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Hi everyone, today, we're going to talk about leaky lungs syndrome. I'm going to explain to you some of the research behind leaky lungs. I want to go over what the pathophysiology is, what the mechanism is. I want to explain to you what the symptoms are for people that have this pulmonary or lung permeability. Then when talked about what kind of conditions and diseases can cause it, and then what's being done for testing for pulmonary permeability, or what we call leaky lung syndrome is. In the literature it's referred to as pulmonary epithelium permeability. The slang term for it is leaky lung, just like with leaky gut syndrome, it's intestinal permeability, and they use the term leaky gut just to make things simple. But when we say that word leaky lung, we're really referring to pulmonary epithelium permeability.

It's a mechanism that is well published in our literature. It's just people don't use the term leaky lung or leaky lung syndrome. If you're trying to communicate with, let's say pulmonologists, they may not understand your definition of leaky lung, just like gastroenterologist for many, many years, didn't understand the term leaky gut until it became more and more and more popular. The basic concept is ... now one of the things that's really critical for everyone to maintain their health is to have healthy barrier systems and we have barrier systems throughout our body. We have barriers that keep foreign compounds or the environment out of our body or less exposed to the immune system in the gut. That's a gut epithelium barrier. We have it in our brain, the blood-brain barrier, and then we have it in our nasopharynx, our nose, respiratory pathways in our upper respiratory areas.

We also have it in her lungs and this is the pulmonary epithelium and leaky lungs. Let me just review some really quick lung anatomy with you and then explain the big concepts. Your lungs are basically very flexible tissue that's there to take in air and really involved with the ability to take in oxygen and blow out CO₂. Now when you look at this sack filled organs think of them as a tree. Then the best way to think about this is to learn the anatomy of the lungs, to just think of the windpipe as a tree trunk and the rest of the windpipe be it the windpipes involved to carrying air into oxygen, and then the tree trunk branches out. Then these branches, I'll call bronchi.

Then these branches or bronchi spread out into smaller branches or these tunnels where air is being transported to your lungs called bronchioles. At the end of the bronchioles, there's something called an alveoli. This alveoli is where you actually have the exchange between oxygen and CO₂ and gases and things you inspire. In this alveoli, you can think of it as like the leaf of the branch. In this alveoli you have a barrier called the pulmonary epithelium barrier and this pulmonary epithelium barrier has tight junctions just like the gut and there's certain proteins there. They're referred to as claudin protein, C-L-U-D-I-N proteins. There's different claudin proteins. There's claudin



three, claudin four, claudin five, claudin eight, many different claudin proteins, but those claudin proteins keep the pulmonary barrier intact.

So chemicals and pollutants, we breathe in don't really get into our lungs and activate our immune response. Basically when these tight junction proteins in the lung breakdown, that's, so-called leaky lung or pulmonary epithelium permeability. This is actually very common and it's really overlooked as a part of chronic disease and chronic triggers for autoimmune patients and chronic inflammation for many people. Just as research continues to show that many people actually have leaky gut syndrome or blood-brain barrier permeability, more and more research is showing that the large majority of people that have chronic inflammatory lung diseases have some degree of leaky lung or lung permeability. For sure there are people that have some subtle lung permeability issues that are having symptoms that are subtle like asthma or other issues. But I think it's an area where more and more research is going to continue to come out. We're going to see the same trends as we saw with leaky gut.

Now in research studies, they are able to do biopsy studies and look at these proteins and see them break down and understand what's happening with them. Different studies have been done with them to find association with inflammation and immune responses and chronic lung diseases, but in a more subtle sense you can have permeability of the lungs from very simple things, just like air pollution. The best way to think of how the lung barrier breaks down, it's really no different than the blood-brain barrier or the gut barrier. Chronic inflammation is what's going to break down these barriers and chronic inflammation or pro-oxidants or oxidative stress these inflammatory reactions, whether it's from an inflammatory disease or from air pollution or from cigarette smoke.

Those are going to cause some destruction of these epithelium cells and the tight junction proteins in the lungs. The only way to combat this, is an antioxidant system and something called secretory IgA cells and secretory IgA cells, we'll talk a little bit later through immune cells. They basically bind to whatever is coming in like air pollution or chemicals. So it doesn't really cause an inflammatory destructive response against the lungs. Then if that SIgA or secretory IgA cell doesn't engulf the inflammatory chemicals, then that chemical or that inflammatory irritant is going to start to cause inflammation or destruction against those tight junction proteins. When that happens our antioxidant system and in the lung the most steady detoxing system is the glutathione system. It seems to be very important. It plays a very important role in maintaining your lung epithelium or lung glutathione epithelium levels, then protect for those cells to get damaged.

You could have some people that have really healthy secretory, IgA cells in their lungs because their immune system is healthy and they've really healthy antioxidant systems. They get around a cigarette smoker, air pollution, and they're fine. Other people that



have a compromised barrier and have very low SIgA cells because their immune system is weak. They can get exposed to retain some chemicals and then those cases, those tight junctions or may break down or are already broken down. These chemicals can get through the lungs and then cause this inflammatory response. But you have to realize we're inhaling chemicals all the time, and you don't even see it, it doesn't have to be something mysterious. If you can just look at air pollution levels in different regions, there's many different sites and apps that can look at air pollution for whatever zip code you're in and give you an idea of what's happening with the amount of chemicals in the air, but they're everywhere and we're breathing them all the time.

