Michael Roesslein:

Recording. We are recording. So welcome everyone. This is part two for the Rebel Health Tribe Gut Microbiome and Immune Webinar conglomerate masterpiece. My guest is Kiran Krishnan. Kiran, welcome back.

Kiran Krishnan:

Thank you. Thank you for having me back.

Michael Roesslein:

[crosstalk] turned off the last one. The reason we're here again, is because your presentation was so packed and loaded, and huge, and awesome that we didn't have time to do the questions. And we wanted to do the questions. So if anybody's watching this-

Kiran Krishnan:

[crosstalk] this isn't the first time that's happened with me.

Michael Roesslein:

Yeah. The last time we went into a part three, right? That's why I have the questions all written up in rapid fire succession. I told Mira what we were doing, and she goes, "Last time, didn't that go to a part three?" And I said, "Yes, but we're going to get it in part two this time." So if you missed part one, go check it out. It's on our blog.

It's an amazing presentation that it's really, really, really comprehensive. I've read and watched a lot of stuff on gut immune and microbiome interrelation in the last five years, and that was definitely the most complete picture of it I've seen. And I still need to watch it at least twice more, because I am not a microbiologist. I have to say I got the most emails about it, out of anything we've ever done.

Kiran Krishnan:

Wow. Awesome. Good.

Michael Roesslein:

From doctors, from practitioners, from scientists, there was a microbiologist that messaged me and said, "Her heart felt warm seeing this information go out to the public."

Kiran Krishnan:

I love it.

Michael Roesslein:

So science nerds are uniting, and I'm glad to be stuck in the middle of it. So let's do it. I have not ordered these in any specific way, so they will be completely random. I feel like this is some sort of microbiome game show we're inventing. There should be some sort of ticker, or points, or score, somewhere on the screen, but we'll do what we can to get through. Oh, somebody said they understood it on their first time through, and they are better at this than I am.

Kiran Krishnan:

Nice.

Michael Roesslein:

So let's just kick it off. I will start question one. Do you have any knowledge or experience around the Ayurvedic diet, or something Ayurvedic kitchari, do you know that word? Kitchari? Kitchari. Okay. Just around Ayurvedic diets specific to individuals, and how Ayurvedic diets would relate to microbiome, or gut health at all. Do you have any knowledge there?

Kiran Krishnan:

No, very little. I mean, I know what I know about the Ayurvedic diets is they are designed to work well with your composition, whether you are like a Vata or depending on your tendencies, as an individual. And a really good Ayurvedic practitioner can basically look at you and assess which category you fall into.

So I've not seen any evidence of how that relates to the microbiome. But, what I do know is within Ayurvedic medicine, the gut is a big central focus as well. So I'm guessing that a lot of the interpretations of how people's compositions are, will circulate around the core, also. So if Ayurvedic is working for you, and a particular prescribed diet is working, it may be a better fit for your particular microbiome.

Michael Roesslein:

Got you. That's about my depth of it, too. And I know they do use a lot of really powerful herbs and spices. I used to have a book that was super cool, and I don't remember what it was called, but it had recipes and herbs and spices in it. And it would break down like clinical research behind herbs and spices, and what they do in the body, and then recipes to use them. And it was pretty impressive, the line of study from India, specifically, around herbs and spices and clinical research, which we don't really do here.

Kiran Krishnan:

Yep. Yeah. There are institutes, Ayurvedic institutes, that are equivalent to the Harvard's here, the Ivy league schools here, and they're quite amazing. I wish I had the chance to study Ayurvedic a little bit more, but I haven't.

Michael Roesslein:

No worries. I am writing something down from one of our people here saying to look at Banyan Botanicals for kitchari. So I can learn what that means. Great, spores or any other products in your line have any impact, or relation to retroviruses?

Kiran Krishnan:

We haven't studied retroviruses. Retroviruses are quite unique in their method of replication. There's no specific data on spores being able to combat retroviruses that I've ever seen. But again, with any other virus, really the first initial key to stifling the viral replication; whether it's a retrovirus, a DNA virus, an RNA virus, it doesn't really matter. But it's interferon that's really the key, right? Interferon is the first line of defense against viral infections.

The cells themselves that get infected can release interferon signals, and then the microbiome triggers interferon release by adjacent cells. And then of course the recruitment of innate immune actors also causes a release of interferon's. So no matter what kind of virus it is, that first line of defense is going to

be the interferon release. And that is equally successful against any kind of virus, no matter what type it is.

The most commonly known retrovirus of course is HIV, human immunodeficiency virus. The reason why that virus become so deadly over time is because of its target. It's target is the CD4T-cell. The CD4 is a really important helper T-cell that helps facilitate the immune response towards a more powerful, adaptive immune response, triggering B-cells and so on, because those cells start to diminish. And that part of the immune system starts to get really deficient.

You started becoming much more susceptible to all kinds of common illnesses. CD8 T-cells tend to go up, CD4 T-cells tend to go down. That's why that virus in particular is difficult because it attacks the immune system itself. So just being a retrovirus in itself, doesn't make it any more special than any other virus. The one that people know about is HIV and that targets immune cells. Interferon is the key. The interferon will negate the replication of almost any virus.

Michael Roesslein:

There we go. That's question number six, where do type one and type two interferon's fit into the overall picture you presented in part one?

Kiran Krishnan:

Yeah, that's the first line of defense. Type one, type three, type two, all of them get released by the infected cell itself. The R-cells have a defense mechanism, if you will, and a self destruct [crosstalk]-

Michael Roesslein:

Kind of like a panic button.

Kiran Krishnan:

Totally. Panic button. Yeah. The moment they get infected, one of the first things, genes that we start turning on, our cells start turning on, is the interferon genes. Then the second genes they start to turn on are the chemokine genes. The chemokine genes are the flares that help the immune system understand that there's a problem going on here. Now, here's what's crazy about it. A study just came out about a few days ago, maybe three days ago, on the mechanism of action on COVID.

COVID, what makes it really interesting is as it enters into the cell, one of the first things it codes for is a blocker for that cells interferon production, right? And that's not unique because a lot of other viruses do that. Influenza does that, respiratory syncytial virus does that, lots of other viruses do that. That's kind of how viruses fight against our immune system, or our detection system to combat it.

And then the second mechanism, the release of the chemokines to attract the immune system, most other viruses also inhibit that part, right? So most of the virus are trying to quiet both those signals in the cells of the infect. So that your body and your immune system doesn't get alerted to the presence of the infection. The COVID does something completely different. It stops the interferon production, because that is the key thing that stops viral replication in its tracks, but it actually amplifies the inflammatory signal.

It amplifies a chemokine signal, which is really interesting. So it does it in a different way, and that's not how the original SARS caused the infection as well. That's why people tend to be very susceptible to this, what we call cytokine storm. Because the virus in itself is amplifying these inflammatory chemokines to attract the immune system to the site.

Now, and I don't know this for sure because this study just came out, but my hypothesis then is the microbiome will play even a more important role in this kind of mechanism. Because, one of the things that the microbiome helps to do, is trigger the release of more interferon. That's one of its jobs, initially, and it does that in influenza, it does that in other types of respiratory virus. It may do that in this case, we don't know that for sure, a study hasn't been done. But that's where this becomes really interesting, is that the microbiome may play a role to try to undo some of that imbalance that the virus causes.

Michael Roesslein:

It's like a battle that we don't have even a player in.

Kiran Krishnan:

Yeah, exactly.

Michael Roesslein:

[crosstalk] it's the other [inaudible] versus the virus trying to control the response of our cells. Like our cells are just the inner dumb objects, in a sense, versus the-

Kiran Krishnan:

Yeah. They're just a victim sitting there going, "Oh, crap."

Michael Roesslein:

[crosstalk] Which way do I do? Do I do this?

Kiran Krishnan:

Right.

Michael Roesslein:

Did you see a doctor Kharrazian and Aristo Vojdani study that came out this week? About the autoimmune markers in the COVID-19, cross [crosstalk]-

Kiran Krishnan:

I did not. No. That's actually-

Michael Roesslein:

It's pretty, they did some cross-reactivity in their lab with antibodies, ANA, DSDNA, and another antibody, and the COVID-19 antibodies that have been pulled from people, and saw that there was cross-reactivity. Which might explain some of the autoimmune-like responses that some people have. I didn't fully understand the paper, I'm sure you would. But just in case, I'm going to bring Dr. Kay on to talk about it. So I'll make sure to send you the video. I just want to put him in front of the camera and be like, "Can you explain what your study means?"

Kiran Krishnan:

Yeah.

Michael Roesslein:

Because he's brilliant. They are too, Vojdani too, but I just don't always follow exactly. So I'm going to bring him on, we'll talk about it. I'll make sure you get a link, but it seems like-

Kiran Krishnan:

That'll be fascinating to see.

Michael Roesslein:

They seem to have connected some dots.

Kiran Krishnan:

Yeah.

Michael Roesslein:

Anything specifically different for kids regarding microbiome health, and gut health, that would be different than adults?

Kiran Krishnan:

In general, no. I mean, once a kid is above the age of around two, they have basically their adult microbiome. And the adult microbiome at that stage kind of functions in terms of its relationship to the immune system in a very similar way that adults do. Our goal really is to have the right balance of bacteria, and not have too many toxin producing members of the microbiome, keeping their relative abundance lower to the rest of the commensal species. Like one of the things that we test in that biome effects test, is your pathol biome.

Pathol biome is really interesting because it's not unusual at all. In fact, it's perfectly normal for people to have pathogens in your microbiome, or potential pathogens. So opportunistic pathogens, that's a normal part of the ecosystem. And in fact, many of those pathogens play important roles. The question is, what is the relative abundance of that path of biome to the rest of the commensal species?

That's when you get into trouble, if the relative abundance is too high compared to the rest of the commensal species, then you're going to end up having a net of problems. You're going to have too many toxins being produced here, too much inflammatory induction, maybe a net digestion of the mucosal layer, those issues start to creep up.

Whereas if you have the pathobiome within control, then the rest of the commensals, the mutualistic, all of those will keep those microbes under control, and under a certain distinct level where they're not causing problems. That's a big difference between the presence of pathogens, versus the absence of pathogens. You really can't have an absence of pathogens at all within the microbiome.

Michael Roesslein:

That makes sense.

Kiran Krishnan:

Oh, and then back to the kids thing. That's the same thing with kids at that stage in life. Now in the first year, the microbiomes going through all kinds of complications. In the first six months or so before you introduce solid foods, the microbiome tends to be very bifido genic. It is really heavy in the bifido side.

Lots of facultative bacteria are in the small intestine, and proximal part of the large intestine, eating away the oxygen, so that eventually the most of the gut becomes anaerobic.

Once you start introducing solid foods, you start seeing a diversification of the child's microbiome. After the first year, you start to see more diversification and a striation of the different types of microbes in the different sections of the gut. So things are kind of tenuous and fluctuating quite a bit in that first year or so.

And then between one and two years, the child is starting to establish their adult microbiome. So I would say in the beginning, in the first six months, the big focus is to continue to improve bifido bacteria levels in the infant. Most of that comes from oligosaccharides in the breast milk, and mothers milk. But you can also utilize some oligosaccharides, or some well-researched infanticide strains to help if the baby doesn't have adequate bifidobacteria.

