Introduction and Dr. McCannu2019s Background

Welcome to another episode of Mycotoxins, Mold, and Chronic Illness Summit. I'm your host, Dr. Ann Shippy. Today we get to talk with Dr. Kelly McCann. She has a dual residency in Internal medicine and pediatrics.

She also did a fellowship in integrative medicine, and she holds multiple certifications. She also sits on two very important boards, the Board of American Academy of Environmental Medicine and the board of the Internal Society.

This is a mouthful environmentally acquired on us, which is amazing. And she's the host of Two Summits. Hi, Kelly. It's so great to have you. Hi. Ann, thank you for inviting me.

So excited about this conversation. We got to talk a few days ago and compare some notes and came up with this topic. And I think it's going to be one of the most important ones of the summit.

And I just you know, I was reflecting back about doing this with you. And I remember seven or eight years ago you were giving a presentation, I think it was at the American Academy of Environmental Medicine meeting.

And you did one of the best lectures I've ever heard, and I've heard a lot on specifically the way that you did your research to pull together really valid information and then make it so that people could understand it because it was like there were a lot of people in the audience who really didn't know much about Phosphatidylcholine And then brought it to a clinical level and then made it where we could take action on it.

You're such a rock star and how you think about medicine and applying these new territories to really help patients is just, I just love you so much. Oh, thank you. Thanks, Ann.

I really appreciate you, too. And thank you for the opportunity to speak and share some of the things that I've been excited about so far, though. Yeah, and we're both we both had our our run through with with mold.

If you want to just tell a little bit about your background and anything else that you want the audience to know about before we dive in so that they can really know who they're talking to.

Okay. All right. Big picture. I, too, am mold survivor, right? Mm hmm. In the bedroom there that I grew up in, in high school was in the basement in upstate New York.

I know it was moldy. We had a dehumidifier running 24/7 so definitely mold exposure there. Fortunately, I didn't really have any major illnesses. It was just kind of allergies that that affected me at that point.

Fast forward in my first job out of residency, I was in Oregon, in western Oregon, and my office was a flat roof building and I'm sure there was mold there started to develop fatigue and depression.

Felt like fibromyalgia at the time, didn't know what was happening at all, but managed to move on from that job and do something else recovered and then probably got exposed to mold several other times and then big mold exposure a number of years ago.

Now, I remember talking to you the day that my movers were coming. I was 16 and you're saying, Kelly, you got to get rid of everything. You've got to put stuff in storage.

And and that's what I did. So I got rid of nearly everything. I put many of my belongings in storage, hoping that I could try and salvage them. Just threw out a ton, moved into a new home.

I had to strip down from the clothes that I had used to move, threw them outside, took a shower, put on brand new clothes that I had picked up at, you know, grass or Target or something like that.

And I started rebuilding my life from scratch. I had literally nothing and it turned out that all the things that I had put in storage I tried to salvage a year later and it was impossible.

I was symptomatic trying to bring those things into the new house. And actually my husband was too. We have the same symptoms. We were both twitchy when we were exposed to mold and threw them out again.

And even last year I had another mold exposure. So I feel you folks, I understand. And one of the things that I learned is not only to how to deal with stuff, but also how to help get people better.

Somehow I managed to work through all of those mold exposures and keep my brain and my wits about me, mostly thanks to Phosphatidylcholine, which is just a game changer.

Magic.

Mold Exposure and Phosphatidylcholine Recovery

For those of you who don't know about it, it is the building block of cell membranes and so any mycotoxins, any toxins in general that might be impacting your cellular health, impacting your mitochondrial health because mitochondria are made with lots of cell membranes, right?

Lots of membrane. The mycotoxins, the toxins can be mobilized out of this of the membranes and you can heal. So but with my little plug for PC. Yeah, that's my like if I could pick one thing on a desert island, one supplement I have dropped into me, it would be.

Easy. Because I know it's part of why I was able to work through the day. So health challenges as well. And I think it really helps to be able to and we're so lucky that we get to help people every day because it keeps you focused on that.

And less on on what you're going through at the time helps you to make that jump to take it. Well. Absolutely. So that's my mold history. I don't know if you have more questions or you want me to answer more.

Oh, well, I'm sure we'll we'll have some more things to weave in. But I love this topic that we're going to talk about today, hypercoagulability and and biofilms and how all this fits in together with the other things that we need to address to help people to get well.

So let's start with hypercoagulability, which just means well you you go ahead and define it how you see it. Sure. Sure. It's a mouthful. And essentially it means increased clotting.

So if you remember at all from biology, there are two cascades that happen. On the one side, you've got what we call the clotting cascade or the coagulation cascade, and there are multiple different proteins and they cleave and cut and make other proteins and that forms the building blocks of a clot.

The molecules of the building blocks are called fibrin. And then on the other side you have fibrinalysis, right? So we got fibrin sticking together and then fibrinalysis or breakdown of clots.

And if you think about it very simply, if you cut yourself, you want your blood to clot to stop the bleeding. And then once that cut is healed, you want the body to break down the clot so the blood flow can resume its normal way of flowing.

And in people who have a genetic predisposition to making more clots, you can either have a problem on the clotting side or the breakdown of clotting.

