Unlock Lyme Disease Detection With T-Cell Testing

Myriah Hinchey, ND, FMAPS
with Felix Scholz, PhD

Myriah Hinchey, ND, FMAPS
Welcome to another episode of the Healing Lyme Summit. I'm your host, Dr. Myriah Hinchey. Today we're going to be talking about testing T-cell activity to detect whether we have an active Lyme infection or not. Here with us is Felix Scholz, PhD. Dr. Scholz is the CEO of Infectolab Americas. Dr. Scholz, please tell our listeners a little bit more about yourself and how and why you got into the field of laboratory testing.

Felix Scholz, PhD
Hi. Thank you for having me here. It's an honor. I started as a basic scientist. I studied biology and then did a PhD. The thesis was about skin immunology, and I did a few years of post-doc research in the same field at the Mayo Clinic and at the Center for Immunology here at the University of Minnesota. Then I switched to the lab world by working for a Biotech company, where I developed lab tests from scratch and different types of lab tests. Then I met Dr. Charles Nicholas, the founder of Sector Labs, at a scientific meeting, and we started collaborating. Then I got the opportunity to build, in fact, a lab for him. Then here we are.

Myriah Hinchey, ND, FMAPS
Let's start by talking about the very basics. Tell our listeners the difference between an indirect and a direct laboratory test for Lyme disease.

Felix Scholz, PhD
There are different ways we can approach checking for infectious diseases. There are very common platforms that are used by a lot of lab tests and by different lab companies and different labs. The most common one for infectious diseases is the so-called laser. It is a serology test where you need a blood draw, and in the serum, the lab looks for specific antibodies found in a patient that bind to a plate. Then you can quantify how many antibodies are per microliter of blood found. That is the very standard method and is employed virtually. Most labs do that. It's

also an indirect test because the definition of indirect is that you check for an immune response against the disease. Here, you check for the PCR response with an antibody, and then you detect antigens. If they pull out the antibodies, and if you find the antibodies, the deduction is highly likely. The patient had exposure, both exposure against that antigen and the proper immune response, and has found and built IgG. A direct method would be like a PCR, where you check in a tissue or blood, depending on what pathogen you look for, and checked with a PCR where specific primers are called the start ends for a biochemical reaction that amplifies the genomic material of the pathogen. Because once you see that product in a PCR, the pathogen has been there; it's direct.

Myriah Hinchey, ND, FMAPS
Direct is looking for fragments of genetic material in the organism, whereas indirect is looking at the immune system's response to the presence of the organism.

Felix Scholz, PhD
That's correct. Another direct method would be histology. Let's say you have a skin infection. You take the punch biopsy embedded, cut it, make thin slices, and then stain directly for the pathogen. That would be all direct, and another indirect way is T-cell responses. T-cells are like B-cells, which are part of the adaptive immune system. Ideally, in any infection, you get responses from these branches of adaptive immunity, regardless of the disease. T-cells are also required for a proper B-cell response. You can check for T-cells that are called Elispot. That is when you draw the blood from the patient. In the laboratory, they isolate the white blood cells, the so-called PBMCs, and they put them on a little plate, like a similar analyzer plate. But in this case, there's a membrane, and then the T-cells get plated on there, and then they get stimulated with specific antigens, similar to an analyzer where the antigens are plated on a plate. Here, the T-cells get stimulated by it, and they're still living T-cells. Once they recognize the antigen, they produce cytokines as they affect the function, and they leave the cytokines like little spots on the membrane, and then that can be stained and quantified. In the T-cell test, like an ELISPOT, you can quantify how many antigen-specific T-cells are present against that antigen that you tested for. That's called the T-cell frequency. The advantage of this type of testing is that you can deduce a little bit how bad the disease is. One parameter that drives T-cell frequency is the antigenic burden, or how much pathogen is bearing the patient right now. If there's more pathogen in a patient with that antigen, you'll find more antigen-specific T-cells. This also lends itself to a monitoring test, so you can check in during treatment and see if the T-cell frequency or the amount of antigen-specific T-cells found goes down over time. Hopefully, at the end of the treatment, they're gone because that means there's no antigen in the patient left, hence there's no infectious agent, bacterium virus, or whatever disease was left in the patient.

