Introduction and speaker background

Hello, everyone. My name is Dr. Richard Horowitz, and I'm the co-host of the DrTalks Healing from Lyme Summit. And it's my great pleasure today to introduce to you Dr. Bruce Patterson.

And today, the topic of our talk will be differentiating treating long Covid versus chronic Lyme disease. There's a lot of overlaps between these two diseases.

And Bruce is one of the experts in the field. So, I'll just give you a quick update on Bruce's bio. And then, Bruce, you can take it a little bit from there as far as how you got into this.

But, Bruce Patterson, M. D., received his undergraduate training in molecular biology from the University of Michigan in Ann Arbor and then his M. D. at Northwestern University Feinberg School of Medicine, followed by residency in pathology.

And in the early stages of the Aids epidemic, Dr. Patterson began investigating cellular reservoirs of HIV. and in the last couple of years, you have really turned out to be one of the lead researchers now and long Covid.

You publish some great papers on the topic. So why don't you tell everyone just a little bit about yourself and kind of how you got into Long-Covid, considering you're one of the few people who actually took a major interest in this from the beginning.

I appreciate that, Richard. It's nice to be here with you. so a lot of it stemmed from the work we did in HIV. And when I first started in HIV, they didn't know where the virus was.

It was is still a big question. You know, is Aids actually caused by HIV because they couldn't put two and two together? and that's what led to the search for, you know, reservoirs of, of, of HIV and of course, using some of the technology, developed at northwestern, we actually found it.

And so, you know, when, when, when Covid came along, it was about my fourth pandemic. and, you know, I was actually in Shanghai and January 6th, 2020, and I was about to go to Wuhan to visit, another laboratory.

And my I got this message the night before that, my trip was canceled. So. And somebody the next day at this, cancer company showed me one of the first reports in Chinese, some of the data from from these, patients with this kind of new, virus that nobody had labeled yet.

And it what struck me was, how, engaged the immune, the innate immune system was and how macrophage driven the immune response was. And that's really started at all, in terms of trying to come up with, strategic ways to treat acute Covid and, and, of course, served us well as we are differentiating long Covid from acute Covid.

Right. So, and I know you and I have talked about this with chronic Lyme disease, but one of the reasons we're talking here today is the symptoms overlap quite a bit.

And so do the pathology. So can you talk a little bit about some of the underlying mechanisms now that you've kind of discovered with long Covid. And interestingly enough, when I keep looking at the literature, I keep seeing more and more of these overlaps between the two diseases.

So can you talk a little bit about that? because it's such an interesting topic. and of course, long Covid is all over the media right now. However, you know, Lyme, what struck me was when we first modeled, long Covid in terms of using two separate algorithms, the severity index and a long hauler index.

there was a very distinct, immune signature, in long Covid. And then when we did what's called a confusion matrix where we looked at MECFS, chronic Lyme, some of the other, chronic inflammatory conditions, what struck me was there was overlap, for sure, but there was also very distinct, areas that we could exploit from a diagnostic sense.

So in other words, we wanted to use the similarities in terms of treatment and can give into that, in a moment, because there is some commonality that we found, in the underlying pathology, other two conditions.

But there were some differences that initially we wanted to exploit, in terms of distinguishing between long Covid and, chronic Lyme and MECFS from a diagnostic standpoint.

And that's actually the topic of our latest paper that's out in preprint.

How long COVID research began

And, is being, peer review it as we speak. Right. So, you know, when, when you and I had talked at IHS a couple of years ago, you were mentioning at that point about CCL five rantes that chemokines and the fact that we were seeing some of the inflammatory cytokines we see with Lyme.

you explain a little bit about that cytokine panel, chemokines, like how they vary with what you're seeing in Long-Covid versus Lyme versus CFS, fibro like, how are these varying a bit.

Right. And since we we last discussed we you know, we're up to I think close to 50,000 registered patients of all of these chronic inflammatory conditions.

So we have more data to fall back on. But what was emerging was this pattern, the first thing that tipped us off was, a cytokine called interleukin eight, which we didn't commonly see elevated, in long Covid, but we saw elevated, in two conditions, either patients who had long Covid like symptoms, who had never had Covid but were vaccinated, so-called long backs and Lyme, both of those had elevations in interleukin eight.

and then also in, in a separate kind of sub, category using these algorithms, we saw elevations in interleukin 13, and these patients, obviously turned out to be Lyme.