But from six months to about a year, year and a half, the most important thing is diet. The most important thing is diversification of the diet, eating lots of roots, tubers resistant starches in, some polyphenols, carotenoids, all of the colored, fruits and vegetables, all of those things play an important role in starting to the diet. Now, from the age of about two or so, all the way until around 10 that's when you're really building your oral tolerance.

That's a really important time to build out your immune system's tolerance of all of the things that you're going to get exposed to, that you shouldn't be attacking. And at that stage, the microbiome was really important in order to present antigens in the right way to the immune system, in order to dampen immune responses that are not necessary, by up regulating something called a Treg system. So that's when kids started developing lots of allergies, if their microbiomes are dysfunctional. So that's why studies show that in the first four or five years of life, if they've had multiple rounds of antibiotics, then their risk for developing allergies and other immune dysfunction's kind of go way up, right? So that's a really important time to maintaining diversity and a growing a healthy microbiome.

Michael Roesslein:

Interesting. Because I noticed that the kids, when I was growing up that had the strictest diets, like where they weren't allowed to eat anything, or touch anything, or do anything, they had the most issues by the time we were in high school with foods.

Kiran Krishnan:

Totally. Yeah, absolutely. And that's because they didn't get the exposure. And without the exposure, you don't build a tolerance. Or if you have the exposure and you have a damaged microbiome, let's say your child has had multiple ear infections and all that. And so they've gone through multiple rounds of antibiotics in the first few years of life. Then their risk for having that dysbiosis, which means a negation of the tolerance generation and the immune system goes up quite a bit. If you're a 45 year old adult, or if you are a two and a half, three year old kid, the object should be the same.

It is about increasing the diversity of the microbiome, dampening the presence of those pathogens and getting adequate amounts of Butyrate, short chain fatty acid production. Because, all of those things actually dramatically improve the function of the microbiome. Another thing about the interferon, one of the things that triggers interferon expression adequately is acetate from short chain fatty acids. Butyrate provides energy for dendritic cells, and macrophages and all that, to go out and do the job that they do surveying the system, and going after things that are invading. So all of those same rules still apply, whether you're two and a half years old, or if you're 45 years old.

Michael Roesslein:

Makes sense. One other kid question, how does it help baby if mom can't breastfeed? Just as much diversity of what you can feed them, getting them outside, getting them in contact with this, get them a dog.

Kiran Krishnan:

Yeah. Get them a dog. Absolutely. And doing as much skin to skin contact, if you can, this is assuming mom is healthy. But one of the primitive things that we've been doing as a species is often moms will chew the food and then give it to the baby. There is a study on pacifier cleaning, for example. The studies compared two different groups of moms, ones who, if the baby drops a pacifier on the floor, they will clean it with a sterile wipe and then give it back to the baby. Versus the moms that clean it in their own mouth and give it to the baby. The study showed that the moms had cleaned up with their own mouth and give it to the baby, had babies with lower incidents rate of allergies. Right?

So that seems to make a difference. So any contact, physical contact with your baby will help diversify the baby's microbiome, if you're not able to breastfeed. Now, once you start introducing solid foods, it becomes extremely important to provide the baby foods that are high in oligosaccharides, right? Fructooligosaccharides and resistant starches because those are the types of compounds that the really important colonic bacteria need that they will typically get from the oligosaccharides from mother's milk. Mother's milk contains over 200 different oligosaccharides as prebiotics for the baby. So if you can't get that from others milk, you've got to try to get those prebiotic oligosaccharides from other sources.

Michael Roesslein:

Right. That makes sense. Where did that one go? All right I deleted that. Different topic. Does taking supplemental D3 thin the blood? I'm on cancer treatments and told not to take aspirin, but I want to take D for immune support. I don't get in the sun a lot, would D be harmful to me? Now, we're not doctors, we can't talk to you about your cancer treatment specifically, but can you talk to that just a second?

Kiran Krishnan:

Yeah. So thinning of the blood is really an effect of the thrombin pathway, right? So that's the coagulation pathway; the thrombin pathway, all the thromboxane and that whole complex pathway. As far as I know, vitamin D doesn't play any role in that pathway at all. So it shouldn't have any of that effect at all. Talk to your doctor about it, of course. But vitamin D shouldn't play any effect in that role, in that pathway.

Michael Roesslein:

How does the body compensate for removed thymus gland? Is it possible to have optimal, or even decent, immune function without it?

Kiran Krishnan:

I think it is. So the thymus is an interesting place the lymphocytes are made in the bone marrow and they go to the thymus for maturation. Now, assuming you can't go to the thymus for maturation. I've seen some data that the maturation can occur in lymph nodes, as well. And especially with the gut associated lymphoid tissue, areas like the Peyer's patches, which are a really potent site of a T-cell and B-cell proliferation. So I think you probably could. It's not that if you had no thymus, you wouldn't have

immune function. I think there's probably going to be some degree of compromise to the immune function. But I think certainly your immune system would provide a certain degree of protection.

The gut associated lymphoid tissue is still the largest component of your immune system, and it still plays a significant role in the maturation, and the tutoring, if you will, of your lymphocytes. And again, the lymphocytes are still being made in the bone marrows, that's still good. So you're still making lymphocyte. Then the hope is that you'll get adequate maturation and tutoring, or training, of the lymphocytes then in the Peyer's patches or in the mesenteric lymph nodes. My guess is that there would be some compromise to the immune response, but it wouldn't be like your immune system not functioning.

Michael Roesslein:

Got you. The body has a lot of amazing redirects for when it loses capacity in some regards. There's almost, other than the heart, the lungs, the brain, like the major organs, and even there's like workarounds. Like when we lose a tissue or a certain piece of equipment, the body seems to be able to... Like, they're just learning that like for a while, they didn't even know that fat adipose tissue produced hormones, or other tissue. And how everything was sterile, and now none of its sterile.

And we're just learning that there's a lot of backup plans involved in the body, which is how it can survive when we treat it like hell. How do might taking MegaSpore impacts a parasite overgrowth or would I want heavier artillery?

Kiran Krishnan:

If you have a true parasitic infestation infection, then you should look at anti-parasites, anti-parasitics. You should get a parasite culture done though. The stool tests that you have available to you in the market, aren't adequate for that. These would be highly specialized parasitology tests. They've been doing them for decades now. They're very common, easy to do tests. They do need stool samples and a good amount of stool samples, actually. You'll have to poop a good amount into a big tray and then scoop it into four or five different little jars-

Michael Roesslein:

Is it the parasitology center? Do you know that place? Is that who you're talking about?

Kiran Krishnan:

Oh, any doctor can order it.

Michael Roesslein:

Oh, okay.

Kiran Krishnan:

Yeah. [crosstalk].

Michael Roesslein:

It's a different thing. It's not your biometrics. It's not a GI map. None of these doctor's data stool tests that market parasite, like there's a lot of false negative, false positive, false everything.

Kiran Krishnan:

Yeah. Because all of those are looking genetics, the parasite genes. These are actual culture tests, where they're culturing up the parasites and trying to grow them, and identify them more accurately. They'll look at them under a scope, and all of that stuff. So you need larger samples of stool to do that. If you do suspect a parasite is an issue, I would go that route. Because you need to know, and you need to have that more definitively known.

You can go to almost any urgent care center and walk in and they can order a parasitology tests. It's a simple, straightforward test, so your insurance will cover if you have insurance. Now, let's say that the test comes back that your parasitology test is really negative. You still suspect that something going on then you just do all the things that you normally do to balance out the microbiome.

The spores can certainly help if there are egregious bacteria in there that need to be brought under control. That's part of the function of the spores, and also improving the growth and the presence of a lot of the other commensals, that help compete against problematic fungi, and bacteria, and so on. So you can work to bring about balance. If you have a really low level of parasites, and you're not comfortable with doing an antiparasitic, you might try the probiotic.

We don't have data showing that the spores will competitively exclude parasites, so it's hard to say. We know people that have used it for what they suspect to be parasitic issues. But again, they didn't confirm the presence of the parasites using the parasitology test.

Michael Roesslein:

I had another spores [inaudible] on that. Do probiotic supplements in foods actually survive the stomach or stomach acid? I've read that they don't. Why are the spores any different?

Kiran Krishnan:

Yeah. They don't and bacteria in food, fermented like food, are going to die in the stomach. And you're going to get some component of that bacteria moving through the system, which is fine. If it's a true fermented food, then there's a lot of other benefits to the food outside of the bacteria being present in your gut. Right? The benefit of fermented food, keep in mind, is the ferment itself. All of the amazing things that were produced during the fermentation process; the organic acids, and the peptides, and the vitamins, and all that.

So that's where the true benefit of fermented foods come from. Spores are different because they have that spore capsule. They have this harden, calcified protein shell that they wrap around themselves, and acts like a biological armor, if you will. So they can survive through the gastric system, low pH is fine. They're tolerant in the bile salts. And then when they get to the end of the duodenum, they start to come out of this shell and then they start to work for you in your gut, both in your small and large intestines.

Michael Roesslein:

You guys talked a lot about bacteria and other organisms in the microbiome, communicating directly with ourselves, how do they do that? And then in parentheses, this may be a silly question, but I don't understand how they talk.

Kiran Krishnan:

Yeah. And so it's not a silly question. And in fact, we know a good amount about that, but there's also still a lot that we don't understand. What's interesting is there's a couple of different ways. One is bacteria themselves can produce compounds like chemical signals, that your immune system can read,

right? So bacteria have complex genomics. They can start producing chemical proteins and signals that can attract your immune system, or alert your immune system.

The second way is they can stimulate the cells around them, which are your cells, just to release those same inflammatory signals. So they do it through chemical messengers. So things called cytokines, or chemokines. So cytokines and chemokine are chemical messengers that alert to things, or create a cascade of other signals, or act as an attractant to the immune system to that area. So a chemokine is actually defined as a chemical attractant to that area.

So they do it in those couple of different ways. They themselves can produce signals, and in most cases they will trigger adjacent cells, which are your cells, to release signals as well. All of our cells, for the most part, with the exception of red blood cells and a couple other cells, all of our cells have the capability of producing these types of chemical signatures. That's how cells defend themselves when they come into contact with pathogens, like viruses or bacteria.

And so if the microbiome sitting here, if I'm a bacteria in the microbiome, and I notice adjacent to me, the cell, which is the human cell, the host cell, is getting infected. And I sense this chemical signature of this infection, then I can get the human cell that I'm sitting closest to, to release some of these chemical signatures as well. So that it attracts the immune system to that area.

Because remember, the microbiome is the neighborhood watch for your immune system, right? It is virtually impossible for your immune system to survey the whole landscape of microbes in your mucosa and go through every second of every day and pick out areas where there may be infections. Because remember I mentioned, you've got roughly 40 trillion microbial cells all over your mucosal tissue, and you've got about 200 million immune cells that are surveying all this area.

So you've got 200,000 times more microbial cells and components in your system, than you have immune cells. So that differential makes it that the immune system cannot, in any possible way, survey the entire field effectively every second of every day. So it needs that neighborhood watch, it needs the microbiome to alert it when it notices things going wrong around it. Right? And it does it through these chemical signatures, either releasing itself or triggering the cells near it, to release it, to alert the immune system.

