the Solari Report

Future Science Series: Parasite Biofilms-Origin of Chronic Diseases with Joshua Leisk

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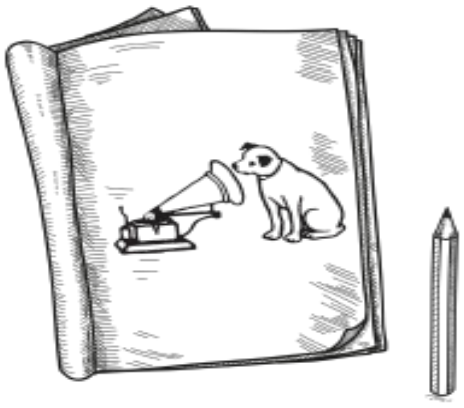
Transcript



Ulrike Granögger: Welcome to our new *Solari Future Science Report.* I have a wonderful guest today who I met in an Australian forum discussing health worldwide. We have had a crisis over the past five years, and in many ways, we're still working through coming to terms with it. My guest today is Joshua Leisk. He's from Sydney, and he has probably discovered a model or a solution to many of the chronic diseases. I have tentatively found the title, but I'm not sure if this will remain the title: Microbiomic Origin of Chronic Diseases.

Joshua will speak about the microbiome and alcohol-fermenting bacteria or organisms in our bodies that may turn out to be the main culprit in many chronic diseases. I don't want to say too much because my approach is very amateurish. You will see that Joshua has an enormous wealth of knowledge. This will be a challenging presentation, but also inviting both for the normal viewer and reader and a person who is interested in maintaining their own health and their family's health, but also for more professional viewers, doctors, and microbiologists, who might find some of the contexts that Joshua is presenting very, very interesting.

Welcome, Joshua Leisk. Thank you so much for taking the time today so we can talk together about a topic that I am also very interested in. Much is being spoken about parasites and microbiomes, et cetera. You have your own understanding of this, and I think it's very, very valid that our viewers and readers know more about it. We will talk about you and introduce you personally perhaps later. Only in the show notes, you will see his website. Please go to his website, bornfree.life. com. What a wonderful title that is. We will talk to you later. Perhaps you want to describe to us the specifics of your model. You have also brought some material to show us so we can follow along.

Joshua Leisk: Absolutely. Thank you very much, Ulrike. It's a pleasure to be here. What I'm going to present is going to be a very high-level conceptual overview of the disease model and some of the concepts behind it. I won't get too far into the 'weeds' where possible. Of course, I'd be happy to discuss any of the more technical concepts, either during this interview or another time, but there are also other videos already available where people can dive into the more nuanced aspects of it. Of course, the diagrams and things related to the disease model are freely available on the website, including some interactive tools to help you understand some of the logic and things.

To start, I'm going to touch on some important concepts. Where I'm going to begin is talking about two blind spots in the immune system. One of those blind spots is important with regards to microorganisms and the way that the immune system can deal with those microorganisms and, in some cases, has difficulties in actually suppressing both their growth, and completely eliminating them from the body. To understand the challenge, we need to first talk about biofilms.

Many people are familiar with biofilms when they think of the oral plaques in their mouth, or they might get a slimy tongue, or if you've ever cleaned out the drain in your sink and found slime there. If you have a boat and there's slime on the hull of the boat that needs to be slipped or cleaned off now and again, that biofilm is a container for microorganisms.

Granögger: They produce it themselves, is it?

Leisk: That's right. The different microorganisms produce biofilms under different conditions. Every different microorganism produces slightly different biofilms. This is a common feature that the majority of them have for protection. Essentially, when they're inside their biofilm, they have protection against immune surveillance, for example. I'll come back to that on a later slide. I'd probably just touch on the fact that we have different microbiomes; we do not just have gut microbiomes. Everybody's familiar with that these days, and there's a nasal microbiome, as well as the oral microbiome. You have a microbiome in the mammary glands and lactiferous ducts.

According to the latest literature, the brain even has a microbiome, and of course, the sex organs and other mucosal tissues; even Kidneys. Anywhere where there's a wet interface is an opportunistic vector for biofilm attachment. Of course, if the microorganisms have gotten into the body, then you can find the microorganisms creating biofilms in the circulation, but also attaching them to potentially arterial walls if there's inflammation, things like breast implants, and other tissue. If you've had surgery, there may be biofilm just around the opening for the surgery, for example, or anywhere where the surgery was carried out.

Granögger: Question: Is Biofilm the same as a mucosal lining?

Leisk: No, very different. The body produces its own mucin, so it has a mucosal lining. Then, the microorganisms create their own extracellular matrix

or a cocoon around them, which is a lipoprotein with a mineral scaffolding. There's a combination of different metals, and there might be calcium, magnesium, iron, and different things in the structure, as well as glycerol and various lipoproteins, which then make up the film itself. It needs to have a slightly filmy, spongy, flexible structure, but there must also be some rigidity for their protection.

Granögger: Interesting.

Leisk: Yes, it is very, very interesting. The chief benefit to the microbes is the fact that it protects them. If they're inside our body in particular, it protects them from our immune cells being able to detect that they're present, which means that the immune system can only suppress their growth. I'll come back to that in a moment. Let me just go into that in the life cycle context. I'll go back to that in a second. In the life cycle, what we have is the surface adhesion. The motile microbes can stick to a surface area.

Granögger: The microbes always come from outside?

Leisk: Generally come from outside but can also come from an existing biofilm. If we have a look at this life cycle, where we get to maturation and dispersion from an existing biofilm, there's an opportunity at that point for the hosts to step in and filter the next cycle again. The immune system can step in at this point and actually identify that those microorganisms are there. However, the immune system is not always capable of doing that because it can be busy with other pathogens, or there can be other reasons the immune system is dysregulated. Otherwise, the person is immunocompromised.

Going back a bit, it's important to understand that when we look at the mucosal tissue, there is a significant surface area available for these biofilms to form. Roughly 125 square meters, and there's not a significant difference between the male and the female physiology concerning the opportunity for these biofilms to grow. The largest area is the respiratory tract, and the second largest area is the gastrointestinal tract. Then, minor mucosal surface areas are listed in sex organs and other tissues.

It is really interesting. As far as an opportunity then for different infections, if you could consider that every single time that you've been infected with these microorganisms, there's a possibility that they've set up a biofilm reservoir in the

tissue where you're infected, or if they're translocated to different tissues, then they can set up a biofilm reservoir in other tissues.

Granögger: We don't notice that, do we?

Leisk: We don't notice that because, at that point, they're not triggering an immune response inside the biofilm. It's only when they come out of the biofilm that the immune system mounts an attack, which can be the source of inflammation. However, while they're inside the biofilm, they still absorb nutrients, and they still produce metabolites, some of which are very toxic. There can be endotoxins and mycotoxins, depending if we're talking about bacteria or fungi.

Granögger: These mycotoxins go into the body. They don't stay in that film.

Leisk: Correct, unless of course, you have probiotic species nearby the opportunistic pathogens. Some of those probiotic species have a function where they can break down the biofilms, but they can also pick up the toxins and metabolize them into something that's not toxic. However, if we don't have those probiotic species, the load is back on the host to metabolize those toxins.

Granögger: What does it mean when you say that they extract nutrients? Are they taking what we eat?

Leisk: That's right. They take the nutrients that we eat, but also that are circulating because our body will produce many things or categorize it from tissues. They will benefit from the nutrients whether or not they are something that we've consumed or something we're producing.

Granögger: They are feeding themselves what should feed our cells and our organs.

Leisk: That's right. Look, the probiotic, the helpful species do the same thing, but the major difference is that they're not producing a toxic metabolite. What they're producing is more helpful for the host. That said, you could consider the symbiotic relationship of all these microorganisms, including even some of these opportunistic pathogens, in the context of some of the endotoxins and the other toxins, which I see as elevated.