And if you make too much clot, you'll have what I call sticky blood. I haven't come up with a better term for it. I think that's a great term because imagine it like, Oh, exactly.

It's a little squeeze. Moving through the capillary beds and getting better needs to go. It's kind of smudging. Yeah, a little sludgy, sticky and the same thing.

If you're not breaking down the clot adequately enough, you're going to get that same problem of the sticky blood. And and so that's coagulation. Yes. And so if you have too much hypercoagulability, things just really don't work right.

And then you get increased risk for heart attacks, stroke, pulmonary embolism, DVT, all those kinds of things. And we usually pick them up when they're to that extreme.

But I think what we're figuring out, especially with all this experience we had over COVID where people got hypercoagulable from COVID, is that it's a spectrum, right?

Like you can be you can have this sticky blood clot kind of clogging up the capillary beds and your organs without actually, you know, having had it be life threatening with a heart attack stroke or pulmonary embolism.

So and I think, you know, a lot of us that were involved in treating mold early were more aware of the where the they were actually bleeding. So we were on the lookout for that, but we weren't thinking, oh, well, okay, when this whole pathways in disarray, we might have things on the other side.

So what are you using to try to figure out if your patients are in this spectrum of hypercoagulability? It's a good question. So I think the other piece that I eventually want to weave in that biofilm, but will it will stick with clotting for now.

Most of the time in conventional medicine, we don't workup somebody is a risk for clotting until after they've had a couple of clots or miscarriages, for example.

So miscarriages, increase clots, risk can increase your risk for miscarriages. So we evaluate that. Very important to know about. And I think that some of the some of the genes that we know about one is called factor V Leiden.

There's a prothrombin2 gene mutation and those are some of the most common ones, common ones, according to the literature and to hematologists. Then there are some other ones protein C protein S deficiency, antithrombin3 deficiencies.

You can also get a increased clotting risk, not genetically, but because of an autoimmune condition. So there's an autoimmune condition loosely associated with lupus called Antiphospholipid antibody syndrome.

And if you think about that, that's phospholipid. And we we started talking about a phosphatidylcholine, which is also a phospholipid. So there's a relationship with the cell membrane and it's not functioning properly.

When you're talking about an autoimmune condition that increases the clotting risk. And and so there's a couple different things besides genetics that will increase your clotting risk.

And the big concern in conventional medicine is to actually form a clot like an we're saying, the pulmonary embolism or the deep vein thrombosis. And that tends to be more associated with things like factor five iron.

And as with everything in environmental medicine, the genes load the gun, the environment fires the trigger. So not everyone with a genetic predisposition is going to have a clot.

There has to be some sort of inciting event, which could be a surgery, a trauma, prolonged immobilization.

Understanding Hypercoagulability and Clotting Risk

Sometimes people have heard about it where you take a long car ride or a long plane ride and you don't know that you have this genetic predisposition and all of a sudden you have a school and leg.

One leg gets one, you get a DVT. And now you know, now you're off to the races with your clotting disorder and what we know from the Lyme world, because I also see a lot of chronic Lyme patients, biofilms and clotting are a huge component to illness for people.

And I'm not sure if it's the infection itself that is triggering that inflammatory cascade and leading to an increased stickiness of the blood in a genetically susceptible person all the time.

Or or if it's just the nature of the bacteria wanting to help itself while off and protect itself in that clotting risk. But we know from Lyme that HyperCard regular ability is an issue.

And it's probably a combination of both, right, With this signature. Yes. That says it's and again, I mean, COVID has been such a teacher for us. Exactly.

Exactly. And then more recently, I learned about another genetic predisposition called Plasminogen activator or inhibitor one or P1. I call it PI one.

And it turns out, you know, all of the other ones that all the other genes that we're talking about, those are mostly on the coagulation side. But Pi one is on the breakdown of the clot side.

I've been testing my patients for the past six, 6 to 12 months now and out of all the people that I've tested, probably 90 plus percent have pai one. Oh, my gosh, this. Is.

Ground breaking. I hope you're going to publish this. This is amazing. That's the intention. So when you go into the medical literature, PI one is thought to be very rare.

You know, of course something is rare if you never test for it. So it's very you know, when we say that something is rare, we don't ever test for it. But, you know, historically hematologists have said that pi one is as rare and it does have this association with increased risk for diabetes and cardiovascular disease.

In the medical literature. But I think that nobody's actually really looking at this. And and so I'm going to look at it. You know, I've learned about it from my mentor, a woman named Ruth, Chris K. R., I Z.

She's a retired nurse practitioner and just amazing woman who taught who has taught me about coagulation and biofilms. But she used to check the pi one activity so you can check an activity in a lab and see if it's actually increase activity.

And then if there's increased activity, she would defer and then go check the genes. The only people I see who have PI one activity increased have insulin resistance and diabetes.

So the PI one activity is not necessarily associated with the genetics. And some of the people who have elevated PI one activity have normal genes, they just have really bad metabolic dysfunction.

So there is not a correlation that she was assuming and doing this work. And so now we're really excited to look at a patient population of chronic, you know, have a chronic illness patient population for the most part.

And the fact that so many people have this gene variant, myself included, I'm homozygous for. This test through like a core. Quest absolute, which. Usually covers it just like they do the antiphospholipid antibodies and factor V and all of that.