Sometimes the air quality is really, really poor. It's so bad that they report that in the news and when that happens, people that have intestinal permeability or at risk for having some inflammatory reactions, if they breathe the air. If people have a really poor antioxidant systems, if they get exposed to bad air quality these tight junctions can break down. Then when they break down, this sets up the stage for a significant inflammatory cascade. The key things to remember as I summarize here as I teach you step by step. The key concepts is we have these, we breathe in air and blow out CO₂. At the end of the tree are these alveoli and then there's a barrier there. Then that barrier has tight junction proteins called claudin proteins.

Then those claudin proteins are very sensitive to anything that is a pro-oxidant like a chemical or a pollutant or any type of inflammatory response that takes place in the lungs. Things like infections can also break down the lung barrier and pretty much every inflammatory lung disease like bronchitis or COPD or emphysema that have an inflammatory component to it have been shown to break down the lung barrier system. Just like when people have chronic inflammatory gastrointestinal diseases like celiac disease or Crohn's disease or ulcerative colitis, studies show, they have leaky gut. They have intestine permeability. If there's an ongoing inflammatory response in the lung from a disease, then we just know the tight junctions are broken down.

Now let's talk about the less subtle ones. There's less subtle presentations of leaky lung syndrome, which is really most important for people that are suffering from chronic autoimmune diseases and chronic inflammation. Is that when they get exposed to really poor air quality, that's when their symptoms flare up. It's just like if someone had leaky gut, they get exposed to an inflammatory food protein, and then they get a reaction with their joints. Let's say that a patient has arthritis. If a patient has arthritis and they have, let's say they have an autoimmune reaction against their joints and antibodies to collagen peptide. They have like rheumatoid arthritis and they have this chronic joint pain and they know that certain things are going to make their joint pain worse which are attribute from their immune system. For example, many people that have arthritis are very sensitive to night shades. If they eat, let's say tomato or eggplant, they notice significant joint pain because of that. Since that immune sensitivity, those food routines for them.



Now, for people that have lung epithelium barrier breakdown and also have RA, they just may get exposed to really poor quality air. All of a sudden their joints really, really hurt, or they could be working in a room or in the garage or working to restore a house or do something and that air quality really then starts to flare up their joints. That's the key difference. So whether a person has, for example, has rheumatoid arthritis or they have Hashimoto's disease or any other type of autoimmune disease. One of the key things that I know I do as a clinician, I always look to determine if there's any clinical patterns of lung epithelium barrier issues. What I find is that many patients don't know. Many patients don't actually know that they may have air pollution for example, or really triggered them. Some may have found the association with cigarette smoke, they get exposed to cigarette smoke.

There's so many chemicals released in cigarettes besides the aspects of tobacco that are in the filters that really can be inflammatory for people that have leaky lung syndrome and auto-immunity. Sometimes I'll have patients tell me that they're really sensitive to cigarette smoke if they're in a room with a lot of cigarette smoke, their autoimmune disease flares up. Maybe it's their joint. Maybe it's their thyroid, maybe whatever the tissue protein is under attack. That's one of the key features thinking sort of, now, most people don't know what the association with air quality. They have to start to look at air quality and start to see if those patterns are there, but that's your biggest clue. You don't have to have a full blown expression of a lung disease. Now they have found studies, linking asthma and wheezing with people that have permeability of their lungs, but not all forms of asthma lead to lung permeability. But we know that people that do have lung permeability, they can't have wheezing and an inflammatory response and constriction of the bronchi when they have an inflammatory exposure.

The key thing is that if you have an autoimmune disease or are suffering from chronic pain or chronic inflammation. If you know, poor air quality or getting around inhalants that could be toxic, let's say chemical based or pollutants that are really causing you to then have a reaction like joint pain or chronic pain afterwards. It may be either that day or the next day, that's a big clue that you may have to test lung pulmonary permeability. Now, the other symptoms that typically take place with pulmonary permeability besides flaring up and auto immune disease is just general fatigue. One of the things that happens is that if you have, let's say a leaky lung pattern, and you do trigger your immune response beginning exposed to pollutants and inhalants that are inflammatory, that's going to cause these particles to get through your lungs.

It's going to cause a systemic inflammatory immune response and your whole body's going to get inflamed. When the whole body gets inflamed, it has an inflammatory response. Inflammation shuts down or uncouples mitochondria throughout the body. Most people just feel really tired, really fatigued and it could be muscle fatigue. It could be brain fatigue it'll have an effect throughout the whole body. Chronic pain and autoimmune flare up general fatigue, brain fog that are triggered by a respiratory trigger



or all clues that a person may have leaky lung syndrome. If you're listening to this and you have an autoimmune disease or a chronic pain syndrome, you may want to be very focused on these mechanisms and know that they may be part of what's triggering your autoimmune disease. What I find is a lot of patients that have autoimmune diseases or chronic pain syndromes, they're focusing so much on dietary foods and not so much in air quality and the fact that their lung permeability maybe involved.