Commonly in people with chronic diseases, it's interesting that the metabolites

produced have an antidepressant effect. Things like acetaldehyde, hydrogen sulfide, or p-Cresol all have an effect where they inhibit the degradation of neurotransmitters, for example. However, that can become a maladaptive response under certain circumstances.

Granögger: Let's also break this down for the layperson. What you're saying is very interesting. It's the effect of the side products, of the digestion of these biofilms or the microbes in the biofilms, their toxins, so to speak, can be that neurotransmitters are more slowly broken down. That means there is a-- what did you say?

Leisk: An antidepressant-like effect.

Granögger: Antidepressant effect. They're more exhilarated. They feel stronger, perhaps.

Leisk: I think it's more a compensation. If we consider the context of the microbiome having a symbiotic relationship with the host, we have no idea how a lot of that signaling works at this point. I think it's something that will be a topic of future study for the next few decades, and I understand how to do it. If, say, for example, somebody is suffering from trauma or depression from various different life events that might have occurred, it's possible that the microbiome can step in to try to restore the homeostasis by slowing down the degradation of the neurotransmitters to try and keep the levels of dopamine and serotonin and norepinephrine and epinephrine at a more functional level.

I think, at a certain point, that becomes maladaptive for various reasons.

Granögger: I see. It can have a positive effect as our microorganisms do have a positive effect, but it depends on the amount and the timing.

Leisk: That's right. The dose creates the poison, as it were, and the frequency. It's an interesting situation to ponder as a chicken and egg situation. Is it that we have small amounts of these microorganisms, and then some sort of traumatic event causes them to bloom? Then you might have some kind of, I suppose, a slippery slope being created depending on what's happening, and the problem just gets worse and worse and worse over time. The opposite is also true.

You could have the infection, you could have various reasons things are overgrown, and then that leads to trauma and depression and various things, which can just form another loop or a cycle, too. I think it's important to communicate that most of the modeling basically describes loops and cycles rather than straight lines, other than the fact that we have these two blind spots in the immune system, which ultimately allow all of this to play out.

Granögger: Define the two blind spots once again, please.

Leisk: The first blind spot is the microbial biofilms, allowing them to evade immune surveillance. The other is one that I'm going to be touching on a little later, which concerns herpes viruses. There are nine herpes viruses. Everyone thinks of HSV-1 and 2, herpes simplex 1 and 2, which might be oral and genital lesions. However, we also find it in the brain later in life. There's varicella, which is known for chickenpox, and later as shingles.

You have Epstein-Barr virus or the kissing disease, and that starts generally in the throat and progresses into all kinds of places. There's Cytomegalovirus, which we typically associate with stomach cancer. There's HHV-6A and HHV-6B. They are very, very interesting in that they like to infect T cells, whereas Epstein-Barr virus likes to infect B cells. That tends to be found later in life in the brainstem, and in certain other places. HHV-7, we seem to find a lot more in European patients than across the rest of the world. KSHV is the ninth virus there.

They all have a latent analytic phase in their lifecycle. When they're latent, they're still present; they're still producing small amounts of certain antigens. In the case of the Epstein-Barr virus, for example, it produces 10 different proteins in its latent phase. In that latent phase, the immune system just ignores it. It's not able to do anything effectively about it. It can't see that it's there or chooses not to do anything with it. In the lytic phase, Epstein-Barr virus will produce, I think at this point, we've measured 99 antigens.

At that point, the immune system can see it and will attack the cells that are lytic. Unfortunately, though, in the process of producing all of these antigens and replicating them, by the time the immune system kills the original lytic cell, we may have 10 more infected cells. The progression is typically only one direction without specific interventions. Other than the herpes viruses, there's also human papillomavirus, HPV, which we associate with oral and cervical

cancer and probably a few others in time, and of course, the human immunodeficiency virus. HIV also has a latent lytic phase.

They're all lipid envelope viruses, which essentially have this stealth ability to exist inside the body.

Granögger: The microorganisms you're showing remain hidden; that's the blind spot. The blind spot is in the immune system.

Leisk: That's right. The blind spot in this case, in this example, is what we're seeing on the left; there is a layer of tissue. It could be skin or some other epithelial tissue. Between the cells, you may have a group of bacteria, fungi, or other parasites and things, and they might be obscured from any immune checkpoints by being covered in biofilm. There's a limited opportunity to deal with those. Of course, those layers of the tissue are gradually turned over in time. Little bit by little bit, the body can deal with a layer at a time.

There are still, for want of a better term, sleeper cells inside of those tissues because the actual process that created them still exists. However, there are things we can do to break open the biofilms, and at which point, in the middle image, we can see that their immune system sees it and mounts an immune response. We can see some discoloration of the tissue. Then, a couple of weeks later, we can see a perfectly clean tissue on the right-hand image.

Granögger: That's a fungus, is it?

Leisk: Yes. That was a candida and a handful of others in this particular case.

Granögger: Enormous.

Leisk: Indeed.

Granögger: You will talk about how to do this.

Leisk: Yes, absolutely. Look, the protocol we're using there is much to read. We won't have the opportunity to go through it in fine detail now unless we have a lot of time. There are 160 odd pages and database tables and things on the website, which walks through the best ways we know to reverse this trajectory. It's important to understand the problem before we talk about the solution. Some of this problem is intergenerational.

In the context of your microbiome, your microbiome is something you inherited from your mother and, to a lesser extent, the other inhabitants of your household. You get a horizontal transfer from anybody you're close to, including animals and everybody else.

If, for example, your mother has had different microbiome issues, gut problems, or mental health issues, there might be various ginny issues. You end up inheriting that microbiome, even if everything goes well, if it's vaginal birth if the baby is breastfed if there are no dramas, no emergencies during birth. There's no antibiotic use or anything in the very early stages, then the seeded microbiome still has a limited diversity if the mother's microbiome is missing any of those species. Progressively, say, in the developing world, this comes down to the hygiene hypothesis and various other interesting ideas. What we see is that the microbiome diversity of somebody who lives in Western civilizations looks incredibly different from that of a Kalahari Bushman.

Somebody that still eats very, I'm going to say, healthily yet primitively would be very similar to a wild animal where essentially they're ripping plants directly out of the ground. They're not necessarily washing them, they're not necessarily cooking them, they're consuming the prebiotics, which is the plants, as well as the matching probiotics, which is, of course, the microorganisms that were on those plants. Those have an opportunity to maintain the microbiome diversity.

However, there are challenges because when those microorganisms are seeded into the host, stomach acid and various other layers of protection will inhibit the viability of any of the cells coming in through the mouth. The way that nature works around this is to package them up in a really useful vehicle, in which case we're talking about breast milk, which also has some other immune functions. It's not just a nutritional benefit, but it is also beneficial for the child regarding the macro and the micronutrients. There are also several immunological benefits.

Essentially, you've got enzymes like xanthine oxidase, which is one we'll talk about a bit later. With interferon-gamma, we have the lactoferrin, we have the IgG and the colostrum, and of course, because of the calcium phosphate and various other things in the milk, we have a bit of a buffer for the stomach acid, which is then potentially helping the bifidobacterium and lactobacillus and various other species seed the gut. Of course, some of those species will get into the nose, mouth, and other places, and they'll eventually translocate into the

other organs.

It'll be very interesting to look at that later. Even during pregnancy and breastfeeding, the relationship that our body has with a number of the probiotic species is such that the body allows things like the lactobacillus species and others to hitch a ride through the bloodstream up to the mammary glands and lactiferous ducts without interfering. The immune system doesn't step in. There is definitely a symbiotic relationship and expectation that some of these species will exist.

Granögger: You are saying that breastfeeding is necessary for a healthy microbiome to grow in the child, in the baby, or it's one of the best ways to do that, especially if there's a generation of mothers before that that have had a whole variety of healthy microbiomes. I think we will be touching upon this also because I am immediately thinking of breastfeeding after the coronavirus pandemic, both the natural virus or maybe artificial virus, and the vaccine, and how this apparently also goes through the breast milk, and how this seems to be impacting on the next generation of children and their microbiome.