So that really led us to what we now have later in this paper, which is a Lyme index. But in between there, we, we, we were able to distinguish Lyme because of either elevations of interleukin eight and interferon gamma or interleukin 13 plus interferon gamma or Lyme patients had a long hauler index twice as twice as elevated, as long Covid itself.

So we saw a long hauler index, which is a composite of interleukin two plus interferon gamma. And I'll explain why that's important in a moment over CCL 4, you know, in in in in long Covid it's between 0.75 and and and three long hauler index for instance.

But when we were seeing long hauler indices it six eight, 11 even somebody with 44, it was almost unequivocally, blind. Right. So, you know, and they just reported a little while ago that the people who've been exposed to Borrelia burgdorferi seem right to be getting more intense Covid symptoms.

And one of the questions I asked you when I was looking at your cytokine panel for diagnosing long Covid, is was the VEGF right, the vascular endothelial growth factor?

And what's fascinating is we see that all the time in patients who have chronic Bartonella as a, you know, symptom that, hey, it's activating the endothelial cells, but you also see the VEGF.

Now do you think there's maybe an overlap with some of these that have Bart or you think it's maybe this the spike proteins kind of affecting the endothelial cells causing VEGF Do you have a hypothesis as to why we're seeing this?

Yeah. So it all comes back to the monocytes macrophage. And we found that S1 protein, persisting in non-classical monocytes in long Covid 15 months, 24 months, maybe even longer.

and and again, these non-classical monocytes bind to, endothelium through the fractal kind receptor to a protein called fractal, which is expressed by endothelial cells.

We have some very preliminary evidence that, we can find Borrelia protein in these non-classical monocytes, which would suggest a very similar mechanism driven by kind of the phagocytosis of, pre pre pathogens.

So post-infection, phagocytosis of or or long lived monocytes that bind to blood vessels both in Lyme and in long Covid. So so let me just be clear. So because I this is the first time I think people really are hearing this, you're actually finding the Borrelia sometimes in these patients inside the monocytes.

You're actually finding it stimulating some of these chemokines and cytokines, making the long Covid worse. That's what you're basically saying. In the absence, replicating bug in the absence.

What we found in long Covid after doing and I haven't seen this from any other group. We did whole genome sequencing of sorted, monocytes for the entire genome of, SARS-CoV-2.

We did that in tissue from long Covid patients before, and now everybody's infected 3 or 4 times. So you don't know, you know, if, if, if any replication is from a, you know, infection last month.

But early on when it was on cycle one, we went and did a whole genome sequencing. And there was less than 5% of the genome, represented in the RNA. these monocytes and in tissue.

and there was obviously fragments of protein, which we also sequenced. and so replication competence is far different than persistence. And it's not necessary.

And we know that from the patients who got vaccinated, who have S1 protein in their monocytes and have the exact same symptoms and there's no virus around.

Okay. So then when we started searching for Borrelia, in these cells and found cell wall, you know, we're doing some more transcriptomics, right now on those cells to confirm whether or not there's actually replication competent bug in there.

But at the end of the day, there's at least fragments, protein and probably fragments of nucleic acids, that are more than enough to cause this, vascular inflammation that we're seeing involved in long Covid and and chronic Lyme right now.

Immune signatures and overlap with Lyme

Now, in the Lyme patients, you know, when we look at the time of reactions and we look at these cytokines that get released, you know, one of the first switches in the nucleus, which is NF kappa, B, you see this massive release of TNF alpha, right?

I one, interferon gamma IL two. While for highly IL ten the whole, you know, so are you seeing when you compare some of the long Covid patients who maybe don't have Lyme or other infections with the ones who do?

Are you seeing higher amounts of TNF alpha in some of these cytokines, like are you able to differentiate just the cytokines and chemokines themselves?

Because previously in the Lyme literature, we knew that CCL 13 was a marker of neurological Lyme. Right. And and Kql 19 John Hopkins said, hey, that's one of the ones you're seeing basically in Long Lyme or right, chronic Lyme TLDs.