Now what does the literature show and what does the scientific literature show that can cause these tight junctions to break down? Well, any inflammatory disease, again, CLPD, emphysema, bronchitis, smokers are going to have permeability of their lung barriers. That's a lot of free radicals that they're exposing to the lung barriers. Areas with high amounts of air pollution. Areas with any type of respiratory infection can cause it. Obesity can cause permeability of the blood-brain barrier. One of the things I said about obesity is that there's a phenomenon called adipocyte inflammation. What this means is that fat tissue has cells called adipocytes and adipocytes create inflammatory chemicals, specifically IL-6 and TNF alpha. What they found is that the more body fat person has, the more inflammation they have and what they found links with this chronic inflammation and lung permeability.

Obesity and just having more body fat is going to cause the barrier to break down, diabetes, especially if the HbA1c levels are high, there's lots of glycation. Those lung tight junctions can break down, malignancies, cancers that cause oxidative stress throughout the body can cause these lung barriers to break down and just as we get older. Now what's really interesting is that when you look at the diseases that can cause these pulmonary barriers to break down there are also the risk factors for people that end up with complications with COVID-19. COVID-19 risk factors are smoking, diabetes, obesity, just being older, having underlying inflammatory or underlying condition. There's a possibility research will show. There's possibility that some of these patients that may be very susceptible and have chronic diseases and progression into things like anti-fossil with the syndrome and the claudin with Corona virus.

They have already some compromised lung permeability as part of the factor. But in any case, the key thing is that those are the risk factors. If you're a smoker, you're going to have leaky lung syndrome. If you're constantly in area, we have lots of air pollution, you can have leaky lung. By the way, they've also done some studies where they've looked at geographic studies, epidemiological studies, and found where there's the highest rates of severity of COVID-19 in populations and they found direct links with air pollution. I think there's some issues with permeability and just our potential to stay healthy in just overall and especially with this pandemic. Now how do you test for this? One of the questions people always ask is, well, how do I confirm I actually have leaky lung syndrome?



This is where we have a problem. In research studies, what they do is they do a bronchoscopy. They take a little scope and get some tissues of the alveoli and then check out the epithelium inside a microscope, and then they can determine pulmonary permeability. That's how they do it in research studies. That's not available for us to do in a real life setting yet. There is a clinical trial being done by the University of Rochester where they're doing something called an inhalation challenge test. They're using that to see if it's an effective test to measure permeability. They've already confirmed that it is a reliable marker with animal studies, but now they're getting into human clinical trials. What they're doing is they're doing a mannitol challenge, respiratory mannitol challenge.

Let me explain this. Long time ago, when they were looking for leaky gut, they used to do a test called the mannitol lactose test, but mannitol was a sugar and what people would do for this leaky gut test is they would drink this mannitol sugar, and mannitol is a large sugar. It's a disaccharide. It's pretty large. It shouldn't be able to get through the gut epithelium. What they would do is that a person consume mannitol and liquid drink, and then they would measure their urine afterwards a few hours later, and then determine if mannitol levels spiked up. If mannitol levels spiked up, that would indicate that they may have a malabsorption syndromes, or they also test the sugar called lactulose, which shouldn't get through the gut. If lactulose got through, then they would have leaky gut.

Mannitol it's very small sugar, it should be absorbed. But if they had malabsorption from inflammation in their gut, that wouldn't get through. They're doing the same thing with mannitol, but they're having people breathe it in and a test and then they're checking their urine levels. This is probably going to pass as they go through the clinical trials, look at the FDA approval. Laboratory tests are much easier to get through FDA than drugs. In the future, we might see leaky lung tests where people inhale mannitol and then check the urine and they can see if this sure is inhaled substance actually shows up in the air. If it does, then that could be assigned for leaky lung. Now, for now, we don't have any commercial available tests. What we're stuck with is this gap between knowing what the research is on leaky lung syndrome or pulmonary epithelium permeability, and no real available commercial testing to test for it.

We have to figure out symptoms that are associated with it. Again, the key thing is when you suspect this, if you feel an inflammatory response or immune response, when you get exposed to air pollution, cigarette smoke or just bad air quality. Now then the question comes up, how do you treat it? Well, the key thing is, first of all you have to reduce systemic inflammation. That's one of the key things. Remember the only reason the lung barrier or any other barrier in the body's going to break down is that if there's not enough antioxidants to deal with the stress of the inflammation, oxidative stress. One part of it could be to raise your antioxidant levels. Antioxidant instead have really been supportive lung epithelium barrier regeneration repair that have been published so far, or your typical ones, vitamin A, vitamin C, vitamin E, vitamin D and glutathione.



Glutathione can be raised by taking like a liposomal glutathione or things like N-acetylcysteine to raise, glutathione.

If your goal is to really repair your lung barrier, you want to take things like N-acetylcysteine or liposomal glutathione, vitamin A, vitamin C, vitamin E, vitamin D and just improve your antioxidant reserves. At the same time, you got to reduce your exposure to irritants to your lung barrier. You really won't need to improve your indoor air. It's surprising how poor indoor air is for many chronic patients. If you do have this or if you suspect you have this lung permeability issue, you should really get a HEPA filter, H-E-P-A filter. Those are the only filter that really clean the air well enough, so you can get decreased pollutants and irritants to the lung barrier. Then if you already have, you are being exposed to dust, or if you are being exposed to an environment that just isn't healthy, you should have a dust mask.