Leisk: Sabine Hazan has done some good work on the loss and destruction of the bifidobacterium. She's mostly been focused on the bifido loss after vaccines. The same thing happens during or after COVID and the same thing we've observed in myalgic encephalomyelitis and chronic fatigue syndrome for decades before COVID. It's not a new thing, but there certainly are specific features over the last few years that have accelerated the loss of those species.

Granögger: Would you say that childhood vaccines, would they be accelerators, could they be accelerators, and how would they impact the forming of a healthy microbiome?

Leisk: It's an interesting topic. There are some interesting papers on this. There was one that I tweeted about recently. It was a longitudinal study. It was very interesting. They followed the life of children from a very early age to adulthood. What they found is that autism spectrum disorders were essentially a predictor of chronic fatigue syndrome in teenagers. Essentially, when puberty hits, then the immune system goes through various changes because of the hormonal changes and various other things that may happen.

In the context of the model, it's quite interesting. If we consider that we have

this issue with the biofilms, then what can happen is that there are several different variables in play. Let's say that you have little Johnny or little Janie in the crib, and everything might have gone really well. They might have had a good birth, they might be being breastfed, but they might then be exposed to some kind of mold or staph or strep or various combinations of that in the nose and sinuses and things like that.

From an adult's or the parent's perspective, you just think the baby has a snotty nose and probably don't think too much about it. It'll normally go away, and everything will come out good.

In the context of setting up these biofilm reservoirs, what's going to happen progressively over time is that every single time that the immune system is distracted with a new antigen from any source, it allows the biofilms to expand their surface area. I might just jump back a few slides to this and highlight that again. In the context of temporarily unavailable immune checkpoint, surface area expansion is allowed.

Granögger: The new antigen would be, for example, the idea that the vaccine gives to the baby to train the immune system to quite that antigen, but then at the same time, it's distracted, as you say

Leisk: That's right.

The problem is not necessarily any individual vaccine as such. What we're talking about, though, is just if we consider it either as an antigen regardless of the source, it's the fact that you have an otherwise healthy baby being exposed to what might be 70 to 130 pseudo infections in the first 10 years of life on top of all of the other infections that they're going to go through because of just the household exposure or daycare, playgroup, all those kinds of things as well.

In the context of the existing co-infections, nobody is looking at that. We don't necessarily have to 'throw out the baby with the bath water'. What we do need to do is say, "Okay. Well, maybe before we introduce any new antigens into the system, we should be doing something to prevent the accumulation of these biofilms so that we can train the immune system against really bad pathogens, but not actually allow this cascade to develop."

Granögger: Fascinating.

Leisk: That said, I think there may also be discussions around how many antigens are delivered to a system simultaneously. I think that a single antigen is probably something that can be done carefully. I think the concept of introducing multiple antigens simultaneously is not something we'd normally see in nature.

Granögger: Yes, in nature.

Leisk: Particularly, there are certain challenges with how the immune system responds to different types of antigens, which might create a conflict or a clash. I will come back to that a little later in this presentation and talk about the differences in some of these antigens. Ultimately, there's essentially a logical gate, a flip-flop in how the innate immune response deals with different types of antigens. Ultimately, this can be considered for future planning and development with how we could most safely achieve the goals we want to achieve without creating new problems in the process.

Granögger: Excellent.

Leisk: Maybe diving into the 'weeds' here. Some of the microorganisms that I've been focusing on as part of my research are the ones that produce alcohol and/or acetaldehyde. Alcohol, as we all know from alcoholic beverages. Acetaldehyde is the next metabolite from alcohol. It has a distinct toxicity in several different pathways. It can affect the energy metabolism quite dramatically.

It can affect methylation. It can affect the inhibition of the neurotransmitters, as I was talking about before, which could have a potentially positive effect. It inhibits the histamine degradation pathway, and at the same time, it triggers histamine release from the mast cells. That's another secondary disease that is well-known to come along with Long COVID or jab injury, or ME/CFS. Everyone is well aware of mast cell activation syndrome.

In the context of microbiome dysbiosis and this acetaldehyde production, it's a very large player in being able to produce this mast cell activation syndrome. Essentially, acetaldehyde can cause lesions and inflammation in the epithelium, including the gut barrier and the other mucosal tissues. Of course, that can then allow other foreign proteins to get inside the body, triggering the mast cells, but then, of course, the acetaldehyde itself can trigger that as well.

Granögger: That acetaldehyde is a product of the digestion, so it's a big system of these biofilms?

Leisk: No. That's actually a product that the microorganisms themselves will produce. Some of the microorganisms will produce alcohol, sometimes at quite a prolific rate. We see this as gut fermentation syndrome or auto-brewery syndrome. Other microorganisms can rapidly convert that alcohol into acetaldehyde; sometimes, both happen in one microorganism.

Granögger: Just for the audience, auto-brewery means that our gut or our system, our body produces its own alcohol.

Leisk: That's right.

Granögger: We do this anyway, I think, when carbohydrates are being digested.

Leisk: Small amounts.

Granögger: In small amounts.

Leisk: The acetaldehyde is a normal metabolite in energy metabolism, but again, it's the amount that's being created and the effect it has on other parts of the metabolism.

Granögger: With an infestment from these microorganisms, we constantly can be drunk? Is that so?

Leisk: Normally, we don't get the fun part. Normally, it's more like chronic hangover syndrome without actually all of the enjoyable parts. What's interesting is that there seems to be a correlation between people actually becoming alcoholics as a mechanism to deal with the tolerance and dependence between meals and the acetaldehyde production that's being generated. I think there is a significant influence on what we call ADHD from this part of the cascade.

I could go into that maybe after the presentation and go into some of the 'weeds' of that, but yes, there's a very interesting part of the cascade where the body actually produces morphine and gamma-hydroxybutyrate, which are both interesting from a narcotics and pain relief and various other angles, but the chronic production of that can lead to tolerance/dependence and symptoms of

withdrawal when you actually start resolving some of these issues. It creates another challenge to the recovery process.

Granögger: Fascinating; alcohol, acetaldehyde.

Leisk: Alcohol to acetaldehyde. We went into some of those areas. I'll skip through, maybe just to the last sentence, which is interesting. When I was putting together the database of all these species, I noticed that all the microorganisms we associate with cancers, dandruff, and acne are all acetaldehyde producers and even cancers. We focus on things like H. pylori, for example. We know that that's associated with stomach cancer, and that's another prolific acetaldehyde producer.

Granögger: When you speak of cancer and alcohol fermentation, I'm reminded of an idea of the production of cancer. It goes back to Warburg and Hendrik Kramer and others, that in cancer, the cell is no longer producing its energy from the ATP but from fermentation`. Is that also playing into this idea?

Leisk: Kind of. Any cell can use Warburg metabolism. I'll try and keep this light. We will touch on this part of the metabolism later in the presentation. Essentially, taking a step back and looking at the same metabolism differently. If we were to look at muscles and if we look at what we call lactic acid threshold or anaerobic glycolysis threshold, what can happen is that if, for various reasons, the cells aren't able to get energy from glucose metabolism, glycolysis, or from fatty acid oxidation, that's transporting fatty acids, whether that's coming from lipolysis from our own fat cells or whether that's coming from dietary fats, then at that point, the cell has to keep going.

Certain enzymes act as metabolic sensors. Those enzymes, if they're inhibited for various reasons, create a cascade, which then triggers this lactic acid metabolism. When we see lactic acid metabolism and also a thing called glutaminolysis, which is where the cell takes glutamine, converts that into glutamate, and then puts it into the citric acid cycle at alpha-ketoglutarate, and potentially there are some other mechanisms there, then yes, we think of that as a Warburg metabolism.