Yeah, as long as you're using appropriate ICD ten codes typically. So what? So, you know, I'm not checking this in everybody all the time, but I'm looking at the pattern of illness.

I'm looking at things like fibrin, fibrinogen activity. Yeah. And then there's another screening test called prothrombin fragment one plus two or 1.2 depending upon if it's LabCorp Quest, same same marker.

And that's really probably the best test for that stickiness of the blood. So if somebody is prothrombin fragment is normal, they may have a genetic predisposition, but they're not yet.

Activated. Activated into that sticky blood. You know, this is one of those things where I think we're going to probably be testing babies before they're born or when they're born and have this whole scan of things and know what to do to prevent illness like I do.

I'm so lucky. I do have a segment of my population of patients that are like, Tell me what to do. I want to stay healthy and healthy now. So, you know, I've got both, both populations, but and then so many people that they want their kids to like know what they can be doing from day one or early on to be healthy.

And I think this is going to end up being a really important one. And I bet, you know, the people who ended up being hospitalized for COVID that this could it's like if they were on the right.

Sure. Yeah, I'm sure it was huge. The infection. This might have prevented the hospitalization, right? Yes. And certainly the micro clots, you know, so we knew that there was an association with obesity, with diabetes and potentially an activation of pi, one that would increase the risk for clots.

You know what I'm what I'm finding and what I'm looking for now is I think that the PI one gene variant has probably a component of cardiovascular disease and cerebrovascular disease, much more so than a DVT, because I just think I don't know that that's my intuition.

And you know what? We'll see. We'll see what we need, our research. Centers that we could just go to town. On these things. Right, Exactly. Exactly. So in anyone who has a family history of cardiovascular disease, cerebrovascular disease, we can do this evaluation.

And anyone who has a family history of cancer, we know that cancer can trigger hyperactive ability. So those people should be evaluated. And then anyone with a chronic illness, chronic Lyme disease, you know, any sort of underlying inflammatory to basically you're talking about a huge broad spectrum.

Almost everybody should. Be part of our you know. It should be part of our screening. Workup screening. Just like we're looking at more of are we should be looking at AP gene variants to help people navigate their cholesterol management, their neurological health so ably.

For those of your listeners who don't know, that's the gene variant that's associated with increased risk for Alzheimer's and cardiovascular disease. So super important to understand your tactics.

Yeah. And which, you know, you have that gene. There are so many things that you can do to be on top of the prevention piece. So I know a lot of people are really hesitant to get their genes tested because they feel like it's pre determining.

But I love your analogy with the gun, right? The gene to the gun. But then there's the environmental trigger. So you can be more cognizant of those triggers and and other things that help to downregulate those pathways that are at risk.

That's really empowering. Absolutely. I mean, people understand that when they have a family history of diabetes, will they need to make different choices or, you know, for me, my mom has celiac disease.

So years ago I gave up gluten because I knew that my risk for developing celiac disease was very high, like 40%. Why would I do something that's going to increase my risk for developing an autoimmune condition?

I don't want. Oh, and now you know, you have the AP. I'm I actually believe I'm 33, so hopefully Gene is good.

Well, it's not good. It's out. Then you have the pie. But I got the pie one.

So yeah, I need to take some special enzyme in order to make that sticky blood not happen. And then do everything that I can to manage Lyme and mold and minimize my inflammatory cascade.

So you're right. And it also kind of helps you retrospectively to like, Oh, why? Why did I have these things happen in the past? And this is probably part of the biology, the physiology that was happening that made your system not be able to handle in the world.

Yes, that being a blessing too, because it's really better to not be in it even if you're not having symptoms because of the other risk factors that go along with that.

All right. You're doing awesome. You explain things so, so great. Okay. It's really simple. I have to keep it simple, right? I mean, honestly, these are things that most physicians would have no idea what you're talking about.

You know, they they did a little physiology. They have their basic labs that they run and they're not really going back. And integrating the two and coming up with these hypotheses about, okay, well, what's really happening here? What's the root cause of this?

Why is this happening in this particular person? And what can we do about it? So really great job of explaining things. So come back. To. Thank you. Yeah. Okay.

So what's what are the next steps then when you move. Right? So if you can get your practitioner to check at least fibrinogen activity and if it's over 300, then there's, there's some sort of inflammatory thing happening.

Right. Or if, you know, you have an elevated high sensitivity C-reactive protein, you definitely want to try and get this assessed and then that prothrombin fragment 1.2 if you're going to quest one plus two, if you're going to lab core, those are the those are the initial screening tests that I would at least ask for if they're normal doesn't mean you don't have the genetics.

It just means they might not have been triggered yet. Ideally, hopefully you'll find a practitioner who is listening to this talk and now you can collaborate and get your workup done.

But let's just say you have a family history of cardiovascular disease or and you're in a moldy situation. What can you do? What can you do? Some people reach for aspirin.

Aspirin affects platelets and it is not going to impact our coagulation or our fiber analysis cascade. So aspirin is not the go to solution for this particular situation.

Testing for Sticky Blood and Genetic Predisposition

Things that work better for this situation are what we call proteolytic enzymes or fiber analytic enzymes. So for people who don't have an elevated fibrinogen or prothrombin fragment, I tend to use narrow kinase because I don't think that they're actively clotting and will use the narrow kinase and and with narrow kinase or with any of these proteolytic enzymes, they have to be taken away from food and supplements.