I mean the N95 masks are great, but we know we have some issues with that with the pandemic, but any kind of dust mask, especially a really good quality, N95 mask would be ideal to reduce this. Then many people have been using nebulized glutathione and nebulized glutathione requires a nebulizer and in most states and countries, you have to get a prescription for a nebulizer. The nebulizer will take a medication or anti-oxidant, that's a liquid, and then vaporize it into a gas and people wear a mask and then breathe in, and they breathe in glutathione. Those are the main strategies to try to support the barrier, the lung barrier itself. All right. I think that's kind of the baseline of the key concepts of leaky lung and lung barrier syndrome issues. I'm going to take some questions here.

My wife's going to help me, but also if you haven't already followed us on Facebook, you might want to follow us. We also have created a YouTube channel under my name. We post some of these old videos if you missed any of them in our YouTube channel, you can subscribe to that. I write new information articles all the time on my website called Dr. K news, drknews.com. Thank you everyone for joining me. I see people from all over, from UK, Australia, from Florida, from Canada, from all over the US. Thank you everyone for taking the time to join. Let's get into some questions.

Hi.

Hi.

[Esitta 00:22:57] asks, what does uncoupled mitochondria mean?

Sorry. I didn't explain that very well. Uncoupled mitochondria, what does that mean? Well, you have the factory of energy and all of your cells called mitochondria and what they produce is something called ATP and ATP is basically the fuel for all your cells. There are certain physiological mechanisms that makes this energy powerhouse not work, and that's called mitochondrial uncoupling. One of the things that causes this energy powerhouse of the cell to not work is chronic inflammation. Inflammation starts



to impact what are these different enzymes in this powerhouse of the cell called mitochondria called complex enzymes, and then these become less efficient and the body makes less cell fuel called ATP.

Chronic inflammation has been known to cause what they call mitochondrial uncoupling, which basically means that chronic inflammation shuts down your body's ability to make energy. It's very common with people that have chronic diseases. Most people that have chronic inflammatory diseases, they just don't recover. They feel tired. They can't exercise. If they exercise, they can't perform well with it. Once they get through their exercise, it's really, really hard to go back and recover from it. Those are all clinical signs of mitochondrial uncoupling.

Nancy asks, how do you lower your eosinophils?

Well, if you have high eosinophils, eosinophils are immune specific cells. When you ever do have a fever for those of you that don't know what eosinophils are. If you ever had a blood test, they measure your white blood cell count. They'll test different immune cells and one of them are eosinophils and eosinophils are triggered by very specific IgE antibody response, and then a cytokine called IL-4. When eosinophils are elevated, you have to look for basically actual allergies, not sensitivities, and they could be environmental. They could be different types of pollen in the air or different types of airborne triggers that you can have an allergic reaction to, to raise your eosinophils.

There's also research that shows parasitic infections activate the same pathway and they can raise the eosinophils. There are also some really common chronic inflammatory bowel diseases that can raise eosinophils or other lung and bowel disease that can raise eosinophils. At the end of the day, it just means that you have either a significant inflammatory response or some kind of allergy or some kind of parasitic infection. You have to find out what that is. You can't just treat [inaudible 00:25:35] eosinophils with a supplement. You got to find the cause of it, and then address that to reduce your eosinophils.

Meredith asks does mold exposure cause leaky lungs.

Yes. Mold exposure can absolutely cause leaky lung. It would just be another inflammatory compound. We did a talk on mold. One of the key things to remember about mold is that mold produces what are called mycotoxins. These mycotoxins are released into the air and we breathe them. These mycotoxins are severely inflammatory and they will cause irritation and breakdown of tight junction, proteins, and cause alveoli inflammation? If you're in a chronically mold or mycotoxin rich environment, like you're breathing in all the time, I just think it's a matter of time before the lung barriers breaks down. It really depends on how healthy your immune system is and how your health and your antioxidant systems are. For some people, if those systems are already



compromised. They can have severe reactions to mold and for some people, those systems are intact.

Even though they're getting mycotoxin exposure, the antioxidant repair of their lung barrier is so fast, and their secretory IgA cells that are these immune cells that just take up the mycotoxin and prevent its destruction on tissues. If those are intact, you're not going to have any symptoms. This is why some people could live in a house and be exposed to mold, and let's say one person in the family is like, I don't know what you're complaining about. I feel fine. Then someone else has a significant issue with it and they feel sick. It's not just the mold exposure. One of the other key factor, is how your body responds to the mold or mycotoxin exposure. The lung barrier is one of the key factors there.

Julie asks, why might one react to glutathione?

There are some people who don't do well with glutathione and the key reaction there is usually with the sulfur portion of glutathione. Some people are just intolerant to sulfur. If you're intolerant to sulfur you're not going to be able to take N-acetylcysteine or glutathione. You're going to have to use things like vitamin C, selenium to really raise your glutathione levels or just use any other antioxidants.