But those are the only markers. And and John Hopkins, we can't get these in regular labs. These are just, you know, research markers. So someone like me who's a clinician, it doesn't help me, but could we use your cytokine panel and chemokines to say, hey, this person doesn't just have long Covid, they've got long Covid in Lyme because the TNF alpha is higher or the IL four is higher.

Are we able to make that kind of differentiation? So those are such great observations. Richard, because number one, we just derived a Lyme index to separate it from a long Covid diagnostics.

and in the, numerator of the Lyme index is interleukin 14 alpha. And the denominator is IL two plus interferon gamma, which is the long hauler index. So it very, very clearly distinguishes between long Covid and Lyme.

But it's driven, as you suggest, by TNF alpha. Now what's very interesting in our paper published last February on long Covid, TNF alpha was the major driver for fatigue.

And of course, you know, fatigue is common to all these chronic inflammatory, diseases. In fact, our diagnostic we really feel like somebody comes into your office because you're not gonna somebody's not gonna come into your office and say they have fatigue, and then you're going to send off for $1,600 worth of Lyme testing.

I mean, that's not your your reflex. So I think we really have a screening test to say, okay, which is symptom based. You're coming in with fatigue, brain fog, post exertional malaise.

You know, a lot of people would say, well, you must have long Covid. Well, you and I know better that, you know, you may have chronic Lyme. You may have me CFS.

We don't know. But if we use, our cytokine panel, which was derived by machine learning, we can actually see these signatures. And of course, with this new Lyme index of IL four and NFL for elevated.

In addition, I o 13 isolate the cytokines that are associated with Lyme. We kind of know we better reflex to, speciation in or at least some, Lyme testing on top of it.

so, Bruce, I know you and I know this, but can you explain to people how they can access your laboratory within cells, like how do they get their doctor actually to do these tests?

Because this is going to be really one of the great overlaps of all time. I mean, we'll discuss in a second, like all these chronic fatigue, any patients or fibro that may have other viruses like HHV six, EBV, etc..

we'll talk in a second about how maybe your cytokines can help with that too. But how do people get these tests? And can you explain a little bit about that?

Yeah. So so we're actually launching, late this week or early next week. our new website. health bio a.com, which will have, a portal and, entry, for physicians to order at a testing as well as, patients with a physicians ordering testing.

Right. Now, the the place to order is through, Covidlonghaulers.com with an s and then patients and physicians can order, the tests right there. The in cell kind 14 complex.

Right. So so again that just so people get this but Covidlonghaulers.com. That's how they can access this testing to be able to do different. Right. And and then when we we run those cytokines so you get a cytokine report on top of it.

It'll have the long hauler index. and very shortly within a week or two, we will also be reporting out the Lyme index, with the report and cut offs for, for positivity.

So, you know, are you dealing with long Covid or you potentially dealing with, chronic Lyme? And I'll tell you, the one thing the entire world, is not recognizing is what we published in 2020, shortly after I got back from China, and

that acute Covid causes immunosuppression.

Okay. And and I mean T cells in the single digits when there should be 30% there, they were 5%, 10%. I'm still seeing 11 to 15%. In some individuals. The T cells are exhausted.

They express PD1, Ctla four and three, etc.. And what we published is they don't make the CD, don't make grand CMA. So there was a significant immunosuppression in acute Covid, and it still exists to this day.

And so you're susceptible if you have undiagnosed or inadequately treated tick borne bugs. They can come out and start replicating in the chronic herpes family viruses start replicating again.

and then, you know, finding this new mechanism in immunology where just as, as we've said in the past, debris or parts, you know, pieces of protein, pieces, nucleic acid carried by monocyte monocytes, macrophages is the new mechanism for pathogen persistence.

that could change really the face of any post. In fact, these post infectious conditions where to the point where I feel like sorting the any immune condition, whether it's autoimmunity, these, these chronic inflammatory conditions, sorting these monocytes and doing proteomics, and transcriptomics to figure out are there foreign, proteins, are there foreign nucleic acids, the being carried in these cells for months, if not years, if not decades.

So so you know what's fascinating? When we've been publishing our studies for the last couple of years, when I published one on chronic Lyme a few years ago, in the journal Health Care, where we showed that immune suppression does happen with Lyme because we got about 20% of our patients with low ECGs.