Myriah Hinchey, ND, FMAPS
Like with an IgG response, even after the infection has resolved, you could still have a low IgG response to the various proteins and antigens, which could just be memory cells and immune

surveillance, or it could still be an active infection. With the T-cell test, you're saying that if the antigen is gone out of the body, there shouldn't be any T-cell response to that specific antigen. Am I understanding that correctly?

Felix Scholz, PhD
Yes, that's correct. The reason is that the way at least we hear it, labs run the T-cell tests, and we see only TH1 cells. We do not see the memory T-cells. That's why we cannot look into the past and T-cell lives. The estimates are rough because most of these studies come from mice, so they're not necessarily extrapolated. But what we see, depending on the disease and the antigen, is that the lifespan of these T-cells is six to eight weeks. That means if you check somebody positive and then treat them successfully three months later, you shouldn't see any T-cells anymore. with the antibody titers, which is a little bit tricky. That also depends on the antigen and the disease, so forth for B-cells and antibody titers. The important biochemical reaction you saw was doing a presentation to the B-cell in the lymph node. That is when the B-cells make the first scrambled antibody and check in with a so-called T follicular helper cell. That's a T-cell that has the antigen, and that can measure how good the affinity is of the antibody to the antigen and give it back to the B7 and the B7 by rearranging the genomics for the antibody again and doing multiple rounds. That way, you get a higher antibody, meaning an antibody that binds better, and hopefully, at the end of the day, is the neutralizing antibody against that antigen. This interaction is crucial to form at the end of the day, A B-cell becomes a plasma B-cell and plasma B-cells are the manufacturers of antibodies that measure the blood. Now some certain diseases or antigens favor that response, and certain diseases and antigens slow that response down. Those diseases, like if you have some inefficient affinity maturation, what that process is called, you can have some of those diseases, a fairly quick drop in antibody titer once the disease is gone, and others, you may have high antibody titers for months or years to come. What happens with certain diseases where we have good plasma cells does say people have years of protection from the IgG and then other diseases advantages fairly quickly, like in COVID, for example, COVID is supported by disease. You get mostly T-cells of the TH1 phenotype. The TFH is neglected. Hence, we see that in people who have experienced COVID, depending on their age, the antibody titers drop fairly quickly, but the reason is because of the physiology of the disease. You have certain diseases where you can do that and employ antibody titers for checking for successful treatment, and other diseases are pointless because the antibody titers stay high. That's the advantage of T-cells because, regardless of disease, if you measure TH1 T-cells after the bug is gone, the T-cells will be gone within a couple of months.

Myriah Hinchey, ND, FMAPS
When you talk about it being specific to the antigen, how do you not know that maybe you got some other infection or that the T-cells are responding to something else in the body as time goes on?

Felix Scholz, PhD
In antigen-specific T-cells, what people use is very specific antigens. Most of the time, it is a peptide. Antigens are proteins or chunks of protein, like digested proteins. An antigen is in the length of 10 to 12 amino acids long, and it must meet two criteria: One, it needs to sit in MHC class-2, that is the presenter molecule, and the other one, it needs to have a fitting T-cell receptor, is the receiving end on the T-cell. Now if this requires 10 to 12 amino acids. It makes it specific on the T-cell that there's a certain amount of wiggle room in, in the T-cell, what it recognizes of this sort because you can envision it's very short. It's like 10 to 12 amino acids. An average protein has 800 amino acids. You find multiple of these antigens and some amino acids. But if, from a perspective, you see, and that is often done in validation, you see that the antigens hit a high population because not everybody has the same T-cell except for the same little MSC class-2. That's different from person A to person B. The idea is that antigen hits 100% of infected people, and you need to check that at a lab. You need to see if your antigen is that efficient or not. That is not very simple. That's often something that takes time. That's why, often, good lab tests take time. It's a new disease that pops up, and everybody thinks that it's a threat. But if you look specifically at laser tests or T-cell tests, even if it takes time until you have a good product on the market, it's like if somebody gets within half a year with a T-cell test out, that's not a good sign. It takes time like this, for example, to work on the common antigen for a couple of years now. Because it's a rare disease, it just takes time because you need, you don't want, one to hit a large population of the infected, but through your two sports negatives, and second, it shouldn't be cross-reactive. With subspecies, that's tricky sometimes because they're related diseases that share related antigens; you cannot distinguish them, but in certain cases, that doesn't matter because the treatment is the same. But if treatment is not the same, it matters if you have an overlap between closely related diseases, but that is also hard to figure out, like for a lab. There's no guarantee. What we do is run our antigens on non-negative people to check that we do not have that issue, because if you have a cross-reactive disease and these people do not have this disease, but you have a cross-reactive antigen, you will find a certain percentage of these people positive. That's one way we try to ensure that we don't have that issue because, theoretically, it's possible.