That's super important because we want it to get to and kind of break down your sticky blood, not your food. So take them away. Usually a half an hour before or 90 minutes after you've eaten.

If you don't. It's a little tricky. If you have people doing binders and proteolytic enzymes, it's like. It's. Like a pill pill carrier for every time of day.

Yes, it's definitely tough. Yeah. You can't take it with your binders. They really have to be taken on their own. And sometimes, you know, ideally you're taking it once a day.

If you're not symptomatic twice a day, if you are symptomatic. So it does get a little tricky with all your binders. Do the best you can. And, you know, maybe intermittent fasting would make that easier because then you don't have to worry so much about the food piece of that.

So that's natto kinase nado is derived from soy. So some people are super soy sensitive, have to be aware of that. Least seem to have people with having problems with it etc..

No, but it's theoretical. Kind of like PC. I mean I, I find that people with soy allergies can usually tolerate that just fine as well. Do you think? Yeah, I do.

Technically in the PC, the PC, the body biopsy that I think we usually use, there is no soy protein and it's just the lipid component. So there's nothing that people typically get reactive to.

I just turn out there just in case I don't see it personally. There are a couple of other kinds of enzymes. Sarah Peptides is another enzyme that often gets used.

And then my favorite really is balloon or laurel kinase. I do find that the brand name Baloo works more effectively. That's what Ruth Chris recommends.

That's what other mentors who who have taught about hyper credibility for far longer than I have recommend. And I tend to reserve the book for the people who are more symptomatic or who have elevated levels of that prothrombin fragment.

And similar dosing. Similar dosing. We'll talk about biofilms at a second, but you can start with just one capsule once a day and then ideally you're ramping that up to get the the prothrombin fragment into a normal range.

Okay. So you follow labs on it again? Yes. That one you need to follow labs on. Okay. Technically it's not supposed to increase bleeding risk at all or and you know, some people complain about bruising.

I think bruising is tough when you're older anyway, and you don't have a lot of subcutaneous tissue and you've got little tiny arms like me, but or my mom or your.

Hyper mobile. Or your hyper mobile, you might get a little bit. Yeah, yeah. There might be a little bit extra bruising, but that's not supposed to be an issue.

It's not supposed to contribute to bleeding at all. Although I did have one patient and put her on balloon and she normally had, you know, two, three day cycles and all of a sudden she had a ten day bleeding cycle.

So, okay, you know, just FYI, I could could happen. You know. That also kind of makes you wonder a little bit about Endometrial biofilms. Okay. Yes. Very good. Price tag.

I going here as perfect. So it's a perfect segue way. So what are biofilms? I think biofilms are really amazing. There's a biofilms are what bacteria create when they live in a colony.

And there are lots of examples in nature of biofilms. So, you know, when you go look at that pond and there's like the scum on the top of the pond, that's an allergy.

Yeah, that's a biofilm. When you leave your glass and the water evaporates because it's sat for too long and the water evaporates and there's that grimy ring around that's like minerals, that's the that's a biofilm.

So there's bacteria that live in the in the water. They create a biofilm. And that that is the mineral deposited left by the biofilm. And anywhere in the body where we have bacteria.

Well, let's see our skin, our nasal passages, our mouth, gastrointestinal tract for females, the vagina, bladder, you're the bacterial biofilms in the bladder.

Now, we're not supposed to have bacteria in the bladder, but let me just say, in doing certain kind of tests, we're finding a lot of biofilm in the bladder.

So there's violence because it's it is part of you know, it's one of those surfaces that's really outside the body kind of protecting us from what's coming in.

So, of course, there's going to be organisms that help that mucosal lining to to try to stay intact and and keep the outside organisms that aren't supposed to get actually into our bodies in their places.

Right. Right. So so there is biofilms and and the way that I the way that I think about them, you know, they're kind of like this mucus aid structure.

There's minerals, there's mucus, polysaccharides. It's this goo. Another great analogy is when you see a little snail like the trail, that the snail leaves, that shiny stuff, that's biofilm.

So I hope you have an idea of what biofilm is. And they're everywhere in the bat bladder. Sorry, They're everywhere in the body and they're they're important.

So we can have kind of healthy biofilm we can have because they're healthy bacteria. So your microbiome in your mouth, if it's healthy, the microbiome in your gastrointestinal tract, those organisms are hopefully healthy and being protected by the biofilm.

But there are certain circumstances where the bacteria are pathologic, they're problematic. They can cause illness, inflammation, disease, and they're stuck in a biofilm.

So, for example, with Lyme disease, Bartonella, those organisms create biofilms in the body and it makes it a thousand times harder to kill some of those organisms.

There's was research done with her by a woman named Eva SAPI and she discovered that Lyme disease behind a biofilm is a thousand times more difficult to kill than Lyme disease without that biofilm.

So we often utilize things like blue Columbia kinase and these different proteolytic and fiber analytic enzymes to break down biofilms in Lyme and Lyme disease treatment.