They had chronic variable immune deficiency, 85% had subclass deficiencies in 1 in 3. And in Lyme classes 1 in 3 go up with acute lyme. But interesting. They were down with chronic Lyme.

But the last paper we published about four months ago in microorganisms, we found that the people who had the most Bartonella species, right, two, three, four Bartonella species, they were also immune suppressed and in fact had the worst immune suppression, where they either were on subcutaneous immunoglobulins or they needed IVIg, or they had a chronic, inflammatory demyelinating neuropathy or the worst autonomic neuropathy with Pots just sort of happened in those patients who had Bartonella not just Lyme.

Now, you were previously saying we're sometimes seeing these non replicating forms. these are you talking about you know lately in Lyme what's really come out.

That's been great in my practice is the notion of these biofilm persistent forms, these dormant forms that are all the way down in the biofilms.

Cytokine panels and Lyme index development

Are you talking that are you talking more like l forms, S forms, cell wall deficient forms of Borrelia, but not necessarily the ones. Are the biofilms on.

Yeah, I'm talking even different than that. I'm I'm saying that, you know, as as infected cells get destroyed and there's phagocytosis by different monocyte lineage cells that they're capable of carrying foreign protein.

And we know from the literature that if you, you know, if you tap at that knee that's swollen and you can find, you know, Borrelia, cell wall, but Borrelia, peptidoglycan, I'm saying that those can be by monocytes, macrophages in an even a different mechanism than, than biofilms, you know, and so there may be even more reason why, there is a chronicity to, you know, to Lyme that transcends, you know, replication and why antibiotics become, you know, nonproductive, over time.

So, you know, there's a I think we're in an exciting time, for immunology right now and most importantly, post infectious, immunology. What you just described, Richard, elegantly, was, you know, this humoral immune system

has deficiencies, in these infections and where we came from, just because we started working on where we're seeing these cell mediated immune, deficiencies and this dominance of monocytes, macrophages, from the innate immune response, from these emerging infections.

I mean, it's, we are really getting to the heart of why there may be these this so-called, you know, you know, no one could find an etiology for years.

We're just tipping the iceberg right now on chronic inflammation and and maybe even, autoimmunity. I don't, you know, we haven't ventured there. All I know is we have five shoguns patients, you know, who are always, you know, fraught with fatigue, who are responding really well to our drug combination.

Merab Rock and statins. who, you know, when we relieve their vascular inflammation, their fatigue, their fatigue gets better, and nothing brings down TNF alpha better than a ccr IV antagonists re polarizing, monocytes macrophages, right?

No thank you. Bruce. Bruce, if people want to contact you directly, what's the best email for them if they have like questions for you. Or I mean, the best email is is Bruce P at in cell Dexcom.

and either that or through the Covidlonghaulers.com both are easy ways to, to get Ahold of me. So great. And I keep that up as everybody is getting old.

I mean, it's been a whirlwind of, activity from around the world, really. I mean, we do telemedicine with, you know, Romania, Europe, UK, Australia. So, it's this is a global issue.

and I was shocked to see how much Lyme disease there is in the UK, but. that is, by the way, that's one of those hidden epidemics that's been going on in Europe.

because I've been following the Lyme politics for years. Europe has a really bad problem. And because of all the different Borrelia species, you know, here we have really a sense who's tricked you.

But we're starting to see some apps like Green Eye, you know, Southern Tick associated rash like illness, which they don't even know. The pathogen over there, they've got 7 or 8 different Borrelia species, you know, with valsartan and all of these other ones that if you're just doing it a lives, a Western blot, you're not picking it up.

So they're having an explosion. And by the way, yesterday I'm writing a new paper. I decided to go look and see how many of the people at this point in Europe, in the U. S, are suffering from these chronic, fatiguing illnesses.

And when I put together my fibromyalgia and Lyme with this, we found that it was at least 14%, including, by the way, long Covid. It was 14% roughly of the US population.

that is complaining of chronic fatigue in Europe. When I added together my fibromyalgia long Covid, it was close to like 49%, like half the people living in Europe.