John asks, what kinds of diet or foods will exacerbate leaky lungs and which ones will improve and get rid of leaky lungs?

What type of diet and foods will help heal leaky lung and help cause it to break or promote its breakdown? Well, I think the key thing is just inflammation. If the tight's inflammatory that's where the risk is. For example, some people are extremely sensitive to milk protein, and some people are not. If a person is sensitive to milk protein, they get a systemic inflammatory response that's going to use up their antioxidant reserves. That's going to potentially lower their SIgA count throughout their body. Their severe reaction to milk protein may be a big issue for their lung barrier to break down, for someone else who doesn't have that severe reaction, it may not be as bad. Same thing could be happening with people that have nightshade sensitivity or reaction to lectin proteins. Technically foods like lectins, like peas and corn and eggplant, tomato.

These things are considered healthy vegetables and foods, but for some people they're not because they have a significant inflammatory response. The key thing is whatever diet has an anti-inflammatory effect on them is probably the best way to support the lung barrier, but any diet that promotes inflammation based on their own individual unique reactions to food proteins would be a factor in causing the lung barrier to break down. Simply by just reducing antioxidant reserves and causing systemic inflammation. The key concept also is when a person has inflammation, we all have some inflammation. But if the inflammation in our body is severe throughout our body and we're dealing with a chronic inflammatory condition or disease for example, like celiac



disease or autoimmune disease or inflammatory bowel disease or whatever type of inflammatory condition. All barriers have a chance for greater permeability, whether it's the nasopharynx barrier in our nose or blood-brain barrier or a gut barrier or a lung barrier. Inflammation in the body generally will be the key risk factor for causing all barriers to break down. The diet you want to choose is one, that's not inflammatory for that individual person.

Lynn asks, can you speak to the optimal levels of antioxidants? What if, is the optimal level of antioxidants?

It's really hard to answer that question, but what are the optimal levels of antioxidants because which antioxidant, which tissue, how much? You're measuring in blood reflective what's happening in tissue? We don't know. Some people have reference ranges for things like vitamin A and vitamin E on red blood cell testing, when they test micronutrients. Some people look at some labs like the ratios between oxides reduced glutathione to get a general clue about antioxidants. But those are really not really clear indications. To be quite honest, the amount of antioxidants you need, well, you definitely want to eat super foods that are really high in antioxidants. Very colorful fruits, colorful vegetables are really high in antioxidant content you really want to consume a lot of those.

Then you may want to just take antioxidants things like resveratrol, turmeric just as a preventative thing. I think for most people if cost is not an issue and they're like pro health and pro supplements, taking a variety of things like turmeric, resveratrol, pomegranate green tea really provide really rich source of antioxidants. How much do you need? Then the main issue always becomes cost versus benefit. You're going to figure out how much do you take and how much does it cost you if you're taking exogenous supply, if you're taking oral supplements and that's basically where most people's decision comes in. We don't really have a really good way of testing. We're dealing with cost benefit issues all the time.

Now in the clinical settings, sometimes when people have chronic inflammation, we'll raise up their glutathione or their resveratrol or green tea or whatever antioxidants we're using until they see a benefit. For some patients, they're going to tell you, I have to take this amount of green tea extract every single morning in order to feel better. For some people they don't even respond to green tea extract, they'll respond to resveratrol. There is some, in a real world trial and error and playing around with different antioxidants. That's the best way I can answer your question.

Susan asks, what is the best way to access the SIgA status?

SIgA is called secretory IgA, I mentioned it a few times. Secretory IgA is in immunoglobulin like an antibody, but it circulates on the surface of the gut barrier. It surfaces on the nasopharynx barrier and in the lung barrier. What these SIgA



immunoglobulins do is when a pathogen or Pluton comes in, they just surround it. When they engulf it, they neutralize it from reacting against the barrier. Now that can be measured in blood. It can be measured in saliva and it can be measured in stool, but how you measure it really reflects on which target tissue you're trying to look at.

I think for the lung epithelium and the gut epithelium, saliva SIgA is a very accurate way to measure it and most the many labs offer that test. For general immune compromise, you're going to see the serum SIgA levels drop. Stool SIgA is probably just best when you're trying to look at the gut epithelium, the gastrointestinal epithelium, not so much for the lung epithelium. I would say saliva would be a really good way. Saliva secretory IGA, it's a really good way to look at that SIgA count. If you do have some signs and patterns of lung permeability, you can use that test to see if that's improving or not with you.

Laura asks, how do you increase secretory IgA levels that are low?

The way you can increase secretory IgA levels when they're low, is the same thing as we talked about. Taking high amounts of antioxidants, reducing inflammatory load and making sure your immune system is healthier, getting plenty of sleep and rest are key patterns. Secretory IgA have been linked to stress and psychological stress has been shown to rapidly deplete SIgA levels. The psycho neuro immune response is found to be very significant with SIgA levels. Basically it's really no different than anything else. Reduce your stress, get plenty of sleep, get tons of antioxidants, get your immune system as healthy as you can. Those are ways to raise your SIgA.