Myriah Hinchey, ND, FMAPS
But so for this information to be clinically applicable, like could you just do this test in the end when you suspect that your patient's Lyme is now in remission, or is this something where you need a baseline or you need to see what that initial response was or like you needed to see their T-cell activity when they were symptomatic and had a large infection? You're comparing that to the result. Or you could just do this test once in the end to confirm, as best you can with any test, that the infection is in remission or gone.

Felix Scholz, PhD
We see that the doctors employ it either way, like from one of our customers, for example, who runs with Crest to do antibodies, and then during treatment, they check in with the T-cells. They

employ it like that. It's always nice to have a positive to begin with because that's where you come from, specifically with Lyme, where we see low T-cell frequencies. That means, for example, let's take Epstein-Barr. With the tissue test, no question was asked because of Epstein-Barr, if somebody has a reactivation of so many viral particles you find through the roof of antigen-specific T-cells, no question is asked. but now you have more tricky diseases like Lyme, where the bug is not necessarily always seen by the immune system. Then you can run into issues with the indirect detection method because if the immune system doesn't see it. That's additional access you get. For example, if you are very good at diagnosing Lyme based on symptoms and you run antibodies and T-cells that can be additional helpful information because now if for sure this patient has Lyme, but the T-cells are negative, then the T-cells are fighting right then, I need to help them to to to get fighting. I think it's always good to have a starting point to compare it to after treatment because then you also know where you come from. A different person has ten antigen-specific T-cells per percent or five, and do you need to be more careful? If you have a slow, low T-cell frequency to begin with. Why do you need to worry if it's negative for others? it's always good to have that data. But also, people should keep in mind what they spend on lab tests with their patients because it's depression. It's like you can spend so many things under the sun or on lab tests. I always appreciate it if the doctor thinks about what is useful in this situation. It doesn't make sense.

Myriah Hinchey, ND, FMAPS
That's what I was thinking when I was asking that question because so many of my patients have already come in, and they've spent thousands of dollars on testing. We already know what they have. it's like, do you think it's important to get a reading on the T-cells at that point, or is it okay to use this almost as a check to make sure that it's cleared? But you just answered that.

Felix Scholz, PhD
The check-in that it's cleared is always a good point. Because then, if the disease is what we talked about earlier, there is still some antigen left, you will find the T-cells. If you don't find it in ELISPOT T-cells, then you can be fairly certain it's cleared. It's not 100% either. But at least it looks good.

Myriah Hinchey, ND, FMAPS
I was, and that was my next question. Like, what is the false negative rate? Would this type of testing?

Felix Scholz, PhD
That is a very tricky question because, if you look at the sensitivity, and specificity that the labs always tell about line testing, that is not a real word measure because, often specifically for serology, there are cohorts you get for chronic and acute Lyme from the CDC or age or from some Lyme nonprofits, and you can get access to those, and they're very helpful and they record these cohorts. But the issue is that they're designed for lab test development. As I said earlier, it's

hard to find good antigens. Now if you get access to, let's say, 100 samples from the CDC that are known Lyme patients, known positives, That's a super good start because then you can check out your antigen, and if you perform worse, then you don't care. That antigen is not a good choice. it's helpful. But so now what can people do because it's hard to get good cohorts as a lab? They utilize these to calculate their specificity and sensitivity, which is then pointless because that's not a real road measure. But clearly, not 100% of Lyme patients have IgG; you can slot it. The real-world immune response to Lyme is way worse, so you'll probably find around 40%–45%. and that is more realistic and similar to what is true for T-cells. The thing is, the immune system doesn't see the bug. Any indirect test will have false negatives in life that, in my opinion, will also never go away. But because it's the bug somewhere in the tissue, like directly, it's pointless because you don't find the DNA in the blood indirectly. If they just sit quietly in the tissue, indirect tests would be negative. That's what we see. But like on both ends, we have some customers that work around with that, and they do when they suspect Lyme and don't see any positives and they start treating, and then they check in with T-cells, and two weeks later, if it truly was Lyme, they see that he said two weeks later because what it does is what the immune system needs. It's like jumpstarting the immune system. You start killing the bug; the dead material ends up in the lymph node; there's the protein; the T-cells can be primed; they can divide and go fight. so contact with blood where you catch them in the blood. That is the issue there. yet that is also a false positive rate. Sorry, excuse me, false negative rate. It's just hard to do proper cohorts on that because you would need to study where people know 100% this hundred people have lied right now and that type of quote is hard to get because people are not sure if somebody is lying or not.