And it turns out that in those people here's the here's the kicker, right? In those people who have this genetic predisposition to make clots, there is a fibrin from the blood that is being incorporated into the biofilm in these genetically susceptible people, strengthening the biofilm, making it more resilient and resistant to treatment.

Drumroll. Drumroll. And this is so important.

Treating Hypercoagulability with Enzymes

So so the so the people with this hyper cargo ability have more have stronger biofilms. Now, I don't know whose fault it is. I don't know if those if it's the bacteria that are like, give me that fibrin, I'm going to incorporate it into my biofilm.

Or if it's the body's way of trying to protect itself from these pathological organisms, I don't I don't know. We don't know intention, but the end result is that's what's happening clinically.

I don't I don't have the randomized double blind controlled trials yet to say that this is what's happening. I can say clinically this is what's happening.

I'll have to go do my my deep dive research and I'll get back to you. But you're, you're seeing and applying this information with your patients, which I'm so excited for you to share.

Yes. I'd love for you to talk about how you're evaluating the biofilms. Okay. So one of the symptoms that shows up in many, many of our mold exposed patients is chronic sinus issues, right?

They have allergies. They have sinus issues. They have breathing issues. They have upper respiratory issues. They might have headaches or migraines. And we know that there's a nasal biofilm.

And part of the reason I think part of the pathology of being in a mold the environment, is that you're inhaling those mold spores, you're inhaling those mycotoxins, you're inhaling whatever toxic bacterial soup is in the water, damage the buildings.

Exactly. So you're inhaling all that, and then they're setting up housekeeping in your biofilm, in your nose, and it makes it that much harder to treat.

But the problem with conventional medicine is they just kind of give you, you know, a zip pack or Augmentin or what have you for your science, your sinus infection.

And what really needs to happen is we need to identify what's actually in them in the microbiome that's pathologic and treat it right. And there are because most of the time most doctors don't even do swabs.

We're not looking for the organisms we're just assuming that it's the usual suspects that should be responsive to augmentin azithromycin. Oh, we don't actually know what they are.

And even if you're looking for Marc Carnes, that's just one organism in a biofilm. There's a lot of other organisms in the biofilm. And Markon for those of you who aren't familiar, that's the multi antibiotic resistant, correct negative stuff.

It's a kind of stuff. And I like that lab. I think they do good work. It's cool that they test for that biofilm, but I think there are there's more to the story than just more cons.

Definitely. And clinically, I didn't really see a whole lot of improvement when I just treated Americans. And I certainly. Just come back over. Well, you know, just come back.

Why would it come back? Because we didn't treat the rest of it or you would see a clinical improvement temporarily. But I didn't see that continuing to test and continuing to treat and continuing to test in like, you know, oh, making sure that they dealt with their Markon like habitations and all of that that never never made a clinical difference.

What has helped is giving biofilm busters orally giving alpha and busters intranasal and that could be ex clear. That could be something with EDTA. I mean there's a bunch of topical biofilm busters, but then the real beauty is looking at next generation sequencing too.

Identify what organisms are actually really living their. And this this is cutting edge technology. We should be doing this not just PCR, not just culture.

So explain a little bit more about what that is and how easy it is to do. Well and out of microbiologists. So it it enable. So imagine that there's a catalog of, you know, tens of thousands of organisms and what's the a test company can do as take a swab and match the organisms that are in that swab to their catalog of, you know, 70,000 organisms.

So figure out exactly what organisms are in the in the space, whatever your whatever your testing, whether that's the nose or the urine. And they can give such specificity that they are able to identify how much how many bacteria roughly is at a high count.

Is that a medium count as a low count? They're able to identify exactly which organisms, genus and species. So and some of the words are words that are very difficult to recognize.

Some of them, like when you do a urine. You'll see how many different types of strep there are. Even. You know, I've never, ever heard of some of these organisms.

And, you know, I always have to look them up because most of them you never heard of like it's not pathologic. I have no idea. So the good guys. There is an answer for sure.

Like the nobody's really studied some of these organisms to know what they do in a chronically ill patient. That. Yes, immunocompromised, right? True. True. But you'd be surprised.

The ones that I'm like, I don't know how to pronounce that. And there it is. The NIH has some study. You know, it's it's been really amazing. And so it will tell you what percentage the organism is.

Biofilms, Chronic Infections, and Next-Gen Sequencing

And and it will also tell you what the sensitivity pattern is. So what are the antibiotics? Antifungals that will kill that organism? Huge, profoundly helpful, profoundly helpful.

And then we can compound appropriate antibiotic regimens or antifungal regimens to address the specific organisms that are in somebodies nasal passages, for example, and with and shifted with.

The biofilm breaker. With the biofilm breaker, so that we can make sure we're cleaning house as much as. Possible getting in there. So when you're treating with these, how long are you usually finding that it's taking?

It's a good question. It's still clinical judgment time. Sadly, the company who was offering these tests was taking insurance. They recently stopped taking insurance, which makes that that makes that testing more expensive.

So that has changed the way that we're doing things, so that we're treating a little bit longer, trying to really hammer away at the organisms to minimize the expense for people.

So that they're not retesting more than. And, you know, that's an interesting philosophy. We're so aligned on this. I find that treating I'd rather treat longer and really get it done because it's like it's like putting a topical antibiotic on your skin.