The cell uses lactic acid to produce some of the ATP, and it's also trying to keep the cycle going, or the citric acid cycle going, using glutamine and a number of other sources by a thing called anaplerosis. That's where other parts of the citric

acid cycle are fed or supported by other pathways. That might be something like the urea cycle or catabolizing GABA, so the GABA shunt can catabolize tyrosine.

All of these different amino acids can enter the cell. Then, of course, we have another system called ketosis, where the liver can ultimately produce these ketones, which can also get into the citric acid cycle via a different route. Essentially, the Warburg metabolism is a sign of the cell being stressed, or metabolically stressed, usually by its own behavior. Essentially, it's pushing, pushing, pushing, build, build, build, build, build, to the point that it generates all this oxidative stress.

Then, when it can't keep up with that, the engine overheats, and something must take over to keep the cell alive, and that's when it starts using lactic acid metabolism and glutaminolysis, or Warburg metabolism.

Granögger: The alcohol produced by the biofilm inhabitors would also contribute, or it would accelerate that.

Leisk: The acetaldehyde can contribute to oxidative stress. Talking about this issue with the blind spots in the immune system, if you want to touch on it briefly, I do have a few thoughts. This is purely hypothetical, although some interesting studies seem to be validating different aspects of this. My view on cancer is: Forget the label for a minute; let's not call it cancer. Let's just look at the features of the cells. One of the things we see is that the cells are immortalized. What that means is they don't have a working apoptosis pathway.

It means that under the conditions that should normally trigger the cell to kill itself, it doesn't; it stays alive. Where we normally see that is if you have a cell that's infected with one of those latent herpes viruses, or HPV, or HIV. That's because one of the first things those viruses do, and other viruses do it too, but they don't have the latent lytic life cycle, so therefore, they don't evade the immune surveillance; the immune system can see it and clean the cells up. In the context of having an infected cell, which has its apoptosis pathway inhibited, and evades immune surveillance, those herpes viruses are particularly well-suited for being one of the mechanisms actually to create that.

Granögger: Again, fascinating.

Leisk: That's it. That doesn't create cancer; you now just have an infected latent cell. That infected latent cell with a disabled apoptosis pathway might then be exposed to other pathogens. That could be fungi, bacteria, parasites. Some of those can be intracellular. We can see lateral gene transfer from those microorganisms into this immortalized cell. If you then consider what you have there; you have this hybrid cell, a hybrid human cell already infected with one or more viruses, which now becomes infected effectively with this single-celled microorganism.

Then, the DNA or the code is updated to effectively amalgamate our cells and at least two other different pathogens. If you start looking at the cell's behavior, it can now behave more like the microorganism potentially. It still wants to go out and build and do everything, perhaps from the viral side. I guess it's really going to depend on the context, but the DNA, which has come from the other microorganism, then is going to alter the behavior, the programming of the cell, and you might see the growth patterns and things more mimic the single-celled organism.

You might look at a glioblastoma or something and say, "Wow, that looks really interesting under the MRI. It actually looks like some sort of fungal growth pattern." Where I've seen some interesting data on this was a study in 2022 that took a range of different cancer cells, and did an untargeted metagenomic test on those cells. They found that 35 different cancer cells had fungal DNA in them.

Granögger: It directly has to do with your model of these biofilms creating chronic diseases and perhaps even cancer. That's what you're saying.

Leisk: If you consider, and we'll get further into this, the more progressively along this cascade you are, the less capable the immune system is. Progressively, what it can do is becoming more and more limited because it's now being distracted by this growing pool of these different pathogens. We're going to associate that with aging and all the symptoms and things that we see that are associated with aging, but also in somebody that's had long COVID, VAX injury, ME/CFS that are immunocompromised from a very early age. We see the same thing if we start looking at other mucosal diseases.

Let's look at endometriosis, polycystic ovary syndrome, asthma, chronic obstructive pulmonary disorder, or various other diseases; more often than not,

they get labeled as an autoimmune disease, which bothers me to a very large extent, but if we look further into it, we'll see that we find actual pathogens in those tissues.

Granögger: In a sense, you're saying that with the cascade of these growing biofilms that are overlooked in their germination state by the immune system and then can proliferate when the immune system is distracted by something that a virus or other infection or by any toxin that it has to fight, by this cascade of this growing more and more in our system, this leads ultimately to being eaten up by the microbiome for aging.

Leisk: Interestingly, we see, for example, Hypermobile Ehlers-Danlos Syndrome is another thing, hEDS, which is commonly associated with long COVID vaccines during ME/CFS. Without getting too far into the 'weeds' again, the same enzymes that we talk about, these metabolic sensors, we must go back to that at some point, which triggers the anaerobic glycolysis, the lactic acid metabolism; they also have a function in the collagen synthesis and degradation pathway. They're wrapped up in some of these issues with collagen synthesis.

Granögger: What is an issue of collagen synthesis for the layperson as an example?

Leisk: The inability to maintain the elasticity of skin and other tissue. It could be wrinkles forming; an inability to maintain and repair damaged tissue.

Granögger: Blood vessel flexibility.

Leisk: Yes. Endothelial damage and things, too, but don't forget that we have the biofilms directly attached to arterial walls and things. There is a localized effect, potentially a more concentrated effect of the toxins around the biofilms themselves, but then there are also the systemic effects.

Granögger: The collagen, you wanted to say.

Leisk: The collagen is not just that hypoxia sensor or oxidative stress sensor. There are these prolyl hydroxylases, which I will show you later. Don't forget those terms. The mineral deficiency cascades, which are created as part of this model, directly affect several different pathways, which is measurable. If we look at the collagen synthesis pathway, what that is is it takes four amino acids, and

that's going to be proline and glycine, and two random amino acids, depending on the strand that it's going to make or the fiber.

Then, the pathway that it goes through requires several different metals. That might be copper, magnesium, and bromine for type IV collagen, manganese in different places, and zinc. Then, because of the prolyl hydroxylases, it needs certain things to be working, like silicon, iron, and again, zinc, and of course, the citric acid cycle itself, which has several things there.

When we get to the point of looking at the data, we're not looking for one little thing that's gone wrong; we're essentially looking at 'Humpty Dumpty' at the bottom of the wall, and all these different pieces are wrong. The system is trying to right itself, but essentially, it has limited resources and progressively more limited resources, and it is being swamped and insulted by these toxins. By removing the burdens and by putting the resources back in, we allow the host; the organism to regain its homeostasis.

Granögger: The resources you need to put back in are, for example, those minerals and metals, right?

Leisk: Yes.

Granögger: The minerals and metals are being consumed by the biofilm?

Leisk: No, actually, it's to benefit the host. What I might do is jump back into the slide show, and we'll go into a bit of that.

Granögger: I probably confused your whole presentation.

Leisk: That's fine. I'll go back to this in a moment. This comes down to inflammatory signaling. It's an interesting thing where certain cytokines, and in this case it's TNF alpha, IL-1 beta, IL-6, IL-10, IL-22, have an effect of elevating a peptide hormone called hepcidin. What hepcidin is generally known for is iron homeostasis. Where you see people with anemia of inflammation or chronic iron deficiency and various symptoms that supposedly relate just to that, what you may find, if you looked at the different parts of the metabolism, is you might find these inflammatory cytokines, elevated hepcidin, and then ultimately the inhibition of two metal transporters.

That's divalent metal transporter 1, DMT1, and ferroportin. Interestingly, while

it's well-known that those transporters can transport iron or ferritin, the literature also states that it can transport a total of 11 metals.

When we see people going to get iron infusions and, "Oh, I can't understand why I've got the iron deficiency," they likely have deficiencies of potentially 10 other metals, but nobody's measuring them, or the way that they're measuring them is not actually detecting the problem. This then has to drag the conversation into a conversation about the methodology because normally, we are testing metals, at least in public health, actually to look at the serum levels.