Michael Roesslein:

So some direct way, some indirect ways, some nudging and almost bating, pulling, kind of ways, and then some completely unknown magic.

Kiran Krishnan:

Yeah, exactly. And there's a bunch of unknown. Because number one, we know, for example, when you look at the gut lung access, there is clear data that when the lungs get infected with a virus or bacteria, that the microbes in the lungs, will signal to the microbes in the gut. And then the microbes in the gut, will trigger the gut associated lymphoid tissue to respond to the lungs. How do the microbes in the lungs communicate with the microbes in the gut? That's still kind of unknown.We're not sure what the signals are.

Michael Roesslein:

I bet its [inaudible].

Kiran Krishnan:

And it could be.

... unknown. We're not sure what the signals are.

Michael Roesslein:

I read it's quantum.

Kiran Krishnan:

And it could be. That's the beauty of this, right? So they all live in what we call the singular mucosal theory system. So the mucosa, which is this 400 square meters of real estate inside your body... And again, for Americans, that's what, 4,000 square-

PART 1 OF 4 ENDS [00:31:04]

Michael Roesslein:

Yeah. It was 4,000 plus square feet.

Kiran Krishnan:

Yeah. So it's a huge piece of surface area. They all live in that same lattice. And one of the things that's true about microbes all over the bodies, they produce and they live within biofilms. The communication that occurs in biofilms is really interesting. Think of the space time continuum, the fabric of space and time. All of them are connected together through these biofilms and through the mucosa. They can communicate instantaneously from different parts of the body. How they do that exactly, still unknown in large part.

That's one of the reasons why we made the big donation to Sacramento State, because that's one of the things in that microbiome research division that they have an interest in studying. And we have a very strong interest in understanding, how do bacterial communities communicate with one another? Because the beauty of it is, if we can understand their language, maybe we can listen in on their conversations. Maybe we can influence their language in some way. Or maybe we just learn something about how the natural world functions, which is a whole level that's more complex and fascinating than we could ever imagine right now. So there's unknown magic communication as well. There's a couple ways that we know about, but there's likely some magic. Microbiome magic.

Michael Roesslein:

I read a book called The Field this year, and it talks about the quantum field, which we don't really understand. It was the biggest book for dummies of quantum physics that exists. If you can take quantum physics and dumb it down to the dumbest, dumbest, dumbest, dumbest point, I could almost understand a little of it. And it talked about biochemical processes and reactions in the body that happen at speeds that we can't explain. Like if something communicates with something else faster than a chemical messenger could possibly be released from that part of the body, get into the blood, and then circulate to get to the other point, there are reactions in the body, especially within the nervous system, that happen much faster than that. And that they think that it has to be photons, like light. It's in a quantum matrix. And that the microbes likely communicate in a similar way. They were talking about it in the book. That's as much as I understood out of about 100 pages of that.

Kiran Krishnan:

Well, that's a very significant part of quantum mechanics itself. There's the mechanics of couple particles that could be on either end of the universe, and when one changes, the other instantly changes as well, and that's much faster than the speed of light. So there's this quantum coupling among particles. So microbes, which are replications of one another, in many ways almost exact replications, there may be some quantum coupling. You don't know where. There's a whole level of science that-

Michael Roesslein:

Interesting. So it'd be an interesting few decades of science, I think, as quantum science starts to merge with microbiome science. Nature versus nurture argument seems to be similar to germ versus terrain argument. Where do you stand on germ versus terrain? Personally, I would answer that I'm somewhere in the middle, that I think both are relevant. But I think the prevailing theory used to be fully germ. Well, originally it was fully terrain, then it was fully germ. And I think we've looped back to some middle ground.

Kiran Krishnan:

Yeah. And when you really look at the data, it can't be either one by itself. It has to be both. Number one, the germ has to be present in order for it to cause an illness. There is a villain here, if you will. Let's take an infection of some sort. Let's take an E. coli foodborne illness infection. You could have 10 people eat the same thing that is contaminated with this E. Coli, but only three out of 10 will get an illness out of it. So they all have the germ, but the difference in whether or not the germ has the capability to elicit an infection is that person's terrain. What does that person's microbiome and the immune system look like? Does it allow the germ to function the way it's trying to function?

Now, here's an interesting thing that we do know about lots of pathogens, lots of germs, is they won't necessarily trigger their virulence factors or their toxins unless they can reach a certain threshold amount. A great example of that is listeria monocytogenes. Listeria is one of the most common foodborne illness pathogens. And listeria, we know once it gets into the body will not trigger any of its virulence factors or toxin production until it can reach a certain threshold concentration within the body, within the system, within the gut, in this case. And if it doesn't reach that threshold level, it's not going to turn on the virulence factors at all. Because it knows that under a certain threshold, if it starts turning on its virulence factors, there's not enough listeria there to cause a profound infection. It's going to get quenched right away by the immune system and other microbes in that area.

So it knows that. It uses the quorum sensing to talk to the other listeria that it's multiplying by to say, hey, have we reached that critical mass? Can we turn on the genes? And if the terrain, meaning you've got lots of strong competitive bacteria, you've got really strong ecosystem in the gut, you've got a well functioning immune system, you're producing lots of short chain fatty acids to fuel your immune cells, you've got a strong barrier function, meaning you've got a strong mucosal layer and you've got a tight intestinal epithelium. All of those things are working well. Listeria may never get a chance to reach that threshold level. So it may never elicit its response or its virulence factors.

So it's both. So the germ is present. The germ has certain capabilities, certain wants, certain goals. And the terrain is going to either allow the germ to do that or not allow it to do that. And you can't have an infection without either of them. You can't have an infection without the germ and you cannot have infection without the terrain allowing the germ to do what it does.

Michael Roesslein:

Suggestions for increasing low sIgA. And sIgA is an immune cell. If you could just give a quick on sIgA. And then I know of Saccharomyces boulardii, I've seen... Of course, here comes a lawnmower. I'll deal with that. Suggestions for increasing low sIgA. I know Saccharomyces boulardii. Is there anything else you'd want to throw in there?

Kiran Krishnan:

Yeah. So sIgA, for those who are not familiar, is secretory IgA. It's the most abundant immunoglobulin in your secretory fluid, so in things like tears and saliva and into your mucosa and so on. IgG is the most abundant antibody in your blood, in your circulation, but secretory IgA is in your excretory or secretory fluids. Your microbiome stimulates the production of secretory IgA. So this data that suggests that the more diverse your microbiome is, the more interaction it has with the immune system, the more secretory IgA you would actually elicit. Because one of the things that causes secretory IgA to be released is the activation of pattern recognition receptors in your immune cells. Those pattern recognition receptors are triggered by the presence of more microbes and diversity in microbes within your microbiome.

And then there's other microbes that have a particularly good capability of pushing the immune system. Well, that's one of the things that spores do really well, is because they can actually make their way towards mesenteric lymph nodes from the Peyer's patches, and then stoke the immune system to release more antibodies and trigger more lymphocyte proliferation and so on.

So having a diverse, healthy microbiome will play a role in that. We're actually setting up a study on MegaSpore to see if we can increase sIgA in low sIgA people. But there are some nutrients, like you mentioned, Saccharomyces boulardii has data on that as well. But there are some nutrients like zinc, vitamin B, that have been shown to be able to increase sIgA production. If you have a genetic deficiency in sIgA, I don't know if there's much that can be done there. But certainly getting outside, getting more exposure to bacteria, improving the diversity of your microbiome, those will all be things that increase sIgA production.

I could talk a while about sIgA, but there's two things I want to mention about it that's really interesting. That's part of how your body builds tolerance to your own microbiome. So often what happens is you've got those two layers of your mucosa, which I explained before, but let me use this illustration to show you this. I've got it handy dandy nowadays. But remember there's two distinct layers of your mucosa. You've got the top layer where most of your microbiome lives, and then you've got this inner layer, the clean layer, called the mucin-2 layer where there shouldn't be a lot of microbes entering into this layer. If there are a lot of microbes that enter this layer, like in the case of leaky gut, then you're going to get profound inflammatory responses going on here.

But from time to time, your own commensals do make their way into this area. But what they look like typically when they're found in this area, which means it doesn't elicit a negative immune response, is they come in and they're covered with secretory IgA. So your own secretory IgA recognizes all of your commensal bacteria, and you continuously release secretory IgA that binds to the surface of your commensal bacteria. That's one of the ways your immune system knows not to attack the commensals, because it doesn't attack it because it has sIgA on its outer layer, especially if that vector is allowed to migrate on the inner part of the mucosa. So part of the way this whole mechanism of sIgA works is that the presence of commensal bacteria stoke the release of more sIgA.

And sIgA is an antibody that's interesting, because it's not designed to be highly specific to any one antigen. It has enough variation in its binding sites where one sIgA antibody can bind to a lot of different things that look like its target. IgG, for example, is highly specific to very specific antigens. It's a very lock and key mechanism. This IgG only fits this antigen and it specifically binds to that. sIgA has a lot of

flexibility in the things it can recognize, and it can bind many different antigens. So the more your microbiome causes the release of sIgA, the more sIgA you'll have floating around that will bind to cells within your microbiome. But then will also latch on to other things within your mucosa, including viruses that may be coming in, bacteria that may be coming in, toxins and may be coming in, and so on.

Michael Roesslein:

Oops, I was muted due to the lawnmower. The lawnmower is now gone. Someone was actually driving it down the road. Okay, just real quick with Saccharomyces boulardii. If people have sensitivity to yeast on an IgG or an ASCA antibody or a known tested positive for yeast sensitivity, will they react to Saccharomyces boulardii? You can't say that for sure.

Kiran Krishnan:

Yeah, it's possible. But I have not seen much reports of people being sensitive to Saccharomyces boulardii. So I would approach that with caution. If you're an anaphylactic to yeast, then no.

Michael Roesslein:

Yeah, IgE, then no.

Kiran Krishnan:

Yeah.

Michael Roesslein:

That's IgE, right?

Kiran Krishnan:

That's IgE. But if you have IgG, doesn't necessarily mean you're sensitive. So remember, IgG, and this is one of the dysfunctions in some of the testing, when you look at food sensitivity tests and you see IgG antibodies to it, it doesn't necessarily mean that you're sensitive to that food. The presence of IgG means you've been exposed to that food and your body's developed a response to it, but it doesn't mean your immune system will react to it.

Michael Roesslein:

That's my big problem with the food tests.

Kiran Krishnan:

Yep. That's a big issue. And in fact, globally, all of the immunology allergy institutes in the world have written editorials and papers against the use of IgG based antibody tests for food sensitivities, citing all of the research that indicates that IgG presence actually indicates tolerance, not sensitivity. IgE, on the other hand, would be a sensitivity reaction.

Michael Roesslein:

That makes sense. I have IgG1 subclass immune deficiency. Do you know anything about this? Will taking the MegaIgG2000 or SBIs be helpful for me or potentially harmful?

Kiran Krishnan:

So if you have a subclass dysfunction and if it's IgG type one, IgG type one is the highest amount of all the four IgG subclasses. I think it makes up 60% to 70% of the IgG in your system. IgG type one typically binds to protein antigens, like in the case of a coronavirus, it'd be the spike protein that it recognizes, versus IgG type two and four bind more to envelopes or fatty acid recognition sites.