I think so little of it's actually getting absorbed so little, it's actually going to affect the gut microbiome. So I too would prefer to treat a little longer and then and then test and see if we've got it.

Yeah, I mean, it's a balancing act, right? So if we're talking about a bladder biofilm and we're giving systemic oral antibiotics and we have the potential of really disrupting the gut microbiome because we have to treat with oral antibiotics, it's it's a fine line.

So yes, to your point, you know, I'm trying to really minimize the damage and and minimize the the cost because life is expensive enough as it is that.

Well, and the insurance thing is so interesting. I wonder I guess, you know, it's just one of those things that ends up saving the insurance companies money, like if they actually did looked at their costs for for patients getting better, it would be like, you know.

Yeah, it would be a game changer rather than having to do water and installation ads and deal with the the consequences of urinary incontinence and elderly people and recurrent urinary tract infections that cause delirium and dementia, not to mention, you know, prostate cancer, prostate hypertrophy and elderly men.

What if that's just a biofilm, bacterial biofilm problem. Right. And if we actually treated it when they were younger and we stopped having so many issues in our elderly population because we were actually treating biofilms, which are the cause of the problem.

So, you know, as people age, as people age, what happens, oh, I'm getting up on tonight to urinate. I'm getting a two and three, four times a night to urinate.

You know, we just assume that that's normal. Guess what, folks? That's not normal. That's not normal. What has happened is that we've accumulated biofilm in the bladder or the prostate or men and or both.

And the the the bladder is responding to the irritation of the organisms that are present and causing that urgency, frequency, inability to hold urine.

I had a 50 something year old fireman and he was sharing that sometimes he would have to pee in a soda can on way to, you know, a fire because he had so much urgency, he couldn't pull off and go find a restroom.

50 something. Well, it turns out he is homozygous for Pi one and he's got a biofilm and and hypercar viability. And that synergy is causing extra biofilm to be laid down and more urinary issues.

Treat the biofilm, Treat the organisms. Guess what's happening anymore. That's beautiful detective work. I mean, that's whole different quality of life.

Changed his life changed his life. Right. I'm curious what antibiotic you ended up having to use for that one. Was it a typical. You know, every time it's different.

So here's here's my analogy. I'm still working on these analogies. You're doing great. So I think it's a little bit like a lasagna. Now. I'm gluten free having made lasagna in years, but as I recall, it's you know, you put the whole layer of the pasta down and then you've got your meat sauce and your cheese and you put another layer of pasta down.

So imagine that the layer of pasta is your biofilm and then your bacteria are is the meat sauce and the cheese. And so you take a biofilm buster and you peel off the layer of the lasagna, the pasta, and then you've got your bacteria there, and then you kill those bacteria and then you peel off another layer and you've got a different layer of bacteria underneath it.

So commonly the pattern is the first layer of of biofilm. The first time we do that test will often see E.coli and requires a bacteria this very common in your gastrointestinal tract.

And it sneakily gets into the bladder. And many people, when they get urinary tract infections, they it's E coli. So here's the image for for a urinary tract infection, right.

What I'm talking about, the bacteria usually live in the biofilm and then occasionally you'll get sporadic little guys that will peel off from the biofilm and then they'll reproduce.

And if you get enough bacteria that are growing and reproducing away from the biofilm, that's when you get a urinary tract infection. And we identify that by a culture.

Culture technology is 40 years old. You can we're using 40 year old technology to identify that you have a urinary tract infection. And if there's only, you know, an and you have to have a certain criteria.

So the lab says, oh, there was over 100,000 colony forming units, so there was a lot of bacteria. Therefore you have a urinary tract infection. We're going to treat you.

But what happens if you only have, you know, 10,000 colony forming units? Well, it's not enough to qual if you for a urinary tract infection, we're not going to treat you.

But there's bacteria there. And we were taught in medical school that the bladder was sterile. Not so much. Not so much. Not so much at all. So after we see E.coli, usually the second layer is enterococcus fecal.

This, which is again another another colonizer, another organism that we typically see in the gastrointestinal

system. But sometimes I've seen lactobacillus species.

Oh, what what's the lactobacillus? Where do we usually see that? In the vagina. I've seen lactobacillus species, normal, healthy bacteria in prostates.

Mm hmm. So we're swapping bacteria back and forth with our partners all the time. Right? So it's definitely a shared microbiome. It's a shared microbiome, Right.

So we have an expectation that these bacteria are going to stay in their playing, you know, in their in their playground like vaginal bacteria, stay in the vagina, colon bacteria stay in the colon.

They don't do they move around a lot. And then, you know, we have our life experiences that make us share these things. So the layers are different and unique in everyone.

Typically with these tests, when we're testing men, we're doing semen analysis to look at what's in the prostate and urine analysis, to look at what's in the bladder.

And then with women, we're doing vaginal, vaginal swabs and the urine swabs too. And sometimes in some women, you know, they'll get normal vaginal bacteria, some lactobacillus species that are normal, that are supposed to be there.

And we don't keep looking at that because that's not necessarily adding to the problem in the bladder. And it depends on what their primary presenting issue is.

What percentage of patients are you seeing have a fungal component with the biofilm? It's not as much as you would think. It's really not as much as you would think.