Even sometimes, if you're lucky enough to have a functional medicine practitioner or naturopath or somebody that's a little more well versed on this, they might look at the red blood cell, the metals, but even that doesn't give you accurate data because the red blood cells don't have mitochondria. Regarding getting that information correctly, the best place appears to be white blood cells, which have mitochondria.

There are a handful of labs worldwide that will be able to measure the levels of these metals inside of those white blood cells and give you an indication of those true deficiencies. Bearing in mind the transporters being inhibited, you might find even an elevation sometimes in the serum of certain metals. Then, you look inside the white blood cells or the other cells and find a significant deficiency.

Granögger: Yes, because they're not transported; they're just floating around.

Leisk: That's it. In a chronic inflammatory situation, the data that's being captured is basically not correct. It's like trying to understand how much water is in the bathtub by measuring how much water is in the pipes and the walls. It just doesn't make any sense. When we look in white blood cells, for example, we find significant deficiencies of a number of different metals, and it will be different in every person. Certain things are going to be common, but the diet and different things will influence the levels of deficiency of different metals.

Also, things like the levels of the seed aldehyde. Anything that we see or that we understand from chronic alcoholism also applies to this cascade. For example, we see thiamine deficiency, vitamin V1 deficiency in the seed aldehyde, or from alcoholism. Of course, we see a zinc deficiency being created as well, and we see a vitamin C deficiency being created. In this case, I believe the person was

taking vitamin C, which is why it didn't show up in this test. What we do see is a fairly distinct pattern of extreme deficiencies.

Granögger: This is a very sick person, it seems.

Leisk: Yes, this is a very sick person. This is a very unhappy person who might be stuck in bed and unable to feed themselves, for example, or wipe their own backside.

Granögger: Maybe for the audience, ME/CFS chronic fatigue syndrome is something that can end you in bed permanently. People get so bad and very quickly, this can happen, doesn't it?

Leisk: It can.

Granögger: You can just lie in bed; you have no energy, and you don't know how to treat this. It's very similar to what people describe as long COVID, as you also have in a text here. There is more research now going into this.

Leisk: It's essentially the mitochondrial dysfunction. The mitochondria are like the engines inside the cell. They generate the ATP, but they also produce several different things for all these different pathways. They're like factories, essentially. Because of the different things that get created or the deficiencies that are created, the mitochondria cannot perform their jobs.

Of course, things like maintaining the electrolytes on the insides and outsides of muscle cells via little pumps called ATPases don't work well if there's not enough ATP for those pumps. That can lead to fluid retention, which can lead to lactic acid metabolism burning. Depending on the tissue, if it's cardiac tissue, you might end up with fluid building up on the heart or lungs and things like that.

Of course, there are some straightforward ways to intervene in some of those things once you understand what that problem is. It's certain things that can sometimes be dramatically changed, even within 20 minutes in severe situations, if you're doing the right things. Unfortunately, it's not well understood at this stage.

Granögger: All these minerals and metals are missing; how do you bring them back?

Leisk: Actually, I'll step back here. The challenge here is that because the body is actively rejecting these metals, it actually shuts down both the transporters, so DMT1 and ferroportin, in the gut. That means that the enterocytes in the duodenum stop absorbing the metals from the dietary intake, and they don't pass them into the body.

Other cells in the body, such as the brain, the liver, and the kidneys, only have ferroportin and are not divalent metal transporters 1. That has a different effect. What that does is, essentially, those cells act like a bit of a sponge. They sequester those metals out of circulation and hold onto them until the inflammatory signaling and hepcidin come back down again, at which time they release them.

The net effect of all that is to essentially deprive any pathogens of those metals, giving the host an advantage in an acute infection. It makes sense. There's a benefit to the host when the infection only lasts a short period, but in the context of these biofilms in chronic infection, particularly due to the co-infections in this cascade that it be going further into. What it does is essentially create deficiencies of these metals over time. The organism ends up declining in a fairly significant and predictable way.

One of the challenges comes down to how the immune system deals with different pathogens. To explain it, we have, for example, different types of interferons. There is interferon alpha, which is particularly good at dealing with viral infections and how it changes some of the metabolism inside the cell. Then there's interferon-gamma, which can also be useful in viral infections, but it has a particularly useful function in removing or killing different microorganisms.

One of the tools that it used to do that is to generate these reactive oxygen species to oxidize the pathogens themselves. That might be like making hydrogen peroxide or superoxides from these immune cells to pop open the other microorganisms then effectively. There are a handful of different enzymes involved in that, including xanthine oxidase, which is also involved in gout, as you probably would've heard. When people have gout, purines are metabolized into uric acid, creating these reactive oxygen species. Gout appears to be a direct effect of ongoing chronic innate immune response, most likely to a microorganism somewhere in the tissue.

We have other enzymes, like NADPH oxidase or NOX, which can generate

these reactive oxygen and nitrogen species. The other one, nitric oxide synthase, is the one that can generate reactive nitrogen species and oxygen species.

In the context of how the body then protects itself against the weapons that it's deploying against these microorganisms, what it also does is, when interferon gamma is elevated, what it does is promote the enzymes that the body uses to metabolize reactive oxygen species. Those are metalloenzymes. They're proteins or enzymes that require certain metals. That might be, for example, Cu,Zn-SOD copper-zinc superoxide dismutase. It might be MnSOD or manganese superoxide dismutase. It might be catalase, which needs heme, which is a form of ion, for example.

There might be glutathione peroxidase and reductase. That needs selenium, which requires certain metals for riboflavin metabolism. That's things like where we need iodine and, again, selenium, and we need calcium as well as a few other things, including zinc, of course, for riboflavin kinase. If any of these are deficient, then we can end up with the body's weapons creating a dysregulated immune response and generating oxidative stress, which ends up damaging the tissues themselves, so the collateral damage to the surrounding tissues where the microorganisms are.

In this way, we might have a clinician believe that the person has an overactive immune response because it's creating damage to the surrounding tissue, but, of course, it's a chronically activated immune response because it's not clearing the reservoirs because of the biofilms, but it's also having the collateral damage being created by the lack of minerals, affecting the cell's ability to have resilience against these tools.

Granögger: How do you fix this? This sounds really, very bad. It doesn't seem possible to supplement these minerals orally because the gut doesn't take them or transport them.

Leisk: That's right.

The solution is to bypass the oral absorption route. What we do in the protocol is use the sublingual route. You can, of course, use IV infusions and any other injectable minerals where suitable, and the clinician will make those available. We're looking at an upgrade. We've had a pharmacy in Australia producing these lozenges or troches, which dissolve under the tongue. Because of their amount

of metals and vitamins, they don't have the most amazing flavor, but they are acceptable if somebody is chronically ill.

We are looking at some other options moving forward that are even more efficient than the sublingual route, which has similar absorption parameters to the IV route, but, yes, essentially, much more accessible, particularly if somebody is not mobile and they have to do all this at home.

Granögger: How about through the skin?

Leisk: Yes, absolutely. This is one of the things: The problem you have is the size of the molecule, or the compound's molecular weight, affects how much of that will be absorbed by the skin. This is why the mucosal tissues are more efficient, but then you can use transport enhancers, like dimethyl sulfoxide, DMSO, which are amazing at transporting different things through different membranes.

Of course, DMSO has several different desirable effects. It's been used by the military for traumatic brain injury, burns, and for all kinds of things. It's something that requires a bit of respect, and it's something that gets labeled appropriately when it's used as a lab region for various things. DMSO is known as one of the most amazing solvents for many, many, many compounds. It's one of the standards against which it's measured. DMSO is likely something that's going to appear as part of a solution to replace the lozenges or upgrade the lozenges at some point in the future.

Granögger: Yes. I heard you begin to smell like garlic if you use too much.