Now, if you have a type one deficiency, then the question is, have you been diagnosed with total IgG deficiency? So typically what happens is when you have a deficiency in one of the four subtypes of IgG, you may not actually have a total IgG deficiency because your body makes up for it by making more of the other subtypes. Now, type one is the highest percentage. So if you have a type one deficiency, depending on how severe that deficiency is, you could have a total IgG deficiency. If that's the case, then there is some immune compromise going on. However, IgM could be making up for it. So what you really need to understand, and this is between you and your doctor to figure out the diagnosis and treatment, but what you really need to understand, what does your total immunoglobulin picture look like?

So then back to your question about will the MegaIgG have a negative or positive impact? The MegaIgG stays in your gut. It doesn't enter into the blood. So all of your IgG is in the blood. All the IgG you make yourself is in the blood, it's circulating around in your blood. The MegaIgG, the bovine IgG, stays in the gut and functions in the mucosa. So it doesn't affect the IgG that's going on in the blood. So it shouldn't have any effect on the condition itself, either positive or negative, that we know of. Because the positive side of the equation is that if you are suffering from immune dysfunction, meaning you get chronic infections, typically people with IgG subclass deficiency will have chronic upper respiratory infection, chronic sinus infection, ear infections, those things just keep coming up. If that's what you're suffering from, then we don't have any indication that taking the MegaIgG actually will reduce any of those effects. So we can't say that it's going to benefit your condition.

We do know that the MegaIgG benefits the gut, the toxicity, the issues going on in the gut. So certainly it will help your gut. But will it help your overall condition with deficiency? We've got no data to support that. Typically people with real severe IgG subclass dysfunction or deficiency will do immunoglobulin therapy. That's something to talk to your doctor about.

Michael Roesslein:

That's something different.

Kiran Krishnan:

Yeah.

Michael Roesslein:

Okay. While we're on it, the SBIs, the MegaIgG, what would be the main reason... How does one know when taking SBIs or IgG is warranted? And then a followup to that, which was a different question. Are the effects of SBIs or your MegaIgG specific to the gut or are they systemic?

Kiran Krishnan:

Right. So as I mentioned, the IgG doesn't go systemic, so it stays in the gut. You can see, if you measure inflammatory cytokine systemically, you could see those change to a certain degree. But that's because what happens in the gut has a systemic effect. So it's not that the IgGs are going in and floating around

inside your body and doing work. They're working in the gut, but any time you bring down toxicity or inflammation of the gut, you'll see it systemically. So that's the connection there.

How do you know when you need to take it? It's something I take every single day. It's probably, I think our second or third. It's typically tied with the MegaMucosa as far as biggest selling products in terms of volume. So a lot of people have found a lot of utility in it as a way of bringing down the toxic load in the system. Because these are immunoglobulins, they do bind things like mold toxins, they'll bind things like bacterial toxins, environmental toxins. It basically helps neutralize and reduce the number of things that cause inflammatory or toxigenic load in the gut. That's its biggest effect. And so-

Michael Roesslein:

So it binds to things. So your immune system doesn't essentially. It doesn't trigger the immune system, it catches it before that.

Kiran Krishnan:

Exactly.

Michael Roesslein:

[crosstalk] as a food antigen buffer. Like if she's going to eat something that might be marginal or questionable that her body may or may not like, we load up the IgG with that meal. And then we used it as a binder, essentially, with mold toxicity in between meals.

Kiran Krishnan:

Yeah, exactly. It's about bringing down the toxic load in the system. It's about reducing the things in the system that can trigger inflammatory response in the body. It's essentially giving the immune system a helping hand. Going, hey, we're going to deal with all of these things so you can worry about the more sinister things that may be coming in through other parts of the body. So that's the way we look at it.

Michael Roesslein:

Which can reduce an overall freak-out response from your immune system, if there's not as many things that get to it.

Kiran Krishnan:

Totally. Yeah. And again, remember one of the main points of my talk was that the inflammatory pathways and the inflammatory signaling is the way that your microbiome signals to your immune system the presence of pathogens and infective agents and contagions and so on. And so when you have lots of inflammatory signals going on, then the microbiome can't effectively signal to your immune system when something is present. That's one of the reasons why, especially in this case, this virus seems to trigger inflammatory responses. Because it's trying to dampen the signal so that your body doesn't know exactly where that signal is coming from.

And that's a really important part of it, is that overall in being resilient, overall in having a tiptop functioning immune system, one of the features is keeping inflammatory responses lower and dampened. If you can have a lower net rate of inflammatory response in your body, then when the inflammatory response is needed, it's going to be much more effective. So one of the ways that I think about doing that is utilizing something like this that binds up and neutralizes lots of things that can come in and stoke that inflammatory response in the body.

Michael Roesslein:

Okay. All right, great. This is interesting, and I've heard you talk on it before. Given microbiome's role in immune response, is there any research showing possibility of a better vaccine delivery response if given orally in some manner?

Kiran Krishnan:

Yeah. One of the things that really troubles me when I look at vaccine research is why do they keep doing intramuscular injection of vaccines? It's just not a great sampling site for your immune system. The vast majority of things that enter your body enter through the respiratory or the digestive tract. And the respiratory mucosa, the digestive tract mucosa, is a huge site of sampling of things, and that's where you elicit a really good immune response to antigens that you encounter into your system. So that part, I still am not still quite wrap my head around why they keep going the intramuscular side. I know with a lot of kid vaccines, they do it as a respiratory one, which makes more sense.

Now, the work of Simon Cutting is really interesting. This is actually one of the reasons why we ended up getting in contact with him, because he's got a bunch of work in a company called SporeGen, which is based in the UK, where they are putting antigens on spores. Like say tetanus antigen, it's for tetanus vaccine. So instead of injecting yourself with a tetanus vaccine, which is a tetanus antigen, they're sticking it under the spore and then delivering the spore orally.

What they've shown in published research is that you get a much better amplified immune response, especially the adaptive side of their immune response, to the presence of the tetanus antigen when sent in with the spore than compared to when it is injected into the arm. So that's the whole focus of the company called SporeGen, is they are trying to develop orally derived vaccine vehicles using things like spores to take it into the Peyer's patches, which is the largest area of your immune cells, sampling, and then deliver it to the immune system going, hey, take a look at this and pay attention to it. Then your immune system elicits a beautiful antibody response against it, and then you have some degree of protection against an antigen next time you see it.

So one of the ways I think about, as you're going through and encountering antigens from different places, one of the reasons I like keeping my spore count up in my system is the spores tend to help with antigen presentation. So as your body encounters antigens, the presence of spores may help the immune system respond better to the presence of those antigens.

Michael Roesslein:

I remember we talked about that years ago, so I wasn't sure if anything happened there. But it makes more sense because that's how we would encounter an antigen.

Kiran Krishnan:

Totally. Yeah.

Michael Roesslein:

One of the problems with vaccine reactions is... Well, I don't want to go down a rabbit hole that's going to get us taken off the internet. But is that never in the world, it's literally impossible for an antigen to just appear in your blood.

Kiran Krishnan:

Right. Yeah.

Michael Roesslein:

It's in your arm. You don't get smallpox in your arm.

Kiran Krishnan:

Especially something like a respiratory virus, like influenza, for example. If you were to encounter influenza, which we all do, you get it through your respiratory tract, you breathe it in, you touch your eyes, nose. That's how it comes in. You never get influenza naturally getting injected into your arm. So I don't understand why they don't use the more natural sampling site as a way of delivering the vaccine. That may be an issue of production packaging. I don't really know. But it would make more sense to use the natural areas of sampling to develop an immune response.

Michael Roesslein:

Makes sense. That's really interesting. Do you have a theory as to why healthcare workers are getting such severe and dangerous cases of COVID-19 compared to the general public? And I've noticed this too. And my own theory is that with viruses, it comes down to load, and healthcare workers are exposed to the highest viral load when working around a room full of positive patients, and with masks and re-breathing and all of that. Is that a good layman guess?

Kiran Krishnan:

That's an absolutely great guess. I would put that as probably one of the first reasons. Viral load makes a big difference. You can actually get a small load where there's not enough virus to create enough replication to actually make you ill, but it's just enough to elicit an immune response and your immune system recognizes it, deals with it, and then you're fine. But if you get a huge load, then that huge load makes it much harder for your immune system to start bringing it under control. And remember with this particular virus, one of its functions is it amplifies the cytokine of the chemokine response in the cells. So when you get a big viral load, you get a bigger inflammatory chemokine response right off the bat, which makes you feel sicker and makes you feel more lethargic, more muscle pain, all of that stuff. Remember, all of the symptoms that you feel from the illness is part of the immune response to the illness, not the virus itself. So the load is a big part of it.

The other part of the guess I would create is that they are under a tremendous amount of stress. The work conditions, what they're dealing with, they're on the front lines of this. We know stress dramatically suppresses the immune response. And stress also increases the virulence of latent viruses. So now you've got other things your immune is also trying to deal with that are proliferating because of the stress levels. Things like cytomegalovirus and Epstein-Barr virus and papilloma viruses, all of those things start flourishing in your body the moment they sense the stress signals. So to me, it's a combination of those two things, probably stress, lack of sleep, and then of course, viral load itself.

One of the things that we're seeing maybe is that this particular virus doesn't transmit as easily in just a general sense. And not everybody is equally contagious. Some people are more super spreaders. And that was actually a really interesting exercise that was done before this pandemic started in a documentary that the BBC did on pandemics. Is that typically in pandemics, the vast majority of people get infected by a very small number of people. You've got single individuals that can infect, 500, 600 people in a chain of events from over a few day period because they are highly contagious. Why are they highly contagious? Well, whatever the composition of their bodies, they're immunocompromised

or suppressed, the virus gets lots of chance to replicate. Then their viral load that they spew out in their respiratory droplets and all that, it tends to be much higher than the next individual next to them. Because remember, a lot of people could have active viral infection and you can't even pick up virus through the nasal pharyngeal swab. So the amount that they're putting out is actually really quite small to almost nothing at all.

So those factors play a role in how well it gets spread from person to person. If you are a medical person in a ward with a lot of people spreading COVID, you're getting saturated with it all over the place.

Michael Roesslein:

Giving that clotting seems to be part of the COVID-19 presentation for many patients, and we are not doctors, we're not treating or curing or preventing COVID, are there concerns around vitamin K2?

Kiran Krishnan:

No, it's a different kind of clotting. Number one, vitamin K2 doesn't really impact the clotting cascade much. It's vitamin K1 that's really doing it, and that's in the liver. The clotting that's occurring in COVID is more systemic. It's occurring in tissues that are getting damaged because they're triggering of a particular type of inflammatory pathway. One of the responses to that inflammatory pathway is clotting in that local area. So these aren't people that are getting clots created through platelet aggregation in their liver or increased clotting factors in the liver. The studies show that it's driven by an inflammatory process, not driven by increased clotting factors. So it's a different mechanism that's going on.

And again, maybe many of you don't follow me on social media, but I posted a study recently, in the last couple weeks, that showed that K2 status, in a study in Norway, was a very close correlation and determining factor in the severity of the COVID infection. This a well published study. So in fact, K2 seems to help with the functionality of the system.

Michael Roesslein:

Yeah. I saw that. You're good at the Instagram. Do you do that yourself?

Kiran Krishnan:

I try to, yeah. I should be posting every day.

