. I haven't actually looked at the data on that. The only thing that I can say is what is certain is that there's increasing EMF exposure globally, everywhere, and that we are seeing the blossoming of conditions that were not really, that 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 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. These patients are discounted as, oh, it's psychological. And if the doctor doesn't know how to treat it, it doesn't exist.

And it's the patient's fault. It's a crazy patient. And it's interesting when I was digging down on, I was looking at all these just tiny, like two amino acid peptides, bipeptides, tripeptides.

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

And I'm like, wow, also all these EMFs, you know, messing that up. It made me really think, you know. That's really interesting. And it makes sense because molecules vibrate and there are harmonics created.

And it would be very surprising if EMFs don't impact on that. And understanding how that affects the function of cells, that can have that can really help to explain some aspects of chronic disease going forward.

And tough to study, though. Yeah. Yeah, it is. Yeah. And just a little bit, as we're getting close to finishing up here, putting your clinical experience with BPC, KPV, your other peptides with comfrey bowel disease or anything else.

just what you basically think people should know about for particular conditions or to stay healthy, just with all your extensive knowledge about so many things?

Well, first of all, 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 gonna stay healthy.

I really think 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.

And as one of the Russian researchers in this area said, these are potentially the most effective categories of drugs that there are, peptides. I am not yet using peptides for general prevention.

That may change. I mean, we'll see. And it will always be individualized. It will be based on the characteristics of this person. Yeah. And what's interesting, I did a genetic test, and when I came back yesterday, we're using that significantly now for looking at how to treat patients.

And one showed I have a terrible snip for IGF-1, that I don't make IGF-1. And so it's like, kind of rethink of, well, okay, let's just kind of replace that.

But, you know, so many other things. I think as that gets more sophisticated, and I think genetics played a big part, I think sometimes we're ahead of ourselves with, you know, somebody's 23 and me, and it's much more complex than one gene causing this and that.

Oh, sure. 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. You can have the genes for diabetes, but if one person eats great exercises, they aren't going to get it. But another person who can eat normally, but they have the same gene, they're going to get diabetes.

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.

Their 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 don't want me to have you back for that because I think that's huge. There are about 25,000 human genes in the body.

Microbiome Complexity and Personalized Peptide Therapy

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 gonna be foundational for the future of medicine. And what do you think the viral biome? Oh, listen, you know, right, that's gonna 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 enter the bacteria.

and they alter the function of the bacteria. So now you've got four million bacterial genes whose activity is being altered by thousands of viral genes 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 phages. 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 the known effects, there are all these indirect effects that nobody has ever thought of before the past year or two. And 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, I was reading a study on bacteria screening butyrate, which is good. But if it gets a certain level, it stimulates the bacteriophage to kill it.

So it doesn't get too high. So it's just like, wow, we've evolved with these things for millions of years. But I think that's going to be the next big thing is the viral biome.

And by the way, we will be coming out with some bacteriophage treatments. So you'll be the first person I talk to about them. And yeah, just really quick, like BPC-KPV combination.

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

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

Yeah, because I think everyone now, I think it's the foods and, you know, all the additives 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 letting 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, and 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. It was published I don't know, what year is this? It was published about 30 years ago.

Oh my gosh, 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 gonna find this a fascinating interview. So I thank you. Well, thank you. Thanks a lot for giving me the opportunity to share the work that I'm passionate about.

No, it's incredible. So keep up the great work. And thanks for being on. All right. Thank you for tuning into Dr. Talks. We hope today's episode has enlightened and inspired you on your path to optimal health.

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