Leisk: That is true, but mind you, if the state you're in is that you've been in bed and you haven't been able to have a bath for two years, the additional sulfur smell is probably the least of the challenges. For somebody who's still getting out and working and all the rest of it, there may be a social component or antisocial component to the use of DMSO. It's not the worst thing ever, but some people have a particular distaste for the smell. It depends on the person and, yes, the coinhabitants of the household.

Granögger: I think if we summarize now, we have those biofilms that grow unnoticed. They take from the host, our body, they take the important minerals, or they disallow the minerals to be transported to where they belong.

Leisk: A bit of both. They will use some of our nutrients to build biofilms and for all their own cellular machinery when it's available. In fact, some of the microorganisms found more often inside the blood, like the spirochetes we see in Lyme disease, are Bartonella, Babesia, and Borrelia. Of course, we have other things like Rickettsia and others that turn up in different countries more often in testing; I suspect that we'll probably find the spirochetes in all countries. It's just that some countries test for it, and some countries don't. We have countries here, like Australia, where we don't believe Lyme disease exists.

Some of those spirochetes are really interesting, as they can actually take manganese. Before, I was talking about MnSOD, which is the dismutase of manganese superoxide. Some of those spirochetes can take the manganese out of our circulation and use it to boost their own MnSOD to protect themselves against this oxidative attack from our immune cells.

Granögger: These become parasites; literally.

Leisk: Literally, yes. They have a parasitic function. Very interesting. I have some slides of blood where we've seen spirochetes invading red blood cells and little biofilms that they've come out of.

Granögger: Do you want to show those or go through the rest of them?

Leisk: We'll go through the rest, and I might go back and show you one of those. We talked about the metals. We could talk about the impact. We can see that various metals and electrolytes affect different metalloenzymes without going into all the details. Then, ultimately, if we model what that does to the throughput of different pathways, that means that we're going to have effects on things like neurotransmitters, energy metabolism, histamine metabolism, fatty acid oxidation, blood volume, nitric oxide, all kinds of different things, and, of course, collagen synthesis.

All the key pathways that we see affected in various chronic diseases can be affected by these mineral deficiencies and by, potentially, the endotoxins that I've been describing, and of course, oxidative stress, and leading onto another common issue, and this has been particularly a hot topic since COVID and things related to COVID is hypoxia.

Hypoxia is when we can't deliver oxygen at a sufficient rate to cells. Cells need

this for several different important enzymatic reactions. It's not just energy metabolism; it's also things like neurotransmitter synthesis. There's a significant impact on the immune response when there's hypoxia. It's something that also triggers the reactivation of the latent herpes viruses and things that we were talking about before.

In the context of what we've seen in the last few years, we've seen the introduction of the spike protein. The spike protein potentially can get degraded into an amyloid-like protein by one of our enzymes called neutrophil elastase. It can then potentially bind with fibrin. We can get these fibrin amyloid plaques, which are believed to be coming from this mechanism. Also, we saw fibrin amyloid long before COVID as well, which was being studied for things like type 2 diabetes. I believe other mechanisms also create this.

Hypoxia, apart from spike protein-related stuff and all the latest influences around that, we also see that when there's low iron because we're not transporting the oxygen, if there's various other mineral deficiencies which are going to be affecting key parts of the metabolism, particularly around that oxidative stress. When the immune system is responding to different types of microorganisms or viruses, the different ways that the immune system can react to them can create aggregation of the red blood cells so that they can clump together in different ways.

We can see that as microclots; we can see that via the reduced zeta potential. That's when the negative electrical charge on the outside of the red blood cell is reduced. That directly correlates with the pH and the potential of hydrogen, so they stop repelling each other; they start sticking to each other. Then we've got various other immune response functions that will potentially create different ways for these things to get tangled up. That's NET, or neutrophil elastase traps. Then, of course, there's a fibrin, fibrinogen, and various other things that could be happening.

In the context of, unfortunately down to the 'weeds', what creates a trap or a cysto between these different co-infections which we're potentially accumulating piece by piece by piece over time, what can happen is that the prolyl hydroxylases….Before, when we were talking about what happens with cancer cells and hypoxia and Warburg metabolism, the enzymes over here, prolyl hydroxylases, function to sense metabolic faults. Primarily, they're known as a hypoxia sensor.

For example, if there is not enough oxygen, which is a co-factor for these prolyl hydroxylases, then the enzymes stop working, and the degradation or hydroxylation of this protein called hypoxia-inducible factors 1 alpha, or HIF-1 alpha, that protein is stabilized. When the protein stabilizes, it alters transcription factors in the cell. In other words, it changes the expression of different proteins or enzymes to then change the way that the metabolism in the cell works.

In this case, I could, and maybe I'll do that, show you another diagram, or in fact, the one that we're looking at here, but just in a bit more detail. What we see here is the citric acid cycle. That should be sufficient. We see the citric acid cycle, and we see these prolyl hydroxylases. What can happen is the prolyl hydroxylases are going to become inhibited if the co-factors are not present or low. In other words, if there's low oxygen and vitamin C, that's oxidative stress.

Of course, the bias for this will be stronger if there's low iron, which we talked about before with the mineral losses, and low silicon, which we haven't talked about yet, but that comes downstream, we believe, of the acetaldehyde cascade. One of the ways that the acetaldehyde in the body can be removed is by binding and forming an adduct with silicon and being excreted as a silicon acetaldehyde adduct. If the silicon's missing, the iron's low, and if the zinc is low, which is there to stabilize those enzymes, then it's more likely that the reaction's going to fail, which means it's more sensitive to hypoxia; it's more sensitive to low vitamin C.

Granögger: It's another vicious circle, isn't it?

Leisk: Yes, it is. The interesting thing with this is the other parts of the cascade. If we are dealing with problems with energy metabolism because we have, for example, problems with metabolizing carbohydrates, so the glycolysis pathway or collagen synthesis and things like that, that's up in the diagram, then we will end up with low substrate. We will end up with low alpha-ketoglutarate, which means that that reaction won't work either.

If we have various things that cause an elevation of the product, which is succinate, and there can be different things in different immune pathways that trigger it, then we can also end up with an inhibition of the prolyl hydroxylases.

Of course, any of these conditions are true, or the more severe this will happen,

the more likely it is that that protein, that HIF-1 alpha, ends up being stabilized. When that protein stabilizes, it then promotes the expression of several different enzymes. We end up with elevated lactate dehydrogenase. We know of lactate dehydrogenase primarily as the one that can metabolize lactic acid into pyruvic acid and back and forth, and that ends on, of course, in zinc, which is part of the anaerobic glycolysis that we talked about before, and Warburg metabolism, what it also is known for is another common issue that we see, which is hyperoxaluria. That's the synthesis of oxalates, which is also promoted by converting glyoxylate into oxalate.

Granögger: Which mean?

Leisk: When you see kidney stones, when you have sand in the urine, when you're getting random joint pains and various other symptoms, it can affect things like thyroid hormones and neurotransmitters. The oxalates also have a potentially antimicrobial function. I believe there is some purpose to it. What this means is that you end up with pain and potentially other symptoms created when you have this hypoxia metabolism active all the time.

Granögger: Arthritis?

Leisk: Potentially, although I believe other mechanisms and localized infections are involved in many of those cases. Yes, certain types of joint pain. Absolutely.

The other thing that HIF-1 alpha stabilization does is reactivate those herpes viruses and convert them from their latent state to their lytic state.

Granögger: Then we are back where we started.

Leisk: We are back on the other side of the cystoscopy. In the context of COVID and COVID interventions, the spike protein potentially causes some different mechanisms that cause the coagulation hypoxia, then leads to the reactivation that we've seen time and time and time again in the literature of Epstein-Barr virus and people getting shingles and all the other things which have been commonly reported. Then, of course, that provides an antigen, triggering this interferon alpha response and distracting the immune system for longer periods in dealing with microbial infections.

Granögger: Then the biofilms can grow even more.