Michael Roesslein:

Instagram's a pain in the ass.

Kiran Krishnan:

Oh, it is. I'm still learning how to do it, and I'm-

Michael Roesslein:

... pain in the ass.

Kiran Krishnan:

Oh, it is. I'm still learning how to do it and I'm [crosstalk].

Michael Roesslein:

The stories ... we're too old. It's always, Jesus ... I always admire anyone, I live on a side street with a 20 mile an hour speed limit. Did you hear that?

PART 2 OF 4 ENDS [01:02:04]

Kiran Krishnan:

That did not sound like they were doing 20.

Michael Roesslein:

No. I always admire anyone our age or older that actually has an active Instagram. It's so much more work than Facebook.

Kiran Krishnan:

It is. Oh, my God, it is.

Michael Roesslein:

It's not like a one-button share, you have to type things in the phone and use hashtags. It's exhausting. Anyways. Let's see, why do we react to foods after being on elimination diets? Does Mega IgG help with this? I think they mean for like a long period of time. Because people, now the elimination diets were a huge thing, and they still have their place, but then it's reintroducing the diversity, which we've talked about a lot.

And a lot of people are now noticing like, oh, I've been on a super strict diet for four years and now I can't eat anything. And they weren't originally reacting to all those foods, they just took out all the foods that they say people react to, to be precautionary. And then all of a sudden they are reacting to things they didn't use to. Can you just briefly explain, that goes back to tolerance and diversity, right?

Kiran Krishnan:

Totally. Yeah. Again, the tolerance is built by exposure. So if you go through a long period of time where you're not getting exposed to certain antigens, your body might forget that tolerance, because remember the antibodies and all that don't last forever, necessarily, in the system. The antibodies that afford some of the tolerance. So then instead of getting this really mild, really non-consequential antibody response, then you start getting an inflammatory response instead, because to your body now it looks like something new that it's never encountered.

So that's part of the problem with the shortening of the exposure to normal antigens, especially proteins from different sources. So again, tolerance is maintained by having continuous immune response to those antigens. And it's the adaptive immune response that comes along with the Treg response, the T regulatory cell response, that dampens any immune response to that food antigen.

The way all of that happens is in the context of the microbiome, the microbiome is the thing that upregulates the Treg system, the regulatory system. So if you're continuously introducing food, like on a regular diet, those antigens are entering the system. They're being presented to the immune system in the context of the microbiome, the microbiome helps the immune system upregulate the T regulatory cells. So then you build immune tolerance against those antigens.

Now, if those antigens go away, and one of the consequences of elimination diets is that your microbial diversity goes away too and start shrinking dramatically. So now you've got a two prong reason for

developing immune dysfunction, is you don't have the antigen to continually get exposure and tolerance to. And you don't have adequate microbiome diversity to elicit that tolerance response. So you've got a double whammy in creating intolerance of food particles.

Michael Roesslein:

That makes sense, double whammy.

Kiran Krishnan:

Double whammy.

Michael Roesslein:

What's your opinion overall on pathogen protocols, parasite detoxes, parasite cleanses, things of that nature?

Kiran Krishnan:

In my view, they're often used for wrong reasons. I think, especially with parasites, as I mentioned earlier, if you truly think parasites are a problem, then you should definitely get a good pair of cytology tests to understand if they really are a problem. In general, in the Western world, and in the modern developed world, parasites are typically not a problem for people. Looking at CDC data and other national databases, you really see very few true parasitology problems in people in the Western world. Unless you've traveled overseas, and you were in the Amazon for a while, or you went to Asia and you were eating a lot of street food and so on.

Then you could have picked up a parasite or two that could be causing you lots of issues. But for the most part in North America, parasites aren't really a major issue. So I would encourage people to get a full, comprehensive parasitology test before doing any of those kind of detox things. Because a lot of those things can really put a lot of stress on your liver, on your gallbladder, on your gut microbiome. And it's not necessarily good for you, you may not be needing it.

Michael Roesslein:

Yeah. Those can be pretty harsh. [Kiran's] Instagram is @KKiran_00. I looked it up.

Kiran Krishnan:

Yeah, so that, actually, is another piece of evidence that I'm terrible at Instagram and social media, because that's a terrible Instagram name. Because it's not logical and it's hard for people to find it.

Michael Roesslein:

I don't even use mine professionally. So you're way ahead of me and I don't ever put anything on it. It's just a time waster that I scroll through and watch your stuff. So someday I will effectively use Instagram. All right, do you have any knowledge about ... How does mucosal immunity and microbiome help trigger T-cell reactions and other immune reactions and which are specific to COVID-19? I think you talked about this in part one.

Kiran Krishnan:

Yeah. Yeah. I mean, all of the immune response occurs in the mucosa. That's the primary site of action. Every pathogen that enters your body enters through a mucosal layer, so it's going to end up in the mucosa. The mucosa has immune cells, they're floating around; dendritic cells, macrophages, and then of course the epithelial cells, or the endothelial cells, depending on which one the mucosa covering. If they start getting infected, they will release the cytokines and chemokines, and then your microbiome in that area will also release the cytokines and chemokines to alert your immune cells that there's a problem there.

Then the antigen presenting cells come along, they start eating up the antigens, they start eating up the viruses, eating up the bacteria, eating up the infected cells. And then presenting that contagion's antigens to your T cells. And then your T cells will then often go to a localized lymph node where they will start proliferating and that's where the battle occurs. So that's why when you have an infection somewhere, you can find a tender lymph node somewhere in that proximity. Because that's where the T cells are going to be draining into, to proliferate and then mount up and get ready and then come back out to the site of infection to neutralize the infection. So all of it occurs in the mucosa, that is the battleground for infection.

Michael Roesslein:

All right. I have a few questions about your BiomeFX test and I'm going to just lump them into one, to bang out a at once. Does it test for calprotectin or any other type of inflammatory or LPS markers? Does it help identify dysfunctional microbiome and how?

Kiran Krishnan:

So, yeah, so we do not test for calprotectin or zonulin. We do not find those to necessarily be that indicative of anything. So what do you do with the calprotectin test? Well, it tells you that there's something messed up in your gut. So if you do a fecal calprotectin and the fecal calprotectin is elevated, you go, oh, okay. That means something is wrong with the gut. Well, what is wrong with the gut? You still don't know.

But if you're even doing the tests, it already indicates that something's off in the gut because you're feeling symptomology. You're feeling cramping, bloating, indigestion, whatever it may be. Bloody stools, whatever it may be, calprotectin is just telling you, hey, something is wrong with your gut. It still doesn't tell you what is wrong with your gut. So we are very focused on identifying the what, we're very focused on giving you a clue as to what may be the problem in the gut.

So that's why we focus our tests on the microbiome component of the gut. So we're not measuring markers, we're not measuring inflammatory markers, anything like that. We're looking at the personality of your microbiome and specifically focus on the functionality of various groups of microbes within the microbiome itself. And a lot of it will tell you that these groups of microbes, based on relative abundance data, tend to be higher in your sample than they are in a large cohort of healthy normals.

Meaning that this function that you have going on in your microbiome may be the cause of your symptomology or your problems. And that's the only way you can know and understand what may be going wrong in your microbiome. I'll give you one quick example of that. Look at sulfate reducing bacteria, that's one of the things we measure. There's a few bacteria that are sulfate reducing bacteria. Now it's normal, obviously, to have sulfate reducing bacteria within your microbiome. They do a job within the microbiome, but if your abundance of sulfate reducing bacteria is too high, then what tends to happen is they will tend to take sulfates from your food and convert them into hydrogen sulfide.

And that hydrogen sulfide is very inflammatory to the large bowel and can cause lots of symptoms like, loose stool, constantly cramping, in some cases even bloody stool. And in fact, in some cases it's actually tied to a risk factor for colitis. So you would never know, and here's the thing, lots of healthy foods are high in sulfates. Things like fish and garlic and leeks and artichoke, all these things tend to be high in sulfates. So you might be eating a diet that you think is healthy, and it is a healthy diet, those foods themselves are fine. But because your sulfate reducing bacteria are too high, what happens to the food in your gut actually leads to negative symptomology and then negative results.

Those are the kinds of things that we look at. At the moment we have something like 18 or 19 of those kinds of functions. We have a 2.0 version of the test that's coming out in June that has, I think, 31 of those functions. So it's ultimately the true functional microbiome test, looking at the functions and dysfunctions within your microbiome. It's not a list of, here's all the bacteria you have. Oh, and this one bacteria is high and that one bacteria is low. None of that is meaningful at all. What's meaningful in your microbiome is a functionality of different groups of [crosstalk].

Michael Roesslein:

What's happening, what's not happening.

Kiran Krishnan:

Exactly. It's a personality test for your microbiome is one of the ways-

Michael Roesslein:

Perfect, that should be on the box.

Kiran Krishnan:

Yeah, because really, if you just look at a personality test for somebody, these aren't absolutes. And nothing is absolute in microbiome analysis either. But if you do a personality test on somebody, you get an understanding, or you can be predictive to a certain degree, of how that person's going to respond in a particular situation. It's not an absolute, it's not 100% sure they're going to respond that way, but you can make predictions. And that's exactly what we want to do with this microbiome test. It's functional, it's focused on function/dysfunction, and it's a personality test.

Michael Roesslein:

All right. I'm going to do a few more and then we got to get to some recommendations, tips, research. Because there's a lot of people in the chat are wanting to talk about the products too. So I just want to do a few more. I'm almost through them. I might not make it to all of them. If there's a handful I don't get to, I'll email them to Kiran and we'll get them to you at some point.

Trying to just pick out ones that have been asked, some people are persistent and put them in the chat and the Q&A, and my email. Someone who's had, for six years, staph. No, reoccurring ... Oh, where did it go? I'll find it in a second. How about, does having lost part of a colon effect the microbiome and does MegaSpore or anything else that you've produced help with a situation like that?

Kiran Krishnan:

Yeah, I think having lost part of the colon will certainly have a impact on your microbiome because you have less surface area in the microbiome, of course, which means you have less microbes and less functionality. A very recent study on colonic colonization of the microbiome actually showed that you

have completely different types of microbes in different sections of the colon. So it's not just this big fermentation vat where you've got lots of similar bacteria all over the place. So there are very specific functionalities within specific areas of the colon.

The sigmoid colon looks very different than the transverse and the ACE ending and so on. So it depends on which section you lost, some of those microbes may not be present and thus may not be providing their functionality. Now taking MegaSpore and all that, that's not going to replace the functionality of that part of the colon, but certainly the focus has to be, how do we optimize the rest of the colonic function?

And we know, in general, that in looking at the different sections of the colon ... Because in our publications, we did look at the changes in different sections of the colon. So we do know that that taking the combination of the spores and the prebiotic do improve the microbial diversity and the ecosystem within each section of the colon. So minus the part that you're missing, taking that stuff is really going to enhance the function of the microbes within the remaining parts of your colon. Which is, at this point, the best thing you can do,

Michael Roesslein:

Partner has had strep for six years on and off after course of anti-malarial antibiotics, thoughts on ways to change this cyclical infection.