Myriah Hinchey, ND, FMAPS
Why don't you tell our listeners how they can find more information about T-cell testing?

Felix Scholz, PhD
We have on a webpage, infectolab-americas.com, some useful information where we go a little bit into the science of T-cells and T-cell detection. People can go to that web page and read about it. If a doctor is curious about this, they can also reach out to us, and we can answer questions. If there are any questions,

Myriah Hinchey, ND, FMAPS
If you're a patient listening who's interested too, you can talk to your doctor about learning more about, Infectolab and T cell activity testing. For those of you who are subscribers to the summit, thank you. Stay with us. We're going to dive deeper into this topic. For those of you who haven't clicked that button below, you can come with us and listen to the rest of this wonderful interview.

I want to ask, regarding all of the different species of Borrelia that cause Lyme disease and tick-borne relapsing fever, how does a T-cell activity test account for all of those? I'm way more

familiar with doing immunoblots and things like that where you're looking at these specific antigen protein chains and the antibody response, and they're looking at all of these various species when it comes to looking at T-cell activity on what species of Borrelia are covered.

Felix Scholz, PhD
That goes back to the beginning. That is the tricky part of antigenic design. Like what is a good antigen, T-cells have another roadblock that you do not have in serology. in western blot immunoblot and Eliza is that we cannot use so-called recombinant antigens. That is a technical term. If you are curious about a protein or you think it's an antigen, you can manufacture that in a bacterium, in large quantity, and then you can utilize it and put it on plates or a G11 and then transfer it to the blot and then go from there. That's straightforward. The issue we have is that for T-cells, these recombinant proteins, for whatever reason, cause false positivity. They will come back to the cross-reactivity of T-cells. that's why we cannot use them. That is another slowdown, because we need to know right now, out of these 800 amino acids, which ones are the ones for T-cells that we can utilize, and then we cannot make them recombinant. We need to manufacture them differently, or we need to purify them out of the pathogen itself. It's a little bit more tricky. That's why we don't have that arsenal of all antigens available for T-cell testing. Currently, we have two different sets of antigens for Lyme. The reason for that is that Lyme, as I mentioned earlier, is tricky for the immune system to see. If you broaden the how do you say the wet chip, we have two for those, and they detect in their specific antigens for different subspecies in there. It's not only the B-31 mix. We have also had a PMX, where we can detect different European species that start to be seen. small pockets in the US as well. You don't have to worry about that. For tick-borne relapsing fever, we have only miyamotoi as an antigen, so not for the other relapsing fever species. We do not have antigens there yet.

Myriah Hinchey, ND, FMAPS
If somebody had one of those other species, this test would not be accurate for them because it's not looking at the specific antigen that they're infected with.

Felix Scholz, PhD
That's correct. Unless there's some cross-reactivity because of the relationship, But also, that is pure speculation because, again, it's not like one of the antigens of Borrelia species. There's not been a lot of research done. There's also Hartz two to get good information. What is the cross-reactivity of or of a Lyme-causing Borrelia species where there's a relapsing fever? They related, but that's like nobody knows what the related antigens or she had. so we cannot know that part.

Myriah Hinchey, ND, FMAPS
It sounds like it all boils down to the immune system. Most of these tests that are being utilized, whether they are IgG, IgM, or T-cell activity, are all reliant on a robust response or at least an

appropriate response from the immune system. What other tests can we do to evaluate a patient's immune system? We have an idea if it's responding correctly.