Leisk: That's correct. We've seen this for decades and decades. If you have influenza and you're elderly and you go into a hospital because that influenza is particularly difficult, normally, they'll give you some kind of antibiotic for bacteria to prevent secondary bacterial pneumonia. It's not that the virus will be affected by the antibiotic; it's the fact that the existing mess in the biofilm in the lungs will be potentially allowed to proliferate and bloom, which then causes this bacterial pneumonia.

Granögger: What you're saying is that bacterial pneumonia doesn't come in the hospital from someone; it's already there

Leisk: It's already present. In fact, from the data I've collected from numerous sources, we can sometimes see even 14 different pathogens in the lungs. Quite significant amounts of it in some cases, where they've had a history of asthma or COPD and things like that; those diseases. Asthma appears to be a buildup of the biofilm in these lung species. Of course, every time, the immune system is capable. It could be, for example, asthma, which is triggered by exercise. Without getting into some of this model's other key parts, it could turn into a long and detailed conversation if we ever wanted to do that.

Granögger: I think we need to wrap up the difficult part.

Leisk: Exactly. This is the part that I enjoy most. Essentially, exercise generates the metabolites needed for the inflammatory response, the interferon-gamma response. If you exercise and you go out, this is where some things post-exertional malaise, the known symptom for long COVID and ME/CFS and post-vaccine syndrome, and progressively more chronic diseases. There was a recent paper on that, which was 'rubbing' people the wrong way over the last few months; yes, what basically can happen is that oxidative stress can end up inhibiting a lot of the energy metabolism.

It's not just the oxidative stress here with the citric acid cycle; we'll also see it in other key places like the glycolysis pathway. That's basically using the carbohydrates, glucose, or glycogen and converting that into ultimately pyruvate and then into the citric acid cycle here. When you exercise, you generate the metabolites needed by that immune response, and then the inflammation is generated. The body is trying to do what it's supposed to be doing. It's actually

attacking the free-floating microorganisms that it can see.

Then, we get concerned about the inflammation in the lungs. We then puff on BREO or some kind of corticosteroid or something to inhibit that interferon-gamma response. Then, of course, that allows the persistent ongoing cycle to occur because we never clear the underlying infection.

Granögger: Yes. That's the detriment of symptom-treating instead of understanding the whole, as you call it, the cascade of things that go back, perhaps even to birth.

Leisk: Yes, it generally would be; that's where it starts.

Granögger: That's totally amazing, Joshua. I now really have to ask, how do we get out of this vicious circle? As a normal person, normally healthy, do you need to do anything?

Leisk: Yes.

Granögger: Can you do preventative things like treating your gut microbiome or taking minerals? I'm not talking about really chronically diseased people who need a very different intervention, I'm sure, but what can a normal person do?

Leisk: If somebody is not suffering from large amounts of inflammation, they still absorb minerals orally. First, you can ensure that the diet is appropriate and that you get the normal daily amounts of various minerals or more. Make sure that your electrolytes and everything else are being maintained. Many different dietary foods and things can break down certain biofilms. There are things like chili curcumin. Some other things could be obtained. N-acetylcysteine is a supplement. It's useful for biofilms.

Granögger: How much do you have to take? Is this every day, and how much curcumin would you put on?

Leisk: I think a little bit every day is helpful. The problem you have is that the biofilms will be in all kinds of different tissues. The different biofilm breakers that you can consume, some of those might, for example, get a small way through the upper GI, so the small intestine, and then get absorbed into the system. They won't have an effect that goes down through the bowel.

Some specific products have been created. There's a product by Dr. Paul Anderson, which is rather amazing. He's done decades of work on biofilms. He's got what he terms Phase-2 Biofilms. There are these particularly stubborn hard ones that don't break down with basic thiols and things. He came out; he did a lot of research into ways to break open those biofilms using things like bismuth thiols that are reacted with alpha lipoic acid and DMSA and various other things. He has different recipes.

There's a product called Biofilm Phase-2 Advanced. You would not take it daily because it'll also break open good, normal biofilm. Even your good flora has biofilms. The recommendation is to use it for four days and three days off at different doses.

Granögger: What do you have to deal with afterward? If you break those biofilms open, wouldn't that release first?

Leisk: Trigger an immune response.

Yes, you have it; very good, very perceptive. Therein lies two problems: If you break open the biofilms and the immune system is distracted or otherwise compromised, all you're doing is essentially liberating those microorganisms, and some of them might go out if it's a gut microbiome, but others might just find a new area to stick to, and the whole process goes over and over and over again. If you combine that with a functional immune system, it will probably work out in your favor in the long run; you're likely still to get an immune response and a Herxheimer effect.

One of the challenges is that when the microorganisms that are producing these toxins get popped open, when the cells are opened up, then the toxins that were inside them, which will often come up when they get happy because they've been fed also then circulate and the body has to process them. There's a Herxheimer or die-off effect, which can cause headaches, chills, rashes, fevers, muscle pain, etc. People might get all the things when they react to food for certain reasons or to fibers and have all kinds of dietary sensitivities. The process of clearing out those biofilms. It's not necessarily a pleasant process, but it gets easier and easier over time as you work through it.

Granögger: Do you do this yourself?

Leisk: Yes. My own maintenance is fairly, I won't call it comprehensive; plumbers and pipes always drip, but I consume several useful things for breaking down biofilms and just staying on top of that. Of course, it's all the other things, too. It's making sure that the diet is good and sure the exercise levels are good. You're either thriving, or you're declining. There's no stationary holding pattern.

Granögger: Before I ask you more about yourself, there's ivermectin and fenbendazole, and many people are talking about these parasitics. It seems to me that they would be breakers of biofilms. Maybe that is also why people are saying that they can work on cancer. Would that play into the cancer, but what do you think of a normal person taking ivermectin or fenbendazole occasionally?

Leisk: I think there's a benefit. If you look at what we do with our pets, animals, and livestock, you deworm, and you need to eliminate the parasites in them at least every year. We forget to do that with our children and ourselves. Parasites are another thing that potentially accumulates. Ivermectin has many different functions, though it's not just an antiparasitic. One of the things that it does well is that it is a biofilm breaker.

I think that one of the benefits people saw when using it for COVID, for example, is not the fact that it does anything particularly amazing with the actual infection. I think it has more to do with the fact that it didn't allow the proliferation overgrowth of these other microorganisms while the body was busy. Some papers showed some anticoagulant functions, and certain things were around different spike proteins, but I don't think that that was why it was having the benefits towards mortality; I think it was dealing with the other co-infections.

Granögger: In your perspective, it is safe to use it once or twice a year?

Leisk: Ivermectin… look, I can't give medical advice.

Granögger:. That's true.

Leisk: But I would say my personal opinion is that I would be okay if I were to take ivermectin sometimes for a year as some sort of clear out for certain types of parasites. The thing is, it will only target certain types of parasites. It's not

going to work with other types of parasites. That's why some of these protocols I've seen online might have a mix of fenbendazole, mebendazole, Ivermectin, and various other things. I have personally seen, thankfully for the people who love to send me photographs of these things, different parasites coming out of people in different places after using some of these products.

Obviously, their health status has significantly changed as they've worked through some of these problems. As far as doing what they're supposed to do on the label, yes, they appear to work very well for those sorts of things. As for cancer and some of the other things, what's interesting with that class of drug, so the fenbendazole and ivermectin, and some of the others, is that they're not just an anti-parasitic. When you investigate the literature and what they were designed for, they have limited effects on fungi and other microorganisms.

In the context that we were talking about before with the hypothesis around the apoptosis pathway being disabled by these various latent viruses and then the lateral gene transfer from fungi, parasites, and various other things, these anti-parasitics/antifungals may be able to selectively target the cells which have these other DNA or the proteins that are being produced via the lateral gene transfer.