Kiran Krishnan:

So in part it'll depend on where this strep infection is. Strep, remember, is a normal part of our ecosystem in our bodies. Strep and staph, both of them. They are very present in the sinus cavities. More research has come out that they are probably, the strep and staph, are probably the two biggest drivers of infection in the sinus cavity. So we'll take that as an example, because I don't know where in the body you're talking about. Because you can have strep infection on your skin, you can have it in your heart, if you have rheumatic fever, different areas of the body. So let's take the sinus cavities, for example.

The studies show that the difference between people that get continuous rhinosinusitis versus those that don't, in the case of continuous rhinosinusitis, it is still the strep and staph that are driving it, but the difference between people that don't get it and the people that get it is not the presence or the absence of staff or strep, it's the diversity of the rest of the microbes within the sinus cavities. So when you have higher diversity in that ecosystem, strep and staph have less infectivity rate. So they don't infect in the same way.

They are opportunistic organisms. So given the right terrain, as we talked about earlier, they will express some of their virulence factors. One of the best ways to keeping them under control is having a higher diversity of other microbes that keep them in check. So depending on what the condition is, you should be talking to a doctor if they've got continuous infections, there may be other risks there. But aside from that, the thing that we could talk about is having a better diversity in their microbiome keeps those under check. And obviously using the spores will help the diversity, improving the diet will help the diversity, and then getting outside more helps the diversity significantly as well.

Michael Roesslein:

Describe any specific ideas, probiotic, food, lifestyle, or otherwise for controlling reoccurring staph aureus, I think aureus. A-U-R-E-U-S, staph aureus skin infections. I don't know if that's a specific type of staph or ...

Kiran Krishnan:

Staph aureus is a very common vector. We all have it on our skin, we all have it in our guts, in our eyes, in our nose, everywhere. Staph aureus is everywhere. Staph aureus is on the skin and typically battles with something called staph epididymitis, or epidermidis, and typically what they show is that when staph epidermidis is lower then staph aureus has a chance of actually rearing its head and causing more infection. So in that case, it's really the terrain issue because the staph is always there. It's there on everybody.

But if the other microbes are at lower level, then it's going to rear its head. There's a couple of things. One, MRSA, which most people have heard of, methicillin resistant staph aureus, is one of the types of staph aureus that people can have. There was a large NIH study that was published about a year and a half ago that showed that people who were well colonized with bacillus subtilis actually had no MRSA levels at all. Those that weren't colonized with bacillus subtilis tended to have more MRSA colonization.

So the bacillus does a good job of fighting with the staph aureus. So that's one of the things to consider is getting some spores into your system. If it's a skin area, now let's say it's just one area of your skin that tends to have more of that staph type infection, of the aureus infection, and you have other areas of the skin that are normal. One of the things I've recommended to people is to engraph normal stuff to this side.

So let's say you've got a patch here that you typically get staph aureus infection and the rest of this arm is completely normal, which means that the microbes are normal here. One of the things I would recommend people doing is taking a wet Q-tip and swabbing the normal area and transferring it a few times a day to the affected area. I actually had a few people do this and they actually saw some measurable improvements. You're basically engrafting from the unaffected side to the affected side. And hopefully you're bringing more staph epidermidis to this area that can then combat the aureus. So that may be something to look at doing as well.

Michael Roesslein:

Makes sense. All right. I think I'm going to have to type the rest of them to you because I want to get to product questions and a little bit of recommendations, tips, suggestions, overall for ... I mean, you had the one slide at the end of your presentation. So go back to part one, it's the last slide. So if you go to the end of part one replay, the last slide had a bunch of recommendations on it.

Kiran Krishnan:

If you want, do you want me to pull that up and show it-

Michael Roesslein:

Yeah, if you've got it we can just have it on the screen while we talk about this.

Kiran Krishnan:

Oh yeah, I think you have to enable screen share.

Michael Roesslein:

Oh, screen share. Okay. That should be on. How does earthing grounding effect the microbiome. I know that it has effects on the nervous system and I know that being in contact with nature has a positive effect on the microbiome. That's all that I know.

Kiran Krishnan:

No, and I think that's about right. I have not seen any studies that show that earthing and grounding specifically change certain microbes, but I think just the act of being in nature in itself, and getting in contact with dirt or natural ground, is going to have a positive impact.

Michael Roesslein:

Oops.

Kiran Krishnan:

Okay, so I'll just run through these recommendations real quick. And these are things, many of the things I've mentioned, and these are no different when it comes to trying to optimize immune function through the microbiome, which is obviously a really big, important part of immune function. Number one is diversifying the diet. I've given lots of recommendations on that. And again, that's about increasing more of the foods, the types of foods, that you consume on a regular basis. And that's more on the plant based side of the foods.

You can, of course, try to increase the diversity in the various proteins and meats that you eat, but that's not going to give you the same kind of impact on diversity. Because ultimately when it comes to protein, ultimately they get broken down into their amino acids. When it comes to carbohydrates and fibers and soluble/insoluble, resistant starches, oligosaccharides, all of those have very different carbohydrates structures that only certain microbes within your large intestine, especially, can utilize.

And so it provides an increase in diversity in those microbes. So that's an important thing. Another way of increasing diversity is, of course, just fasting. Fasting does a couple of things for your microbiome and immune system. Number one, it increases the diversity within your microbiome, which is great for your immune system. You have a bigger neighborhood watch, a more diverse neighborhood watch for your immune system. But the second part is, fasting also helps the housekeeping mechanisms. So it cleans up damaged cells and it turns on autophagy and mitophagy. So it cleans up damaged DNA and cells and debris and cellular debris, and it helps fix damaged mitochondria and replace it with better functioning mitochondria.

So all of this housekeeping, cleanup stuff is really important for your immune system to function in tip top shape in general. So fasting, add that to the diversifying diet, lowering stress is really important. It sounds really casual, but just a couple of main points on the lowering stress. Number one, stress is one of the biggest drivers of leaky gut. Stress induced intestinal permeability in a 2015 publication in The Frontiers of Immunology, and that's a tough journal. I can tell you, I just had a paper of ours that we were trying to get published in Frontiers of Immunology and we had to go through quite a rigor on the peer review process. So it's a good journal with really good peer review.

That study showed, and they concluded based on meta analysis of lots of other studies, that intestinal permeability driven by stress is the largest driver of mortality and morbidity worldwide. And the relate that to the types of chronic illnesses that stress induced leaky gut endotoxemia drives. And then of course, all of the immune dysfunctions that it drives as well. So that's a big part of it is lowering stress. The other thing that occurs during a stressful state is all of your latent viruses and bacterial pathogens start to rear their ugly heads at that point.

So there are lots of viruses and all that within your system that learn to recognize your stress hormones as a signal to tell them that your body is compromised and your immune system isn't functioning well. So this is a good time to proliferate. For example, herpes simplex virus, cytomegalovirus, Epstein-Barr virus. They all tend to increase their virulence factors. Streptococcus, staphylococcus are another

example, we just talked about those kinds of bacteria. They all increase their virulence factors during a stress state.

So if you imagine your immune system is potentially encountering a new contagion, and then at the same time, if you're highly stressed and your body is dealing with all of your resident contagions amplifying themselves as well. So stress becomes a really important thing to manage. And again, there's lots of mindfulness work you can do, breathing exercises, there's endless recommendations out there of how you can try to manage your stress. Getting outdoors is really important, and I don't mean just walking around the sidewalk of your neighborhood. That'll have some benefit, of course, but if you can get out into a natural environment, like an area where you can go on a hike, be within the forest a little bit, be within natural soil.

Around here, around my house, we have lots of forest preserves. I've been going, over the last couple of weeks, doing lots of mountain biking in the single track trails in the forest, and then you stop and you enjoy and absorb that an environment. That has a huge impact on the diversity of microbiomes. The spore based probiotics, of course, we've got a bunch of research on their functions, but one of the key things when it comes to the immune system is the ability to train the immune system and trigger those pattern recognition receptors. Trigger toll-like receptors, different things that keep the immune system ramped up and keep the immune system functioning.

They also bring about the ability to diversify the microbiome, which of course is a very important part of immune function. They also compete against pathogens that are within your system so that you can bring down the negative impact that pathogens may have on the system. Which may stoke a more inflammatory response, which will then dampen the inflammatory signals from new contagions entering the system. Focus on leaky gut solutions, because remember leaky gut causes system-wide inflammatory response. The same exact inflammatory cytokines that are being used by the microbiome to alert the immune system to the presence of a pathogen.

Those are the same inflammatory messengers that get amplified because of leaky gut. And that becomes systemic. So imagine if a fire alarm is a signal that a fire exists, you want to make sure that throughout the day, you don't have any other fire alarms except for when a fire exists. So that then the firefighters can hone in on that singular fire alarm. If you've got fire alarms ringing all throughout the system all the time, then when there is an actual, really dangerous fire, that signal is going to get drowned down and you're not going to get firefighters getting to that area.

So leaky gut is a big, big driver of systemic inflammation and can compromise immune response in a dramatic way. And that, again, goes hand in hand with bringing down inflammation. In the case of diet, if you do have foods that stoke inflammation, for example, gluten. Gluten is a food that does stoke inflammation in virtually everybody, even if you're not gluten sensitive. Even if you are, if you're not gluten intolerant, even if you're not celiac, studies show that everybody gets transient permeability when you're exposed to gluten.

And for that reason that transient permeability can cause a phase of inflammation for a period of time after you've been exposed to the gluten. So if you can avoid certain things, like gluten, in your diet. If dairy's inflammatory to you then avoiding dairy for a period of time to bring down that inflammatory response. Those are things that can help from a dietary standpoint. Of course, we know that processed foods are inflammatory. We know foods that tend to be high in oxidized fats, like deep fried foods that are fried in old oxidized vegetable oils. Those things all can be very inflammatory. So reducing the exposure to inflammatory foods.

Prebiotics are huge, major components to the function of the immune system. We cannot say enough, and people don't think of prebiotics as part of your immune regimen. But the oligosaccharides, in

particular, and the short chain fatty acids that they elicit are really critical for immune function. The acetate, as I mentioned earlier, plays an important role in stoking that interferon response in the body. That interferon is the first and most powerful antiviral response in the body, that is dependent on acetate.

Butyrate, which is also produced as part of the short chain fatty acid collection, is a very important energy molecule for your circulating dendritic cells and macrophages. Those are the first antigen presenting cells that are going to go find those antigens and present it to the T cells and B cells for immune response. So your short chain fatty acids are going to be really important, and those are typically stoked by oligosaccharides. So oligosaccharides are really critical.

Right now, I kind of wax and wane a little bit with the prebiotic that we use. I use the Mucosa product every day, I use the IgG product everyday, and the spores everyday, but I wax and wane a little bit with a prebiotic. But over the last several weeks, month and a half or so, I've been doing the prebiotic religiously because the studies are so clear on the importance of oligosaccharides in immune function. So, in fact, that's what I'm drinking right now. And then polyphenols and omega fatty acids, now this is really important to speak to because omega fatty acids bring down inflammation in that arachidonic acid pathway. And that's really important. That's inflammation that's stoked and triggered ...

...and that's really important. That's inflammation that's stoked and triggered by tissue damage that's going on in the body. The tissue damage in the gut lining, in the vascular tissue, and so on. So tissue damage triggers that inflammatory response through the arachidonic acid pathway. That is a very powerful inflammatory pathway. And again, it causes a lot more inflammatory alarms in the body. Now, that inflammatory alarm, when you couple it with a cytokine storm response, really creates lots of issues within the body from an inflammatory damage perspective. Not to mention, of course, it drowns out the inflammatory signals that are required where the microbiome is alerting the immune system to the presence of a contagion.