Felix Scholz, PhD
There's a test we just developed a couple of months ago or last year but published a couple of months ago, where we assess the functionality of the immune system in a patient right now, like at the time of the blood draw. What we do there is check in on CD8 and CD4 T-cells that have seen their antigen. meaning we check for all of them, and we can quantify how many of these antigens experience T-cells, regardless of the specificity, important distinguishing. You are currently in fighting mode. We quantified that in two ways. One way is the percentage of responders, like when we stimulate the cells, give them a few hours to do their thing, and then quantify how many have responded to the stimuli. We see people who do not have an infection currently respond with 5% activity, or 5% of T-cells C positive, and around 10% to 13% of the active in these people. then we also quantify the number of sector cytokines made per set on average, and that is a very unique measure because that gives you some insight into how effective the T-cell is right now. Like, is it on the low end of the effect or function on the high end, or exactly where you want it to be? There are differences between infected and non-infected as we often see in the city force and the CDC. We have an arbitrary unit for this. We call it the immune activity index. We see five K units in healthy people. But if they're fighting an infection that jumps up to ten K units or 15 K and viral infection, we even seen something close to 20 K, there's a significant more amount of effect of cytokines seen in T-cells that are coming from a patient who is currently fighting infection. That is a nice addition, to see if you test the patient negative for everything. You look at the immune system then you see if the immune system needs to fight or if I'm good. If you see it's upregulated in fighting, then you need to search further on what's going on with the patient.

Myriah Hinchey, ND, FMAPS
To be clear, this is a general assessment of whether the patient is fighting an infection, not specifically a Lyme or Borrelia infection.

Felix Scholz, PhD
It's a test that you can use to quantify the immune response. You would also see if somebody is immune-impaired, like, for example, a similar test was used to assess how, for example, prednisone works. If you look at those old publications you can tell that people on prednisone, for example, produce way less cytokine amount per T-cell. You would also see that, in a scenario like that, you would expect that if somebody has an infection but the immune system is not up for it, then you would expect the percentage of active T-cells to be higher than in a non-infected person, but you would expect the effector cytokine unit to be lower than in other infected people. Because then that would be the definition of immune impaired. Somebody who had an immune response was not mounted appropriately. That's what this test is designed to show.

Myriah Hinchey, ND, FMAPS
What other things can you look at? What other infections are viral, bacterial, or parasitic? What else is a lab test for using this T-cell analysis?

Felix Scholz, PhD
For the antigen-specific stuff we have, I always don't know 100% of the 17 antigen mixes that have been validated. That is from the tick-borne complex we have selected for Borrelia Burgdorferi, Afzelii, and Garinii. The mixes that we have are the Bartonella, Babesia, and Rickettsia.

Then we have some superinfections like chlamydia, pneumonia, and mycoplasma pneumonia that people tend to pick up with chronic disease. Then we have a whole slew of viruses like COVID, which is interesting for long-haul COVID because that's the only test where you can check if there's still a viral reservoir in a patient because there's like a third of long-haul patients that may still have some COVID viruses are the last of two viruses left, and it's been published that they are highly likely in the lungs and inappropriate in the gut. But as a doctor, you don't have the means to test. But the T-cells are there. If you have a viral reservoir, you'll find the T-cells. That's one of the only tests you can employ to check if a long-haul patient has a virus to present, yes or no. Then we have the Epstein-Barr virus, so we can check for lytic latent. We can distinguish the two scenarios because of the specific antigens we have for each of those scenarios. HSV1, HSV2 Human Herpesvirus 6: What's Important and Long COVID-19 and Varicella-Zoster. We have quite a few in the viral compartment. They're all good immune inducers. Like the question will be at earlier with Lyme of the false negatives, it's virtually not there like because if somebody has these viruses, there's enough antigen around and the t-cell numbers that we find are fairly high.

Myriah Hinchey, ND, FMAPS
That's very helpful. I see a lot of these viruses, at least from an antibody perspective. We see a lot of high antibodies to all of these viruses that you're talking about when we look for them. I'm explaining it to patients, as these are all like straws on the camel's back of the immune system. The more infections you have, the more synergistically they're going to work together to cause immune dysregulation and inflammation, and then the easier time all of these infections have living inside of us and breaking down our bodies and feeding off of us. Fascinating.

Felix Scholz, PhD
The beauty of the T-cells in this context is that you know which of these are issues currently. Again, we look for antigen-specific T-cells in that context, and we can tell if that is the current issue or not because these viruses are good immune inducers. That means you will find long-lost plasma cells, and hence IgG, for a time to come. That means if you find the early antigens for EBV skyrocketing high, they can come from a reactivation right now or from one that was six months ago, and the T-cells would fill in the gap and tell you if it is right now or not.