I'd say it's probably more like 10 to 20%. It's it's. Not again. There's there's definitely some there, definitely some there. And, you know, as part of my mold treatment. But, you know, circling back to mold, yes I do often treat with antifungal nasal sprays, even if I don't have a test that tells me that they have fungal or yeast colonization in the nasal same. That's so fascinating.

We almost have to get on these calls to. Interview so we can share information, sharing information. A little bit more. That's great. It's really great.

And then circling back to mold, I know you have some theories on why, in addition to the hyperactive ability, the the mold patients are more susceptible to these biofilms and and in get in this predicament right.

Mold, Total Load, and Treatment Priorities

Yes I mean I think I think it goes back to total load idea on a certain level that and what I mean by that for those of you who aren't familiar with that total load idea is the body is a vessel.

I like the sink analogy myself. That's my favorite analogy because we understand it, right? So you turn on the faucet, the faucet is all of the environmental chemicals, the mold exposure that are coming in to your sink.

The size of this sink, whether it's a big deep sink or a little teeny tiny shallow sink, is determined by your predispositions and then the size of the drain.

That's also genetic, but it can be compromised by the amount of toxins that you have. So if you have a big large drain because you great got great detox, but it's all gummed up with toxins, just like a pipe can get gummed up with with stuff, you're going to have a theoretically smaller drain and you're going to have more problems with detox and getting that stuff out. So patients who have mold exposure, that's just one of the things that they often have going on.

And we've seen in many of our patients, mold patients also tend to have chronic infections or if they were tolerating their chronic infections beforehand and then they got mold exposed.

The mold suppresses the immune system and enables these organisms to flourish. And so now you've got more symptoms, more problems, more root causes that we need to address.

It before it gets to be such a vicious cycle. Right? It really and it's very complicated. I do find that we do have to address the mold at the beginning, though, I know a lot of people feel okay, Lyme is the thing or parasites are the thing.

No mold is the thing. I think that the most disruptive to the immune system, the most suppressive, the most inflammatory. And so when we're talking about hypercar viability and biofilms and, you know, mold and yeast and fungus can get into those biofilms as well.

And that's going to complicate things. The only thing that I think needs to come before mold in treatment protocol is muscle activation hypersensitivity.

So if people are exquisitely sensitive, not able to tolerate treatments, then absolutely, we have to address that first, I have to calm down that nasal activation hypersensitivity reaction first and then go after the mold in the and the mycotoxins and then worry about Lyme bartonella, parasites, virus viruses or last viruses or not.

I don't find that to be clinically relevant for the vast majority of people. And if some people come in that I have Epstein-Barr like you may, we all do.

We've all heard of Sambhar. If you're over 20 years old or older, you probably hadn't seen Barr. So maybe, maybe not clinically as relevant as you think.

And then and then probably last, I would worry about heavy metals. I really don't do Cubase and I don't do heavy metal work until people are 70% better because they're just too you're just too fragile.

You just can't. Say fine with the heavy metal. It's just by doing the things that help to detoxify the micro toxins, the metals come down to saccharine.

It's a beautiful, gentle process. But as long as they're not getting new exposures to the metals or, you know, eating too much fish, using the aluminum foil, having led cookware or those kinds of things. Right, Right.

Smoking, smoking is huge for cadmium, and cadmium is a big player. So you've really gotten to be well known for the mass. So do you want to just say a couple of minutes on that?

Because I know you have your course on Mass Island. There are going to be people interested in that. So let's just give a little preview to that. Yes, happy to do so.

I love to talk about myself. Okay. Especially because it's so important. It's so important. And for our our mold patients that become hypersensitive, it's it's dire.

You have to understand mass activation, because many practitioners out there, shoemaker practitioners out there, they don't notice their muscle. But even some of the, you know, functionally trained naturopath and and functional, they may not know how to manage hypersensitive patients.

And so you need to find a practitioner who does get MAZO So What is this mast cell thing? Mast cells are a normal part of our immune system and they are born in the bone marrow.

So they're related to our red blood cells. In our white blood cells. They then move to the periphery and they line the areas of body to protect us from the outside world.

So many of the areas that we were talking about today, that's where you will find your muscles and nasal passages and your sinuses and your mouth and your GI tract there and your lower GI there, your lower respiratory tract, too, in the lungs.

They're in the skin and they have a high affinity for the blood vessels and the nerves. So they hang out around there, too. And their job is to survey the land for foreign invaders.

And when they see a foreign invader turns out that they're filled with all these chemical messengers, they're very pretty cells. They got these little granules.

When they see a foreign invader, they dump their chemical messengers that we call mediators, which are cytokines and chemokines and histamine and inflammatory mediators to send out a signal and say, alert, alert, there is a danger here.

And so we want our mast cells to do this. This is what they're program to do. However, in some wacky people, there's some sort of genetic component to this.

The mast cells. B can can become hyper vigilant and hyper reactive, hypersensitive, however you want to phrase that.

Mast Cell Activation Syndrome Overview

And especially when patients are in a moldy environment and they're exposed to mold all the time and they're breathing it in, those mast cells go haywire and they start to misperceive foreign invaders and start to react to things that we should tolerate, like the food that we eat, our dog or cat smells, perfumes, fragrances, just about anything. Everything.