So using something like omega fatty acids, especially ones high in EPA and DPA and pre-resolving mediators, that's what we created with the MegaOmega, that plays a significant role in dealing with the inflammatory response and the arachidonic acid pathway. Polyphenols are extremely important for diversification of the microbiome, for production of things like urolithin so that you get better mitochondria function, better energy production. Your immune system has better fuel. In order to function as an immune system, it cleans up damaged mitochondria, which can suppress immune response. And then polyphenols also have been shown to bring down inflammatory response in the gut. So again, lowering that net inflammatory blanket in the body so that the signals from localized areas that are getting infected are much louder and much clearer, so the immune system can get to that site. So those are the most basic recommendations, and I don't know if we want to jump into questions from there.

PART 3 OF 4 ENDS [01:33:04]

Michael Roesslein:

Yeah, that makes total sense, and I do have some product questions that would be great to follow that up. You have Ava, whose Mira's mother. Hi, Ava. She's on here. She's a case study actually on our website. Ava was the first person that I ever gave your products to, was the MegaSpore to her. And then her pet allergies and dust and mold, all these allergies and asthma, she had forever just went away really quickly. And then I called your company and was like, "What is going on?" And then we talked.

Kiran Krishnan:

Yeah, I remember that was the first thing we talked about.

Michael Roesslein:

Yeah. I was like, "Can you explain to me why this woman no longer has asthma?" And then two hours later, I was like, "Can you come on a webinar?" And then that all happened, but said "I've had a major reduction in asthma from household pets." She actually comes over to see ours all the time. And we brought pets to their home and we're able to live there for a little while. And she's a therapist and she couldn't see clients who had pets.

Kiran Krishnan:

Yeah.

Michael Roesslein:

It was so severe. So she said she's been taking two MegaSpore a day for the past five years. Is it safe to take that amount indefinitely? And should I add prebiotics?

Kiran Krishnan:

Yeah. So it's absolutely safe. And not only is it safe, it'll continue to help. It'll continue to help maintain the system. It'll continue to keep your immune system in the right balance, which is really important. Because keep in mind, we've got lots of things all around us that continually drive the microbiome into the wrong direction. That continuously drives the immune system in the wrong direction. So having those spores in there to try to elicit some balance is always useful. And for me the IgG is really important. While you may consider using the MegaIgG. I know that's a key thing that Mary uses. We've all got all kinds of toxicity around us and bringing down that toxic load, and bringing down the, the impact on the micro, on the immune system is a big help to a lot of people's systems.

That's one of the products I use religiously, the MegaIgG. And I would say the prebiotic, if you can start adding it in the increase in butyrate, the increase in the keystone strains that we see from using the prebiotic cannot be overstated, as to how important that is for just overall health and function in a longterm. You'll see some short term benefits on it, but in general, the longterm health of your microbiome, maintaining the diversity of your microbiome is absolutely critical to longterm health.

One of the features of getting older is that your microbiome diversity starts to shrink over time. And as your microbiome diversity starts to shrink, you become more susceptible with higher risk for chronic illness. That's a very clear interaction between microbial diversity and then increased risk for chronic illness. And so one of the keys to aging more gracefully and with more resilience, is maintaining that diversity. So the prebiotic plays such an important role in that aspect. So those are the two things you should think about adding into your daily regimen.

Michael Roesslein:

Yeah, you just had that study get published with the prebiotic and the MegaSpore together too. So the two of them are pretty powerful. I actually noticed more in the way of my own digestion optimization when I started the prebiotic than I had with the spores alone.

Kiran Krishnan:

Yeah. And that's exactly why we have it. These are all really important adjuncts to what the spores do in the gut, the IgG, the prebiotic, the omega, they really enhance aspects of what the MegaSpore is already trying to do in the gut.

Michael Roesslein:

And I should say this out loud, I've put it in the chat. We are doing a special right now in our shop for every Microbiome Labs product in our shop, for the next few days, you get five bucks off for each product added to the cart. That's something we've never done before. And it's only for the Microbiome Labs products. So you order five of anything, it's 25 bucks off. And there's a code there ML5OFF it's in the chat box. ML5OFF. It's not case sensitive. I put it all caps. The links to the shop is there. That's a sale we've never done before. I wanted to celebrate this amazing presentation and give people an option to try a whole bunch of different things. So I have a question here that would be really good to answer, that I'm going to paraphrase because it's about a four year old, but it could be about anybody.

And it says, "My four year old is taking MegaSpore for the last two weeks." And to answer the question, I just posted Ava's case study link in the chat too, if you want to read about Ava's story with the pet allergies. But four year old is taking MegaSpore for the last two weeks. When and how to add the Prebiotic and or the MegaMucosa, how to stagger titrate or introduce. I know you guys have a preferred way to kind of do that. And I think a couple of weeks in on the MegaSpore is a perfect time anyway, to go with that. So can you just go... Because you guys call that trio, the total gut restoration. That was the first three products and that's kind of the foundational thing is the MegaSpore the Prebiotic and the Mucosa. Where the rest of the products kind of have like unique uses or situational uses. That would be more specific at times.

Kiran Krishnan:

Yeah.

Michael Roesslein:

So how do you stagger those, I guess, how do you answer this? Stagger, titrate, introduce?

Kiran Krishnan:

Yeah. So he's been taking the probiotic for four weeks?

Michael Roesslein:

Two weeks, four years old.

Kiran Krishnan:

Two weeks. Okay. So in the next week or two, you would look at adding in the Prebiotic. For a four year old, you would add somewhere around a quarter with scoop. You don't need to do a full scoop. You would add it in... My kids, for example, take it in a smoothie, which they both seem to like smoothies and are happy to drink them. One of our ways of getting more vegetables into their system, but they're happy to take it in a smoothie. So you would add about a quarter scoop of the... And there's a scoop in the jar itself. So you do about a quarter of that scoop. Mix it in water juice whatever you'd like that he would be more comfortable taking. And then give that to him daily and have him sip it throughout the day.

He doesn't have to sit and drink it all at once. It can be kind of his drink for the day as he's playing around and you would have him keep taking sips of it. After a couple of weeks of introducing that, you can try to go to a half a scoop. You will never need to really exceed half a scoop or four year old, typically somewhere between a quarter and a half of scoop is perfectly fine. He's getting plenty of oligosaccharides into a system. And, and it's having a huge impact on his microbiome. That may be all he needs at that point. You may or may not need to move to the MegaMucosa, which typically happens in month three of the cycle. So you can assess him at the end of that second month, once you've already introduced the MegaPrebiotic. And remember you maintain the spores throughout that time, you can kind of assess, and whatever the end points are that you're looking at.

If he's doing really well, you may not need to go to the MegaMucosa at all. At four, they've got a lot of flexibility in the microbiome. They can make significant changes in their gut lining in a very short amount of time. So you may be in a perfectly good place just right there itself. If he's continuing to have issues, then you can go again with about a quarter scoop of the MegaMucosa added into that mix. You can mix both the prebiotic and MegaMucosa together and again, shake it up in juice or water or whatever is a preferred way for him to eat it, get it, and then have them sip it throughout the day.

Michael Roesslein:

Yeah, we really... I do about a half a scoop of day right now myself too. We make a drink, Mira calls it, her hydration beverage in the afternoon. It is a full big water bottle like this size.

Kiran Krishnan:

Mm-hmm (affirmative). Oh yeah.

Michael Roesslein:

With a half a scoop of Prebiotic, a half a scoop of Mucosa. And then we put a little electrolytes in there, a little vitamin C, a little magnesium and some sea salt for minerals. And just shake that all up and drink that every afternoon.

Kiran Krishnan:

Nice.

Michael Roesslein:

Over the course of a couple hours. See anyone experienced constipation... So someone asking me, they experienced constipation with MegaIgG and was wondering if you knew anything that there could be a mechanism there, or...

Kiran Krishnan:

It's interesting. I mean the medical food version, the prescription version of the MegaIgG is for chronic diarrhea. So it's there to treat basically diarrhea induced by pathogens or inflammatory processes in the gut. So that's what it's there for. The indication is, I think it's something like the dietary management of chronic diarrhea from irritable bowel syndrome, inflammatory bowel disease, and so on. There's a number of conditions mentioned. So if it's caused constipation in you, and maybe it should be a temporary thing. And the question is, did you go from loose stool to constipation, or regular stool to constipation? That would be part of the question. Let's say you went from loose stool to constipation.

That's a normal transition. And then you should go into regular stool formation because it seems like the microbes that were controlling the bowel movements were driving constipation.

And then now they've basically, those microbes have been dampened. And then now you've got nobody really in control of the bowel movements, but that should shift over a few days. And then you should have regular bowel movements. If you went from regular bowel movements to constipation, then maybe back down to the dose, and see if that changes. If it doesn't, if it lasts more than two or three days. It's hard to say if it's related, it's not a report that we get very often. I'm trying to think of if we've had anyone that I know of-

Michael Roesslein:

We haven't. It says, "No loose stool tend toward constipation." So it sounds like it was already near that spectrum. To me, the thing that got things really moving was the prebiotic more than the IgG.

Kiran Krishnan:

Yeah.

Michael Roesslein:

But your answer, I think, speaks to that. We just got a couple more. Won't the prebiotic feed yeast?

Kiran Krishnan:

No. Yeast have no capability of breaking down these oligosaccharides. They're not able to do that. Yeasts do really well with sugars, with simple sugars, monosaccharides, disaccharides, they did not do well with complex oligosaccharides. These oligosaccharides are very complex carbohydrates. They've got lots of branches, unique carbon bonds, and they require very specialized enzymes in order to break them. That's why we call them precision prebiotics, because most of the bacteria in your microbiome can't break them down either they really are specialized towards certain groups of bacteria like [faecalim] bacteria. Like akkermansia. So some of those keystone strains, so they will not feed yeast.

Michael Roesslein:

Okay. I have a family member who avoids FODMAPs, can they take the MegaSpore? We've actually had... when MegaSpore, I'll speak for Krian and let him answer. When MegaSpore first kicked off, the way so many people found Rebel Health Tribe and our webinars and the MegaSpore and all of that was because there's all these groups on Facebook, like low FODMAPs and salicylate groups and histamine, low histamine groups, and SIBO groups, and all these various GI related illness groups. And people from those groups would try it and see that they no longer had the issue with FODMAPs or had the issue with salicylates or had the issue with whatever. And all it took was one person posting that. And then that entire group would be on our next webinar.

Kiran Krishnan:

Yeah. Right.

Michael Roesslein:

And so FODMAPs is generally avoided due to suspecting SIBO, I guess, would be the word suspected SIBO. And also some other dysbiotic situations. But the spores definitely wouldn't make that worse.