Myriah Hinchey, ND, FMAPS
I imagine that you have full panels that one can use to evaluate all of those at once.

Felix Scholz, PhD
Yes. We have a full panel for that, a lot of our customers are ordering those specifically, like some cancer clinics because some of these are viruses, and most of them that you check for belong to the herpes complex. As we've seen, some of them are associated, and more literature has come out on the choice, but the other ones, like EBV, were not suspected to be associated with cancer, but now new research shows that they are, so it's a good test also for people that deal with cancer clinics to check on those on what's the status of these viruses there. That's what we do.

Myriah Hinchey, ND, FMAPS
Is there anything else that you would like to share with our listeners?

Felix Scholz, PhD
I don't know. I'm good.

Myriah Hinchey, ND, FMAPS
I mean pieces of advice when it comes to testing for any of these complex chronic illnesses.

Felix Scholz, PhD
Yes, ask your doctor questions and why they want to order certain things because I believe that there are a lot of lab tests out there and not everything is the same. It depends on what a doctor wants to see. But it also needs to be that a doctor needs to be able to explain and justify what lab tests are ordered because it can be anything under the sun, and I'm a big fan of ordering to cater to a specific situation, like direct or indirect, like a lot of PCR tests are not that expensive, but it doesn't make sense in all scenarios. If the bug is not in the blood, you can skip that. You don't need to do it. If it's justified symptoms, what could be possibly going on, and then picking specific tests that have the highest chance of giving good answers, like a good utilization of how to approach it. That's what I try to tell people: think a minute about what needs to be solved right now with the lab tests. Also, as a doctor, the more specific you ask the question, the more specific your answer will be. There's lab testing wherever you can, I don't know, the whole genome of pathogens. But does it answer the question?

Myriah Hinchey, ND, FMAPS
You can only test or get answers to what you're ordering. A lot of people don't realize that I have so many patients coming to me. They're like, I saw my doctor. I'm negative about everything. I'm like, everything. What's everything? 5000 different tests could have been ordered on you. It's like, they had like a CBC and a CMP, and they think they're good to go, and they have a clean bill of health. It's just crazy that a lot of people are walking around thinking and doing when it's like you can only get answers to what you asked for.

Felix Scholz, PhD
It's like the approach. Something is wrong with this laundry list of lab tests. I just don't know; it's like blindfolding and throwing the dart. That's pointless if you aim for one specific test; you may have a better answer then, because if you then run some certain tests that are also uncertain, but then you suddenly get distracted and go down a rabbit hole to something completely irrelevant, like certain lab tests give you suddenly answers that you didn't look for. But they may not be relevant to getting the patient better. I always like thinking about what's necessary and relevant and what my question is, and the clearer the question is asked, the clearer the answer will be.

Myriah Hinchey, ND, FMAPS
It comes back to just the basics of learning how to be a clinician. We make a differential diagnosis for a reason. Then we have to go through and rule those certain things out or rule certain things in. When it comes to Lyme and tick-borne diseases, it is a clinical diagnosis. It's like we've already come to the clinical conclusion that this is what is going on. Then we're trying to use laboratory findings to confirm that. To help support that diagnosis. It's funny because the CDC, on its website, when it talks about Lyme disease, even talks about Lyme being diagnosed based on three things, like, number one, clinical findings and symptoms. Number two, could you benefit from being exposed to this infection? Then, number three, says that laboratory results help confirm the presence if you're using validated methods. Not that I put all of my faith in the CDC, but it's like, they're even saying this. A lot of clinicians are just looking to, even through a conventional lab, which is just dismal, the word positive or negative. That's where they're going. It is like maddening to me because I have so many patients that come in and they're like, I've been tested for Lyme like 20 times. I don't have it. I've been to 16 other doctors. No one can figure out what's wrong with me, but every single one of them has told me it is not Lyme disease.

Felix Scholz, PhD
It's complicated. Specifically in Lyme. I guess that's also where the CDC probably mentioned that. If you only find antibodies in around 42 to 50% of people, then it has to be a clinical diagnosis. But then flipping a coin has a higher chance of 50/50. That's why it makes complete sense to them to get good training on what symptoms match the picture of Lyme, and so what some people do is what I mentioned earlier pretreat and then two weeks later check because then you can use at least as somewhat confirmatory because if you start killing the bug and was there, you see an immune response one way or the other. That is also a choice.