Temperature changes, vibration touch. You know, you can get that dirt chromatograph. Yeah, right. So if you can write your name on your arm or your leg, you probably have nasal, or at least you have one symptom.

But because these mast cells are a so many different places in the body and the genetics is more somatic than germline, so, so the genes are still being worked out, but on average, muscle activation is thought to be in about a 17% of the population, 18% of the population.

That's a huge number. That's one in five. So if you have a family of five living in a moldy house, at least one of them will probably have muscle activation symptoms.

And so what happens is that they become really sensitive to everything. And you have symptoms in multiple systems in the body so people can have fatigue and muscle aches and joint pains and brain fog and neurological symptoms, headaches, migraines, tingling, numbness, psychological anxiety, depression, mood disorders, and then every system in the body can be impacted.

Some people have a lot of allergy symptoms. Some people don't have any allergy symptoms. And so it looks like so many different diseases and it looks different in every person, but it's very difficult to identify.

So it's really a clinical diagnosis. And what I'm looking for or what my colleagues are looking for in making that diagnosis is multiple inflammatory allergic or growth symptoms in multiple systems in the body.

And there are two broad groups we call them consensus one and consensus two, who who test and treat for muscle activation. Consensus one is mostly your conventional allergist and immunologists.

They have defined criteria which is very strict and looking at usually just triptans and days is a marker they look at in blood and many, many people who clinically have muscle activation would not meet their criteria.

So if you don't meet their criteria of elevated triptans levels, blah, blah, blah, go find another practitioner. So

consensus two is Dr. Laurence Afrin, myself and many of my other colleagues who wrote an article on the diagnosis diagnostic criteria of nasal activation.

And in that we list out the clinical criteria for the definition of muscle activation, as well as some of the laboratory values that you can look at. People can also have biopsies of tissue such as bladder or colon, esophagus, duodenum, you know, so anyone over age 50 who's had a colonoscopy you can ask for a special stain to be done in the biopsies that were taken.

The special stain is called CD117 If there are greater than 20 muscles per high power field, particularly in the duodenum, but also anywhere in the colon, that also meets the criteria and supports the diagnostic definition of mast cell activation.

And then the treatment is very complicated, but the good news is there's lots of options. So antihistamines, there's pharmaceuticals, there are some pharmaceutical mast cell stabilizers, not too many, but a number of them.

And then there's a ton of supplements, things like Quercetin, luteolin, Pycnogenol, resveratrol, Turmeric Paramin there are certain probiotics that can be helpful.

There's a low histamine diet that can be helpful. So there's a ton of things to do for mast cell activation syndrome. That was great. But you know, nice little though, very quickly.

And give a really awesome overview. And I think it really is it is the first thing to really look at. And it's so, so clear with the clinical diagnosis and and then starting to treat you start to see improvement.

So you know, you're on the right path. Exactly. And that's one of the other, you know, kind of diagnostic criteria is if we give you something that treats mast cell activation and you get better, great. We're on.

We don't need the biopsy. Exactly. Yeah. I don't send anyone for colonoscopy unless they need it for another reason. And some people do really want they want that diagnosis down to that degree because, you know, that's just it makes them feel better about having a clear diagnostic diagnostic.

So it's fine for the people that want it, but for the people that just want to start feeling better, we can get going. Absolutely. Yeah. I mean, I do believe that diagnosis is important, particularly if you have a number of conditions and you have a number of practitioners on your team, right?

So if you have cancer and you have a cardiologist, you've got an oncologist and a cardiologist and a nephrologist and you're, you know, functional doctor who's working with you and your other doctors are nice, but they don't necessarily believe in this whole functional thing.

A diagnosis is really important. Is important. Yeah. Testing is tricky, though. Blood and urine, they have to be handled properly, so the practitioner has to be on board with finding a lab that's going to handle things properly.

The blood has to be chilled, spun in a refrigerated centrifuge, you know, and kept cold from drawer into a chilled tube all the way to the lab. And it's really difficult to have that chain of command be all on board and in.

So it's a lot of legwork on the part of the practitioner to make sure that they have a lab that works well. And unfortunately, you know, conventional labs like LabCorp and Quest only have a couple of markers that they can do.

But the couple of markers that they do do if, say, for example, you have a histamine drawn at Quest or LabCorp and you it's high and it's high 2 times.

Guess what? You've met the criteria because we want to have at least we want to have two labs that are positive and they can be positive at the same time.

So histamine and a elevated Tryptase or Chromogranin A And you can have those drawn at LabCorp or Quest and

if they're elevated at a second time. So you've met your criteria pretty cool you one.

Well this has been just so fun to get to and have you share your brilliance and your heart and your care and the you you are definitely making a huge impact in this world of a chronic illness and piling new territory and putting new puzzle pieces together.

And I'm so grateful for you. Thanks. And I'm so grateful for you and for all the work that you're doing in helping people get more information so that they can get well.

Thank you. Let's share how to find you. Yes, I am. I have a brick and mortar practice in Southern California. It's called theSpringCenter.com You have to put the in there theSpringCenter.com And I also have my personal website, drkellymccann.com, and I have that.

I have things that you can check out there too. Thank you so much. And I look forward to the next time we get to talk. Me to me to take care. Thank you.