So we've got a really big problem here with all of the politics interfering. And I think, you know, your cytokine panels and the way we're looking at it is definitely it's going to be one of the answers that people are looking for because the overlaps of these illnesses.

What is a virus? What is it bacteria. Do you have both. Do you have, you know, several different things. It's confusing for doctors and that's why we really need, you know, better testing, like you're doing it in your laboratory.

You know, it's so true. And, you know, it's really a disservice that, you know, the press and even the government to say, oh, there's no diagnostic. Well, you know what?

if I was sitting in an important seat, I'd be calling anybody who had anything close to a diagnostic and saying, hey, can we vet this? Can we try this? Can we do X, Y, and Z?

And here we are. You know, about to submit our fourth paper on it. And, we've used it on tens of thousands of patients. and you know, it's nothing, you know, crickets.

And for me, it's a disservice to patients. We are getting patients better. we're very excited. our announcement next week will be that, the FDA has, given us the green light to start our, clinical trial on, crab rock and statins for long Covid, which, of course, addresses, the vascular inflammation, caused by, you know, high, non-classical monocytes and carrying out swan, etc..

So, you know, it's going to be out there, but, I feel like, you know, the word is, is about a year. And a half or two years behind, you know, what we've been doing and, you know, the it'll change next week, right?

You know, so one of the things clinically and you can please feel free to, you know, give more information when we're trying to differentiate long Covid from people with chronic Lyme, one of the things I usually tell people with Lyme, the symptoms come and go.

There's good and bad days, the joint pain, muscle pain and nerve pain, tingling, numbness, burning, stabbing, migrates. It's actually the only disease in medicine that does that.

And we do get neuropathy of course with with long Covid too. but especially with antibiotics, you're going to see Herkimer's cytokine release or improvement.

If you're just dealing with long Covid, obviously with a virus, you're not going to see that necessarily, with the good and bad days and migratory pain.

And of course, with Covid you find a lot of these people with long Covid have lost their sense of smell. They've lost their sense of taste. can you elaborate a little bit more on the clinical symptoms these people are having?

You know, the the interesting thing is it can all be traced back to chronic inflammation. I mean, we've talked a lot about TNF alpha. Well, TNF alpha is a major driver of fatigue from from our paper.

But you know a team of alpha and interleukin one beta are major down regulators of serotonin, which can lead to anxiety and depression. We see that all time long.

Covid is a great few papers on anxiety and depression and chronic Lyme. There's now a paper that I'm aware of on disorder Omega in chronic Lyme. So as you suggested earlier, the the overlaps are even more profound.

But you know, it's the underlying pathology that is also similar.

Accessing testing and interpreting immune suppression

It's similar because it's being caused by different organisms. But at the end of the day, you know, we're seeing very much the same pattern that's ocular inflammation, which causes, of course, dilated blood vessels, headache, migraines, brain fog, ringing in the ears, dilate blood vessels, lower blood pressure, increase heart rate plots.

You know, all these things can be related to, the chronic inflammation. And this upregulation of F is is notorious for causing depression, neuropathy.

So that's what we see in Long-Covid is more of a peripheral neuropathy. I don't see if I see someone with joint in muscle pains. I'm starting to start thinking, maybe Lyme at that point.

because I don't see a lot of it. But that sense of taste and smell I see obviously in acute Covid, that's predominant. I don't see a lot of it in long Covid.

and, you know, I'll have patients come in who have long Covid and they're, they'll say, you know, I don't it my taste and smell, they still have elevated interleukin six, which tells me that, you know, maybe they were on a different

another cycle of acute Covid that went undiagnosed.

So, you know, these days with mild or acute Covid, I mean, it's it's the wild, wild West. I mean, there you have long Covid patients are on their third, fourth round of acute Covid and long Covid, acute Covid immunosuppression.

You know, the chronic herpes family viruses reactivating lyme. I mean really you really have to break this, vicious cycle. And I have this figure in my slide deck which shows infection, inflammation, symptoms, treatment, infection, inflammation symptoms, treatment.

You have to break that cycle. And that's why we got away from steroids. Because steroids are going to do nothing but accelerate that cycle. So, that's why we look towards, you know, the Ccr5 antagonist, which are immune modulators as opposed to, you know, anything that's immunosuppressive, right?