Granögger: It makes very much sense. I also started to use ivermectin fenbendazole for myself, and it's not medical advice because I'm not a doctor at all, but I do see benefits. It's also happening with much more clarity in the brain and much more likeness. Personally, I would use it. Let me ask you how did you get into all of this and that you're so passionate and deep, as you're saying, 'in the weeds'? What's your background?

Leisk: My background's quite interesting. Originally, I was somebody who had ME/CFS as a teenager. I was one of the lucky 4% that went into remission. I then had a very interesting life that followed from there. I had various other health challenges that came afterward. In one case, it was, for example, antibiotics after a motorcycle accident, which then led to me not being able to consume gluten for about 15 years without bleeding rectally for about two weeks.

Later, I think we discovered a candida infection that was allowed to overgrow because of the antibiotics.

My background was largely engineering, so my childhood was very different. I

used to have what some might call an eidetic memory. I had a stroke in my late teens. I no longer have quite the same memory, but I still have a very good memory normally. I started with computers at a very early age, at age three. I was writing software at seven, reverse engineering software, and then moved on to doing electronics engineering, mechanical engineering, and then running an IT company for the better part of 16 years.

On the side of studying; biochem, endocrinology, pharmacology, neuropharmacology as side interests, and ultimately when I sold up the IT business and put my feet up and was reaching a bit of burnout there, but it was time for a change, the universe basically dragged me into studying chronic fatigue syndrome again. Not that I realized that that's what it was at the time.

I was already passionate about mitochondrial metabolism and endocrinology. I was deeply more into exercise physiology at that time. Going back after 16 years of IT and some decades of engineering in total, my body and health were not exactly at their peak then. That was a bit frustrating, so I decided to focus on all the available time that was on my hands and try to put things back together.

Then, while I was doing some mentoring and coaching and things for general health and wellness, I found that I had a pattern of different clients that were coming to me, which had the same kind of markers; they had the same challenges; food sensitivities, fatigue, insomnia, hormonal dysregulation, all these different things. I was quite intrigued by that. I started looking at that data and pulling it apart, and then I started seeing the patterns.

Initially, I was a bit myopically focused on the herpes viruses, thinking that that was what was creating all of this. Then, as things progressed, particularly once we got into the COVID period, I was suddenly drowning in data showing me that many other things were going on. At the time that we came into COVID, I had already had some people around me who had gone into remission for chronic fatigue syndrome, and then a number of them went from being in remission to being bedbound again after taking certain interventions for COVID.

Of course, I found that fascinating and horrifying at the time, so I went ‘down the rabbit hole’ for a few months trying to understand what had happened there. From a little informal study that I'd run, I found that there was a coagulation and clotting or micro-clotting issue that was quite broad. Then, that

led me to look at the hypoxia issues and the reactivation, going through the literature.

That then, of course, led to the mineral deficiencies that we picked up, and then the challenges and the methodology and the reason that we didn't see that and a lot of the other data. Then, after that, trying to understand what was sitting above all of that, I started looking at the cytokine patterns and trying to understand what was actually triggering those cytokines, but normally try to do serology actually to understand what pathogens are there. It's a bit like throwing darts in the dark.

You think you might have, for example, Epstein–Barr virus, so you'll run a test for it to look for IgG and IgM for different proteins or antigens. You might have like EBV, EBNA, and VCA, and early antigen diffuse just to see what infection, or if you've got EB Epstein-Barr virus, whether it's reactivating or recent, all those kinds of things. Let's just say that's one pathogen out of many, many, many. Unless you're going to run every single pathogen individually, which is incredibly invasive and expensive, it's not really going to be a very fast way to try to identify any common patterns.

Now, when we look at the microorganisms and some of the other viruses, the way that we can look at things, there are labs now, in fact, these are mentioned in the protocol, where they can test 57,000 species of microorganisms coming from one microbiome test, like from a nasal swab, oral swab, any of the other tissues or blood sample. That has been a bit of a game changer for actually looking at some of these things.

If you then correlate the data that you're seeing there with the cytokines that you're seeing, you then start to see the patterns of how the body responds to these different threats. Then, once I started looking at these patterns, I started understanding that we're not looking for one mystery infection; we are looking at how the body responds to various pathogens.

Then the question was: Why is there persistence? Then we started digging into the 'weeds' of finding the biofilm research and everything else. Then we understood the problems with the blind spot and that we essentially had multiple co-infections, each requiring a different immune response. Then, the challenges arise because the whole system is in a state of disarray. It was a process. I probably put 15,000 hours just into this topic over the last, say, five

years, and unpaid. It is just done because, ultimately, I figured it needed to be done.

Granögger: I feel you've discovered something, but who am I to say this? I'm sure you're working with professionals, you're working with institutions, and you're doing studies that are being prepared now. I think you may have found an underlying, fundamental level of disease, at least of chronic disease, maybe of most diseases. I hope this was informative and that people will at least check out your website. Your website once again is bornfree.life. Can people contact you? Would you like that, or are you also giving advice? Many people who have chronic fatigue are watching or reading this.

Leisk: There are many challenges. The biggest challenge I have at the moment is replicating myself. Just looking at my inbox and DM queue and everything else these days, there are 400 people just in the queue at the moment. There is a bit of a delay. I do get back to everyone, but it takes a little time. For very severe cases and things where people are bedbound, tube-fed, and things like that, there are mechanisms in place to allow more rapid response to that.

What I would suggest is checking out the website first. One of the things in addition to the volumes of information that's already there, there's also an AI that we've set up as a study guide. You can use it as a study companion to actually absorb the concepts and even get down into the 'weeds' for both the protocol and the model. That will continue to improve, particularly when the paper is finished, and then the AI will be trained on all of the nuances around that.

We're in the process of setting up a clinician education platform, which is going to be the stepping stone towards scaling things up. Some doctors and other practitioners are studying the model and using it with their patients. That's essentially the way that we're going to be continuing to scale this out. Anecdotally, until the clinical trials are finished, we have seen people go from very severe states, palliative care, and feeding tubes out back to work and normal life.

Some of these things can happen very rapidly. Some of these things will take a little bit longer, and it just depends on various variables, how good the data is, and how aggressively you can actually intervene. Some people are very fragile and need a very slow run-up before we can get them into the next stair-step

levels. Good things are coming, but certainly, I would suggest checking out the website, seeing how best you go absorbing the model, and reaching out if there are challenges, and there are mechanisms to do that on the website.

Granögger: Those products are sublingual, those lozenges and maybe drops or something. Are there recommended links?

Leisk: They're in the protocol. As it's currently set up, the protocol takes everyone step through the process, what tests you need to do, how you need to interpret the data, and how to acquire all the products. Because it's a little bit of an early adopters type arrangement at the moment, we are trying to solve some of the challenges where there are essentially two pages of products that are required because there is no existing manufactured product with everything in it.

That's one of the next problems to solve. I was hoping that would've already been in place, but at least we've solved some of those challenges with the lozenges. There are some efforts in the works at the moment to try to have either a compounding pharmacy or another manufacturer actually step in and make the rest of this process a whole lot easier.

Granögger: Excellent.

Leisk: People who are severely ill; it's challenging enough to get through the day, let alone some of the overhead required at this point in the game. As things progress, as the clinical trials pan out and assuming everything goes well, it'll continue to scale, and we will get even more efficient with the process.

Granögger: Wonderful. Joshua, I hope that not only lay people like me will listen or read this, but also professionals and perhaps feel inclined to look into this, to look into your model. Those people should contact you, perhaps, if they are high-level professionals in laboratory settings.

Leisk: Researchers and clinicians, absolutely. Look, that's definitely the focus at the moment, is to try to scale it up.

Granögger: Thank you so much. If need be, we might have you back on, especially if you have more to say, and if you have new breakthroughs, I would love you back on *The Solari Report*. Thank you so much, Joshua Leisk.

Leisk: Thank you so much.

MODIFICATION

Transcripts are not always verbatim. Modifications are sometimes made to improve clarity, usefulness and readability, while staying true to the original intent.

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