I do want to point out something, there are many, many laboratories that are doing SIgA incorrectly. If you're getting SIgA levels measured with a lab test, that's also measuring your hormones like an adrenal cortisol profile or a female hormone profile. Unless they gave you a separate vial for SIgA, that's going to be a test that's not accurate. You have to have different reagents to stabilize SIgA than what's used for hormone testing. Be aware of that. If you're getting your SIgA levels measured when you get a hormone test in the same file you probably did not get an accurate level, correctly measured.

[Dreidre 00:35:30] asks, if you have multiple chemical sensitivities, does that mean you have leaky lung?

If you have multiple chemical sensitivities does that mean you have leaky lung? No, it's possible. When you have multiple chemical sensitivities, that's what's referred to as loss of chemical tolerance. When you lose chemical tolerance, there's lots of things that can happen. One of the mechanism is that the barrier can break down. That could be your lung barrier, could be your gut barrier, your blood-brain barrier, your nasopharynx barrier. Another part of it is you can have what are called dendritic cells that overreact or T reg cells that dysfunction. There's a whole list of different immune mechanisms that are involved from having you to have an overreaction to chemicals. I did create an



online program called the 3D Immune Tolerance Protocol, the 3D stands for de-pacify, distinguish and down regulate. If you go to Dr. K News, drknews.com. You can enroll in that course, and it goes through all the different mechanisms of physiology of how you lose tolerance and how people develop either food or chemical sensitivity. I think you'll find that information really useful to you if you're suffering from chemical sensitivity,

Michael asks what is better or more preferred test CRP or homocysteine? What is the preferred? Which is a better or more preferred test?

Which is a better or preferred test, CRP or homocysteine? Well they're measuring different things. There isn't one that's better or preferred. CRP is going to measure just a general marker for systemic inflammation. What happens with C-reactive protein is when you have inflammation, your immune cells produce called cytokines. The cytokines then trigger immune responses to cause inflammation. But those cytokines end up in the liver and they get metabolized to an end product by the liver called C-reactive protein. That's what they measure. The key thing you need to know about that is that not all inflammatory response is going to go through that pathway. Many people have systemic and chronic inflammation, and this therapy levels may be fine. It's not a test to measure all inflammation in the body. It's just one marker.

Homocysteine is a complete different mechanism. Homocysteine isn't measuring these end products, these cytokine proteins homocysteine is measuring the body's ability to methylate. Homocysteine is an inflammatory chemical that our body makes just normally and eventually gets converted to cysteine and cystine, which are very important for many metabolic processes in our body. In the conversion of homocysteine, that's inflammatory to cysteine that's not requires a transfer of a one carbon group. They call a methyl group. They call that a methylation pathway. When you're trying to see if someone has a methylation problem, or where they need things like folate or B-12 that's where you would measure your homocysteine. They're totally different tests.

The person who asked you about how to test for SIgA, she, and a few other people are saying, that's exactly how they got the results from the thing, there'll be a test. Then the follow up is, any labs you recommend or how do you test [inaudible 00:38:39]?

Because if you had your hormone levels measured through a lab that was doing your hormones, let me guess your results. It was low. Let me guess your followup. It's still low. Let me get your next follow-up. It's still low. This is why I get so upset with some of these functional medicine labs that just, I don't know what they're doing. Test with Cyrex, Cyrex labs, saliva, SIgA. Get a reliable test. They actually use reagents to measure your chemical. That's the one I use. There could be other labs that are doing it properly, but a lot of these hormone labs are just throwing that in to make their profiles look better. You're going to be like so frustrated because you're going to see it low. You're never going to see it go up and you're not actually getting an accurate test. If



people in the lab are listening to this, wake up, read the literature, create the right reagents and make your panels appropriate for actually measuring SIgA.

Okay.

It's frustrating for everybody.

I know. I'm glad you said that. I'm so sorry. Michael, who had asked you about that CRP versus homocysteine question? He said it was for systemic inflammation.

For systemic inflammation, again, CRP, they're both part of systemic inflammation. When you have systemic inflammation, looking for all different mechanisms and triggers. CRP can be a useful tool. The most common test people are using for systemic inflammation would be CRP or ESR, erythrocyte sedimentation rate. You can also see markers like ferritin go up. These are called acute phase reactants. They just go up in the inflammatory response.

If you were just screening for systemic inflammation as a biomarker, I would check CRP. I would check ESR and I would check ferritin. Then once I find one of those high, that would be the baseline I would use with someone. Then I would do follow-ups to see if those levels are coming down. You might not see your CRP high, but you might see your ESR high. You may not see your ESR, but you might see your ferritin high. What I typically do is those three screens, and then once I get a baseline, which inflammatory pathway, I'm seeing some changes in, I can use that to compare them over time with any kind of protocol or intervention.

Bob is asking, is SIgA different from IgA for example?

Yes. SIgA is different than IGg?

IgA.

I don't know what IgA is.

It might be a typo.

It might be a typo. There's different immunoglobulins, there's IgA, IgG, IgM, IgA, and they all have different immune responses. So IgG is typically involved with your classical antibody response when you're reacting to a food protein or a chemical that's delayed. IgA is really the immunoglobulin that's in your barrier is trapping different food protocols and pathogens to dampen the immune reaction to it. I'm generalizing. There's a lot more, but ...