You know, talking about immune modulators. When I was going through the literature, I'm in the process of writing up another paper on, high dose apps, on combination therapy with case studies, because we published a, an article a few months ago in microorganisms.

And before I spoke to you, I made sure I went through the literature to go through every point of the 16 point absence map, because I knew that there were at least 8 or 9 factors on my 16 point.

And it's exactly what you said. It's infections, inflammation, and immune dysfunction in both diseases. The only difference is once a virus, once a bacteria.

But the six causes of inflammation that I find in my lyme patients chronic infections. Right. And they've shown in Covid there can be viral persistence.

In fact we'll talk in a second. They're finding it in sometimes affecting the microbiome of the gut in the intestines. Right. Lowering serotonin right.

This came out a while back. So we're finding chronic infections. We're finding microbiome changes like they are with Covid. We're finding that, leaky gut inflammatory.

You know, changes are there with mast cell disorder, sleep disorders, mineral deficiencies. early in Covid, air pollution. Right. Was a problem because the people living there had the worst outcomes from free radical stress.

And then the downstream effects of the inflammation were exactly the same. They're getting pots, they're getting mitochondrial dysfunction. They're getting hormone disruption, liver abnormalities.

Every one of the 16 points on the message map is showing up in long Covid. So, you know, it's fascinating. You're right that these diseases are following the same track.

And I think it's fascinating that you're one of the only guys who's actually tried to figure out by chemokines and cytokines, how are we differentiated apart from me as a clinician.

Right. Looking at it and, you know, from a slightly different perspective, but there definitely major overlaps. And people are getting of course, both diseases or as you said, multiple courses of Covid, right.

It's it's mixing everything up at the same time. Right. And to not have a means to pass through that, like we do, I think is like I said, it's a disservice.

And, and, you know, one of the big issues is I see these long Covid patients, papers coming out all the time, 10,000 patients in and out looking at symptoms, fatigue, brain fog and post exertional malaise.

Seriously? Not one Lyme test and any one of those 9000 symptoms. I mean, I just every paper I see, I'm like, how do you. But you know that Bruce that's that's medical politics because Lancet Global Health two years ago

published that 14.5% of the global population has now been exposed to Borelli Bergdorf.

Right. But you don't hear anyone saying, oh my God, one out of seven people now in the world have been exposed to Borrelia. Oh boy. This would be a big overlap considering how many people have gotten Covid right, you'd think, and that's medical politics.

That's not medical science. No. And you know, it's true. In and in those first patients that we looked at, they were, you know, having cut my teeth on HIV from 1989 on.

I mean, those first patients, were as immunosuppressed as a patient with HIV. Wow. And probably even Aids. I mean, we saw percentages of a CDA, which work in concert with the CD4 is percentage of CDA as low as the percentage of the CDC, US in HIV Aids.

And you know what? that just brings on anything and everything. I mean, in in our initial court, we had a we had a woman in New York City who died of, cytomegalovirus.

When was the last time you saw that? 1991, 1992. I mean, it was like I said, people are not recognizing that acute Covid causes immunosuppression. I mean.

So so just for the audience, because not everyone's a doctor here. Just explain a little bit about, you know, CD8 and T cells and natural killer cells and why they're so important.

Because I do check them in my Lyme patients. And there's definitely a certain percentage their natural killer cells are low. They're functioning. Natural killer is not working.

Just he explained a little bit to the audience just why this is so important. Yeah. So there's two major arms of the adaptive immune system. We've talked about it.

There's the humoral arm which makes antibodies, which you know, is major, first line against usually bacteria, you know, and then of course there's cell mediated immunity, which is basically CD4, T cells, which, work in concert with CD8 T cells, to form cell mediated immunity, which takes care of intercellular viruses, intracellular bacteria, tumor, and and you know, when, when one or both are, affected, you know, you're going to have sequelae, of whatever made them, you know, abnormal or low.

And we're seeing both of that. And, you know, one of the things that was really interesting by using the CCR five antagonist was we saw an increase in lymphocytes in Covid patients who have run notoriously low lymphocyte count.