Kiran Krishnan:

No, if anything they'd improve them. I mean, so one of the big issues with intolerance to FODMAPs is the inability to break down, digest carbohydrates more completely. So you don't get the fermentation of them, which then produces the gas and the bloating and the sensations that you feel in the gut. And one of the roles at the spores plays, is improving the diversification of enzyme activity in the bowel. Exactly which enzyme activities we don't know. That's one of the studies that we're doing where we're trying to quantify the different types of enzymes that they produce. We know that they produce certain proteases. We know that they can produce certain cellulases. But in general, what we see is that there's a help for those who have FODMAP intolerances. It doesn't necessarily mean you're going to take spores and then you can eat a big FODMAP meal and be fine. But in general, we see people having significant digestive improvement who were intolerant of FODMAPs to begin with. It's certainly not going to make anything worse.

Michael Roesslein:

I can't find MegaMucosa on your site. Do you have a link? I can post that in the chat. You said that the Prebiotic got things going. What did you mean? I have a patient with chronic diarrhea and small intestinal inflammation. I just mean that my digestion and elimination got awesome. And there were champion poop stories going around in our Facebook group for about three months when that product first came out, that were pretty graphic. And I don't think, I wouldn't worry if someone with chronic diarrhea could be really to do a dysbiosis that might actually be helped with the Prebiotic. And it's not that it pushes you more towards diarrhea. It just, everybody just commented on how amazing their poops were.

Kiran Krishnan:

Right. And again, that's all because of the increase in short chain fatty acids, and organic acids that are produced in the large bowel and the protective keystone strains, all of which have an impact on bowel movement. If you have a patient with chronic diarrhea, then the best thing is the MegaIgG. I mean, that is a-

Michael Roesslein:

Yeah, that's what it's for.

Kiran Krishnan:

That's what it's for. That's what it's designed for. That's what the studies are based on. It does a lot of other things, but the root, the anchor for what it's used for the medical community, and this is in gastroenterology offices and all that as a prescription product is for chronic diarrhea. But you'll have to get them somewhere around four gram dose would be the key,

Michael Roesslein:

Which is eight of the capsules.

Kiran Krishnan:

Wight of the capsules. Yep. You can do that either once a day or you could split it up and try to do split two grams, get two grams twice a day.

Michael Roesslein:

I have multiple myeloma. Has high IgG, is giving someone IgG then a problem? It doesn't have a systemic effect like that.

Kiran Krishnan:

Right.

Michael Roesslein:

It wouldn't further raise their systemic IgG. Is the FOS and MegaPrebiotics different than the usual FOS or inulin that cannot be tolerated. It often tolerated so well when one has SIBO, according to the Wikipedia article, FOS can also feed pathogenic bacteria. I've seen most people with SIBO do okay on the prebiotic. I have seen some need to go really low and slow with it. Do you want to speak to that?

Kiran Krishnan:

Yeah. The FOS is different. So, fructooligosaccharides is a big general category of oligosaccharides. It just means it's an oligosaccharide derived from fruits from sugar containing fruits. And so fructooligosaccharides versus galactooligosaccharide versus, the xylooligosaccharides they are bigger. Terms that represent big categories of oligosaccharides. So not all fructooligosaccharides are the same. There are different FOS is with different structures and different functions. What we really like about this FOS, which comes from golden green kiwis, it comes from New Zealand and it has just an extensive amount of studies on its ability to specifically increase keystone strains in the large bowel. Which speaks to its specificity for those keystone trains in the large bowel.

That's why we started working with this particular fructooligosaccharides. It really speaks to that specificity that it has. And that is different than the response you get from other fructooligosaccharides. Inulin has a bunch of tolerance issues, and inulin also has more of a sweet taste to it. So it's got different structure to it, inulin can be used both as a sweetener and a prebiotic. These fructooligosaccharides have much different sweet perception to it because the structures are different. The sugars that make it up are different. So they have different functions in the body.

Michael Roesslein:

Okay. I think we have time for one more and then we're going to hit the time limit to two hours. I think we've gone three hours once before, but we will..

Kiran Krishnan:

Yeah. Oh, I'm surprised it's even at two hours, wow. Time flies.

Michael Roesslein:

I'm trying to see, histamine intolerance. So there was a histamine group where we had a lot of people come back with just the MegaSpore. So in addition to MegaSpore and your IgG would love to know any suggestions we can do for histamine intolerance probably caused by antibiotics for eczema wounds that became infected with staph A, we have been using your IgG and MegaSpore for one year already. Thank you. Are there any other suggestions?

Kiran Krishnan:

Yeah. I would go high dose of the HU58. That could make a measurable difference. That's a standalone product, which is a strain that's in MegaSpore obviously, but in MegaSpore, it's at about a 2 billion dose per two caps. But in the HU58 product, it's at 5 billion per cap. You actually could go two caps a day, if you wish and try that. That might be enough to make the difference for you.

Michael Roesslein:

And I'm just typing that out. The HU58's a high dose bacillus subtilis, generally used for infections, overgrowths, things like that. It's more of a powerful shot. In the shop. 10 billion CFU versus the 2 billion in the MegaSpore.

Kiran Krishnan:

Yeah.

Michael Roesslein:

Cool. I just typed it there, for someone said high dose of what for his... It's possible that would be helpful at we can't make blanket diagnosis and prescriptions and things like that. I cannot have dairy. Why does the MegaPrebiotics have dairy and what can I do instead?

Kiran Krishnan:

Yeah. So it doesn't have dairy in it. It has the galactooligosaccharides, which comes from dairy. But remember dairy that the thing that causes intolerance with people and dairy are the proteins in the dairy, right? Those, the caseins and so on. There's none of that in the product. These are the carbohydrates oligosaccharides that are purified from that source. So there isn't any dairy in the product. Now because of allergen labeling laws. If a product derived from a potential allergen source, you have to put that warning on the label. So people often misinterpret that there's actual dairy in the product. So there isn't, if you are anaphylactic to dairy we say, okay, stay away from it. You just don't know how you're going to respond and it's too risky. But if you are dairy intolerant, you should not have any issues with it then.

Michael Roesslein:

We haven't had a single person that has dairy issues complain. Mira's got dairy issues. It's dairy free entirely and she drinks it every day and we've never had an issue. I get congested when I eat dairy, even though cheese is my favorite food group.

Kiran Krishnan:

Yes.

Michael Roesslein:

And every time I eat it, I end up like walking away all congested. And it's never done that before. The link is right above there, it'll also be in the email. Like I said, the code is ML5OFF. It's five bucks off per product added to the cart. That is probably the biggest sale that we've ever done. So I wanted to celebrate this. We also in the blog, for people who don't know, we have fully comprehensive posts on almost every one of the products. We don't think we have them all yet, but most of them, if you go to RebelHealthTribe.com and go to the blog, there are at least four or five posts. As you scroll through the blog articles that are fully comprehensive, detailed posts on each product. The research behind it, the

ingredients, the best uses the contraindications, the using with other products, all of that stuff, it's all there. So head to the blog, you'll be able to find that. I think that's all of the product questions. Terrible spring allergies allergies, for me personally, I would just go with that trio and possibly some MegaIgG.

And I think that is it for today. So I think, let me see. There's one more message. Great job. Thank you. Awesome. Can I order more than one of HU58 and get percent off for each bottle? It will be $5 off per product. So per bottle of anything. So yes, that's how it's set up. Let me know if that doesn't work. Because it's the first time we've ever tried to set that up and it's excluding products that are not Microbiome Labs. And backend coupon tech things don't always go exactly how they're supposed to. So let me know if something gets screwy and we'll have somebody fix it for you, but it should work just with that. And we will get this recorded. There is no three months container of MegaSpore. It's just a 60 capsule bottle.

Kiran Krishnan:

Oh, we do have the big one now.

Michael Roesslein:

Oh.

Kiran Krishnan:

The 90 day version of it. Yeah.

Michael Roesslein:

Well, geez. I didn't know about that. So we will have to get some of those. Write that down.

Kiran Krishnan:

Yeah. We launched it a couple of months ago and it's become really popular now.

Michael Roesslein:

Get big MegaSpore. That's the note I'm writing to myself here. And then I'm also going to turn in what you did at the end here with your slide and everything that you went into with words that weren't on the slide. I'm going to watch this, use your slides, use your words. And I'm going to create a little PDF ebook.

Kiran Krishnan:

Awesome.

Michael Roesslein:

That I'm going to send out to everybody with recommendations for optimizing the gut, immune, microbiome axis.

Kiran Krishnan:

Love it.

Michael Roesslein:

And I'll share that with you. You can put it wherever, if it's up to your standards or you can look at it and say, "This thing sucks. You need to change it to this." So let me know. Okay. So I'm going to put an email there. Jason@RebelHealthTribe.com. Send him an email with the issue you're having with the tech stuff. Jason@RebelHealthTribe.com. I'll reach out to him now and let him know that somebody is having that issue. We'll try to get that fixed really quick. Thanks to everyone. So this will probably be up Friday afternoon, I just got to wait on the transcript. So it'll probably be Friday. We'll put the two parts together. So you'll have three and a half hours of microbiome, gut immune craziness to dive into and I'll create the PDF. I will not have that done by Friday. Please give me email of Doctor Danenberg. Doctor Al, I will have on a video very soon, but if you google Doctor Al Danenberg, he will come up. He will come up. He's very well published on the internet. And let me see.

He might even still be on here. Doctor Al Danenberg. His website is DrDanenberg.com. He's a nutritional periodontist. He's a functional medicine dentist.

Kiran Krishnan:

You should spell the Danenerg.

Michael Roesslein:

Yeah. D-A-N-E-N-B-E-R-G. The code is ML5OFF. All upper case. I don't think it matters. So all one word ML5OFF it'll all go out in an email. We're going to run the sale through probably Saturday. So I'll send it out on Friday and we'll let everybody know. Kiran, thank you so much.

Kiran Krishnan:

Of course.

Michael Roesslein:

That was a record for questions in one webinar, we did like 36 started with 32. We did 36 because about another 20 piled in as we were answering them. And I've got a small handful of them. I might send you in an email to try to [crosstalk].

Kiran Krishnan:

Okay. That sounds good. And we of course did our tangents that we went on, which hopefully we were helpful for people, but this is always fun to be able to do this.

Michael Roesslein:

Yeah.

Kiran Krishnan:

And thank you for everyone for hanging in there. Hanging with us.

Michael Roesslein:

We had very little drop off. The highest, it was... Yeah, it was, people started to drop off like 10 minutes ago. So most people met the two hours and stayed and hung out, which I've watched a lot of things about hosting the best webinars and doing the best webinars. Rule number one, never go over 60 minutes ever.

Kiran Krishnan:

Right.

Michael Roesslein:

That's what they say because no one will watch anything for more than 60 minutes. And rule number two is if you have Q and A, don't put the Q and A on recordings ever, because it will incentivize the people to stay to the end of the webinar, if you don't include the Q and A on the recordings so that they can get the Q and A, as an incentive teaser to get people to actually stay. We put everything on the recordings and just did a 90 minute standalone Q and A.

Kiran Krishnan:

Right. Or two hours, right?

Michael Roesslein:

Two hours. Yeah. Screw the experts and...

Kiran Krishnan:

So much do they know.

Michael Roesslein:

Maybe we should be the webinar experts.

Kiran Krishnan:

All about the content.

Michael Roesslein:

Thanks guys. Thanks everyone.

Kiran Krishnan:

Thank you. Have a good night.

Michael Roesslein:

Yeah. All right, everyone have a good night. Thank you.

PART 4 OF 4 ENDS [02:02:03]