Someone's asking you, if you can do a talk on histamine.

I can do a talk-

Histamine intolerance, a video on histamine intolerance.



Yeah. We can do one. We have a list we're trying to go through. Right now we've been trying to cover the barrier. Last time we covered the blood-brain barrier. Then today we're talking about the lung barrier, and then next time we're going to talk about the gut barrier and really talk about all the current research and talk about how it's not as simple as just doing a leaky gut protocol. But thank you for bringing that up.

Nancy asks Dr. K, Do genetic tests help with designing the anti-inflammation diet? If so, do you have one you recommend?

Genetic testing has a long way to go. Does genetic testing really help to calm down-
Design.

Design.

Help design the anti-inflammation diet.

Design the anti-inflammation diet and if so.

If so which do you like to use?

The problem with genetic testing is they're measuring individuals, what they call SNPs, single-nucleotide polymorphisms. The ones that the labs are testing are really the ones that are commercially available and cheap to test that, they're not testing all of them. It's not like strategic, where they fared out, which SNPs to measure for everything and they're measuring all of them. They don't have the ability to do that. Labs, know there's thousands of these SNPs. In the reality of genetic testing, you're going to measure all your SNPs, not just a few. They're going to do machine learning to see how these things work, which you're still understanding. Then once they have those SNPs, then they can really find some protocols. Now that being said, there are some single-nucleotide polymorphisms that do show evidence that by themselves, they are an issue. For example, MTHFR methylenetetrahydrofolate reductase polymer, the inability to methylate has been shown some strong researcher.

That individual SNP by itself could be a factor for like B12 or folate. But when you see some of these labs, like breaking down, just like one SNP for glutathione and one SNP for a sulfation pathway, that is literally a joke. That is basically once again lab people don't know what they're doing. They're trying to make a commercial test that doesn't really clinically answer the reality of the science behind it. With the exception of some COMT SNPs and MTHFR SNPs some of the things such with AP proteins we don't have enough information to know what those other ones are. I don't personally do a lot of SNP testing. I find the panels that they're doing are very, very limited. I will test things like MTHFR or CMT or the AP proteins, just as a common test. But I don't do these full profiles where they measure SNPs and then customize a nutritional protocol. I think those are inefficient inaccurate ways of doing it. That's the best I can do to answer your question.



Sean asks [inaudible 00:44:32] Sean asked, what are you recommending as preventative measures for maintaining healthy lung barrier, especially during the COVID crisis, any particular supplements?

I think for the lung barrier in COVID I think it's the same thing as the antioxidants, glutathione keeps popping out all over the place. There's a brilliant summary review paper in articles published by some of the researchers that one of the Russian universities. Where they broke down all the science behind how important glutathione is for maintaining lung barrier and its potential role of people suffering severe reactions to COVID-19 versus not the theoretical, glutathione play a role. I would agree with that could be part of the picture. We don't know, it's just opinion. But the key things is basically high anti-oxidants ACD, glutathione, clean indoor air, plenty of sleep, plenty of rest, healthy immune system. There's no real major secret or breakthrough that we know about so far.

Carolyn does low, very low nitric oxide pose a problem to causing all the things you were talking about? My whole body is full on inflammation.

Yeah. Low nitric oxide, and we're talking about, there's different types of nitric oxide. There is endothelial nitric oxide. There is neuronal nitric oxide. There's cytokine induced electron. There's different types of nitric oxides, but you're probably referring to endothelial nitric oxide. Endothelial nitric oxide is important for blood flow, vascular dilation and circulation. That will definitely impact all your barriers from healing because you got to have healthy amounts of vascular dilation and blood flow to be able to heal and repair. In the literature, what they found, that things that can really increase nitric oxide. One of them is arginine. I personally, don't like to use arginine because arginine also activates the inflammatory isomer or branch of nitric oxide, but beet extract has been shown to very profoundly raise nitric oxide levels. Hawthorn extract as a botanical and vinpocetine. So beet extract, hawthorn extract, vinpocetine are all botanicals that can help with raising nitric oxide and also improving blood flow and circulation which may be things you may want to consider.

Jodi asks, how about your thoughts on astragalus?

I'm not aware of astragalus being a very powerful nitric oxide stimulator.

In life astragalus?

The key mechanism of astragalus is, well, at least the ones published they're probably many of my criticisms for astragalus, but the main one is that it helps activate nitro killer cells. It's a very good botanical to really improve your immune system. The different parts of the immune system, the cytotoxic T-cell part where you actually kill infections and pathogens would be where astragalus would really come in.



Back to testing again, Susan says I'm actually tested low on SIgA with a stool test and was curious whether to also test saliva and, or redo the stool test? I have IBS Hashimoto's, cigarette smoke really bothers me. Taking the saliva test ... Carry on.

If you had a stool SIgA test that's low that's probably going to be a very good reflection of what's happening in your gut. It may not be the best reflection of what's happening in your lungs. I would just say, we might want to do a saliva SIgA test also to determine if that's happening or you may just want to treat your gut for a while, get the inflammation down in your body and then go from there. Because having gut inflammation and a low SIgA cell count in your gut means you're dealing with some immune challenges in your gastrointestinal microbiome. You may want to get those under control.