All of a sudden their lymphocytes are coming back. and, and also, you know, brought back the CD8 t cells, in 3 to 4 weeks. So, you know, there's ways to, help the immune system recover, which allows it to fight off or continue to fight off these chronic herpes family viruses, which have, you know, a known mechanism for latency.

and, you know, some of these other, organisms, in particular bacteria, intracellular bacteria, which, have their own way of evading the immune system and, and, and forming reservoirs.

So, you know, everything right now. And you know what? sometimes I tell my patients, I don't care what I label it right now, I'm going to treat your vascular inflammation first because that's causing your symptoms.

Fatigue, brain fog to a patient. long Covid patients have hot colon sensitivity, I think.

Persistent pathogens and monocyte reservoirs

So, to chronic Lyme patients because their blood vessels are inflamed, they're dilated, they can't regulate their body temperature. So my quick window into what their blood vessels are doing, if I see him at 3 or 4 weeks, the medications, don't appear to kick in until maybe 4 to 6 weeks.

I'm like, well, what about hardcore intensity? Well, that's gotten better. so that's sort of my window into the blood vessels. But, you know, it's this this is this interrelationship between the two.

whether it's viral or bacterial. I think this, issue of persistence where you can actually have persistence, pieces of the organism in addition to the entire organism, is a new area that we really need to study, because I think that's in play in a number of these conditions.

No. And, you know, in, in Lyme, a couple of years ago, some of the researchers had discovered peptidoglycan, that there were also pieces of Lyme, and, and of course, these are doctors that were not necessarily believing that chronic Lyme is a persistent infection.

They were just basically saying, oh, it's the pieces of the organism driving some of the autoimmunity. And of course, it's never black or white. You can have pieces of an organism.

You could also have active infections. so, so regarding that, you were talking about these viruses with your cytokine panels and you're looking at this, you were talking about cytomegalovirus.

Should we be looking for I look for, for example, HHV six and Epstein-Barr reactivation. And a lot of these patients we should also be looking for CMV.

Are there other viruses or things we should be looking for? both an antibody and PCR testing, let's say through, you know, local labs, not even specialized.

Is there anything you recommend? Absolutely. And and to that point, I, I highly recommend doing, DNA, PCR. So cytomegalovirus DNA PCR, Epstein by Barr virus, dna PCR, v6 back at Stanford I at assay they can look at RNA to DNA ratios which we're we're trying to bring up right now.

So we can tell on that day at that blood draw, you were reactivated because your RNA was three times the amount of your DNA. So it must be reactivation because the serology and, chronic, herpes family viruses is very unreliable.

I'm is is basically worthless for these, chronic herpes family viruses. I, I'm not a big believer in the the titer of IgG making, you know, some sort of, inference as to reactivation or not.

just do, DNA PCR because that gives you two scenarios. You're infected and it's latent, or you're infected and it's reactivated, and it eliminates this.

I've been exposed to it in the past. Okay. You know that that is so fascinating because so many of my patients asked me this question because they're EBV V type HBV six.

There's always these high. And I said to them, you know, normally, you know in immunology we learn to stock. So it's a four fold increase in titers that tells you it's rare.

But you don't see that with these kind of viral infections. So so your point is we really should be doing broader viral screenings. Now I can of course get all right.

The only RNA I do as a alive doc is I'll use, for example, I jynx for a fish test. Right. for the BCR Bartonella or T labs. But I can get those kind of, you know, ratios.

Is Stanford in your lab one of the only places that's doing this? We didn't even get it up, and live at Stanford, but we're working on it. and so because I think it's a game changer, I mean, if you can say at that moment, you have reactivated EBV or you have reactivated CMV, it's so important because what we'll do is if we see the markers of vascular inflammation as cd40 L, CCL, five VEGF, which are all made by activated platelets, and on inflamed, you know, blood vessels, you know, we'll, we'll, we'll treat that.

But if we sense, you know, if there's a DNA positivity, we may add Valtrex to that or a cycle of beer, you know, to

the more Amarok and statins to treat, presumably what's, an exacerbation of me CFS and maybe not long Covid because it's going to have the exact same symptoms.

So again, during that acute Covid phase, if there's reactivation like herpes family virus and oh, by the way, has the same symptoms as well, long Covid, you have to say, well this is an exacerbation of me CFS this isn't long Covid.