Myriah Hinchey, ND, FMAPS
You see stronger T-cell activity when you've pretreated for two weeks as opposed to just doing the test right off the bat.

Felix Scholz, PhD
Yes.

Myriah Hinchey, ND, FMAPS
But is it like shrinking the infectious load takes a little bit of stress off the immune system, and it's not able to respond?

Felix Scholz, PhD
No, you're killing the bug. and so it's now Lyme sits, let's say, somewhere in interstitial tissue. There's no immune system.

Myriah Hinchey, ND, FMAPS
It's like it's the reaction to the LPS.

Felix Scholz, PhD
Suddenly, you make antigen by killing the antigen available in the lymph node, and then, as an outcome, it's on T-cells and the antibodies, and we see that that pre-treatment method is fairly successful like we have here in our neighborhood. The doctor has been using us since day one, and when I looked through the data one day, I realized that he, from one month to the next, had a higher positivity rate. I was like, so I gave him a call and said, What happened? It said he switched to a trigger treatment, like jumpstarting the immune system. So that seems to help get more detection and makes sense.

Myriah Hinchey, ND, FMAPS
In a way, it's like using a provoking agent if you're going to do something like a heavy metal test, or like using glutathione for several days leading up to doing something like a micro toxin.

Felix Scholz, PhD
It's the same thing, but it's just a different method because it's roughly like you need to help the immune system see the antigen. That's all you need to do. We also see that in people who test positive in the first round and if their doctor rechecks within a few weeks, the T-cell numbers go up because, for the same reason, you kill the bug with your treatment, and then the immune system sees more antigen, makes more T-cells, and then the T7 will go up. It makes sense. then, after successful treatment, it goes slowly down again. until if it's cleared, it's cleared, and then the T-cells are gone. But you see that too. Sometimes, when people start treating, they call and say, Hey, I was just confused. It looks like the disease got worse, but the patient feels better. But then you say, No, you just killed a lot of bugs, and that made the system see more antigens. because more antigens, more T-cells; that's why temporarily the T-cells go up. That means it is exactly happening. Whatever you intend to do, you're killing the bug. That's another place you can use T-cells to see if the treatment works. But it's not a requirement because it's also good to see that after clearance is just gone, you don't need to check every two weeks. It's a waste of time.

Myriah Hinchey, ND, FMAPS
I wish these tests were covered because you would get so much more peace of mind from patients. Luckily, seeing the severity, the frequency, and the sheer number of symptoms coming over time shows that what you're doing is working. But with herx in the mix, this also takes time because, in my opinion, it's not just about killing the infection it's about healing the body and fixing the immune dysregulation caused by the infection. It just takes a long time. It would be great to be able to do a test like this every few months and to have that feedback.

Felix Scholz, PhD
What we would do yearly is have a form to fill for diagnostic codes so the doctors can put appropriate codes there, and then itemize the bill so each antigen gets its line with their CPT code and the CPT code for our way of testing is $135. There's a chance of patients getting money back if they sell themselves to insurance. I'm not an insurance person, so I don't know how good the chances are, but people can try.

Myriah Hinchey, ND, FMAPS
They can try, and they can also call ahead and see if it'll be covered if they have the appropriate ICD-10 codes and the CPT codes.

Felix Scholz, PhD
They can check in with insurance before. That's also good.

Myriah Hinchey, ND, FMAPS
That's a great point. I want to thank you so much. Go ahead.

Felix Scholz, PhD
Some do get reimbursements. That's between them and the insurance. But I had a call last year from a patient who just wanted to get our bill resubmitted to her. She said it was the year before she got the money back from her insurance. However, she also disclosed that she has experience with insurance billing. I don't know if there's a trick.

Myriah Hinchey, ND, FMAPS
There's always a trick when it comes to insurance companies.

Felix Scholz, PhD
That's not my forte.

Myriah Hinchey, ND, FMAPS
Well, thank you, Dr. Scholz, for joining me. Thank you to all of you at home for joining us and listening. I hope that you found this information helpful today and that you learned something. I

certainly do every time I talk to this man, I hope that what we have shared with you today helps you on your journey to healing Lyme. Take care. We'll see you next time.

Felix Scholz, PhD
Thank you very much for having me.