People are asking, if you can do a talk on methylation and mast cells.

Okay.

Debbie, when would you not do intermittent fasting?

When would you not do intermittent fasting? Well there's a lot of reasons you wouldn't do it. You don't like to do it. Well, intermittent fasting for sure is so much great research, more and more coming all the time of how it helps protect the brain, how it helps with controlling blood sugar and diabetes, how helps calm inflammation. For all those reasons, I mean, there's a lack of evidence that it can be beneficial to many people. The issue is there are some people who can't jump into intermittent fasting. I think that's the key problem. If they tried intermittent fasting, they get severe hypoglycemic. They fall apart and they're not actually able to do it. They have to be carried through that process. People that have significant high insulin levels like they're insulin resistant are going to have a really hard time to, for example, jumping into it because their insulin levels shut down a pathway that's called lipolysis breaking down fat.

Since the insulin is high throughout most of the day, because they have may be too much sugar and too many carbohydrates and everything. Their pathways to break down fat, this lipolysis pathway is inefficient. When you go into a fasting state, you're trying to get into ketosis, inactivate that system and it just may not be there for them. A lot of times if people can't, I mean, if they want to do intermittent fasting and they can't do it, I'll have them first cut off sugar. That'll start calming down their insulin. That's a quite good place to start. Then get into ketogenic and ketosis for a while, and then eventually have that transition to intermittent fasting.

But if you look up, what are the health benefits of fasting? There's a lot of health benefits there. Everyone should be doing it just like everyone should be exercising and everyone should be sleeping and everyone should have healthy social relationships. The research is there. It's just, do you do it and are people willing enough to make that sacrifice just like, are people willing enough to wake up every day and go exercise? It's hard to do, but it has a lot of benefits.



Brandon asks, I have read studies on ashwagandha and olive leaf extract being good for mast cell stabilizers. Any experience with this? A lot of mast cells questions.

Yeah. A lot of mast cells questions. Olive leaf extract and ...

Ashwagandha.

Ashwagandha, well, they both have anti-inflammatory properties, whether it's a mast cell activation syndrome or histamine response, they're going to have some benefits. I personally have not used those two compounds for like a mast cell response. I can't comment on it. My guess would be that it may be beneficial, but I haven't researched papers and read papers on it and haven't looked for those papers and have no experience with it. I can't answer it for you. I'm going to say, I don't know. My guess is they're probably being useful.

Okay, good. Let me see, Tammy asks ideas to reduce mucus in the lungs and throat from asthma?

Well, if you have mucus in the lungs from asthma, then that mucus is being produced by an inflammatory response. You got to reduce the inflammatory triggers. Everything we talked about for lung epithelium barrier applies. Having a healthy HEPA health outfitter, taking lots of antioxidants for a good place. Making sure you're wearing a dust mask if you're around a lot of dust or air pollutants, maybe even nebulized glutathione may be useful, but you got to find the main trigger. Mucus is an inflammatory response. Something is triggering the inflammation and it also possibly of infections too. You have to look at the big picture and try to strip the immune system. The general concept is, is there's going to be some trial ... In a real clinical setting there's some trial and error. There's some testing. You see what the person responds to you. You try to find as many things to remove and add, and it may take some time to figure out, but that's the only way to do it.

Seth Sinclair says, how do you test endothelial nitric oxide levels?

There's some controversy between this. There's some labs that can measure some of these in serum. How accurate and reliable they are is still being tested. I would say I don't feel too confident about testing them. I don't use in my office, but there are people that I know that I do the testing that I think are pretty smart, so I don't know what to do.

Let's see. I think that might be it.

That's it.

Let me just do quickly. Jonah, can low stomach acid cause leaky lungs, if so, will taking HCl fix this [inaudible 00:53:19]?



Low stomach acid isn't going to directly cause leaky lung. Low stomach acid can cause you to not digest your proteins and have gastro reflux and if undigested proteins and leaky gut, maybe that causes inflammation promotes leaky lung. But lack of ACL itself is not going to be major. It's not going to be direct cause of leaky lung syndrome.

Let me feel like really at the moment, because some skipped up. **Richard asks, how do leaky lungs slash gut affect someone with giant cell arteritis, how do you calm that flare?**

Well, if you have giant cell arteritis, how does leaky lung and leaky gut impact it?

Yet affect it.

Well, I mean, giant cell arteritis, you're looking at the most common cause being an auto-immune response to vascular target proteins. Anything you can do to calm down your hyperactive immune response or systemic inflammation would be a good idea. Just like any other autoimmune disease, the barriers are important, antioxidants are important, whatever you can do to work to dampen the autoimmune reaction. In those cases, you really might want to work with a skilled functional medicine practitioner to help you guide you through maybe an autoimmune diet and lifestyle to calm down that potential of inflammatory trigger.

Okay. Go ahead.

Just phospholipid auto-immune disorder that it's pretty serious.

Other people are just saying thank you [inaudible 00:54:56].

All right. Well thank you everyone for joining me. I hope you find some information useful. Thanks again and please follow us on Facebook and our YouTube channel and check out Dr. K News and our newsletter there. Thanks again.