I mean I truly believe in long Covid being its own entity. There's no doubt about that. But I think you have to ask several questions when a patient comes in with fatigue, brain fog and post exertional malaise.

A is it, is it acute? Covet? B is it long Covid? C is it an exacerbated, me CFS or, you know, D is it, a Recrudescence? one of the tickborne, organisms. Right.

And and of course, it can be all of these. I've also because in my population, and again with Covid and Lyme, chronic Lyme, the adrenals can be low. If you don't address the adrenals, they don't pick up their energy.

If they have got parts and they've got low blood pressure and you don't pick up their blood pressure. Right. And treat the disorder name you, you control the antibiotics you want.

They're still going to be tired and have brain fog because they're not producing their brain. So it's never a one or you know, in my world it's always an overlap, right?

I tell people the analogy is a patient goes into a doctor's office with 16 nails in their foot and says, I have foot pain. And the doctor pulls out one of the the nails and says, come back in two months.

You and I both know these are multifactorial. It's like there's never one cause for one disease that we learned in medical school. It's now these all these overlapping factors of multiple inflammatory triggers, multiple downstream effects of the inflammation.

So by the way, so I want to get back to the Maraba and the devastating to get this covered. Because again people who have not been following your work just tell them a little bit because they may not realize this is an HIV drug that you're repurposing here.

First of all, how did you get this covered? Because this is a question I've been having from some of my patients, and is the only way to prove that we want to give fact at this point.

Have a stat, and then I'll get to ivermectin in a second, because I think you were using it early on and you know all the controversies about this, if you could just address it a bit, like do you think there's any role at this point for, you know, for the virus with ivermectin or at this point is it mostly of pravastatin?

And and again, how do we get this covered. Yeah. So great question number one. We only used ivermectin when we were testing a variety of different drugs with our panel to see how they influenced inflammation.

I mean, that was three or we haven't used it in three years. you know, I don't know that it had a role in long Covid, you know, seen many different publications.

Now on The Cove.

Clinical symptoms and differential diagnosis

And I just this do not get go there because I think we found a really great combination. and and yes, Brock is not an HIV drug. It's used in HIV because it's an entry inhibitor.

And I think that was a total fluke, okay, that HIV happened to us. CCR five to gain entry into immune cells because you see the Ccr5 ranty CCR five CCL five axis in immunology is so exquisitely elegant.

number one, it tells inflammatory cells where to go in the body in response and which is CCL five or Arantes okay. It tells the immune cells where to go.

by the same token, in cancer, if cancers, we just published a paper on CCR five expression in different cancers because CCR five is also involved in metastasis.

And so you see tumors expressing CCR five and they'll migrate to areas of CCL five expression. And that's where you'll get, implantation of the the met.

The other thing is, in the paper, that I've sent you, the the crosslinking of CCR five on monocyte macrophages and switching the, morphic macrophages from a proinflammatory phenotype, which is promoter pro tissue damage and is what we're also battling in, in local.

And they switch that to more of an effector phenotype. So you lower IL six, you lower TNF alpha, you lower interleukin eight, interleukin one beta, etc.

that's what we want. And and moreover IHC doesn't immunosuppressant anything. It it embellishes the immune response because it relieved the CDA exhaustion in in our earlier studies it, it increased the CD8 numbers.

It restored expression of grands. I'm a at all these positive effects on T cells. And at the end of the day, into fighting off viral infected cells and in terms of fighting off human first cells, it inhibits influx of T reg cells, which come in and shut off the immune system.

So, you know, and you can adjust, the effects of Moravec by dosing. So say in tumor, you want to you want to, inhibit T regs which are expressing Ccr5, but you don't want inhibit the, effector, CD8 cells against the tumor.

But the effector CD8 T cells have much higher receptor levels on the surface of the cell. So by monitoring receptor occupancy, you can you can manipulate, that immune response.

I think that is going to be an exquisite, mechanism for cancer therapy. going forward. But in the meantime, it's also allowing us to shut off, immune responses that we don't want, i.e.

vascular inflammation. Right. and and by the way, Bruce, what's fascinating about the vascular inflammation in tumors, just like with Bartonella increasing VEGF, we know this VG of course goes in with tumors.

But Bart has now been associated with melanoma. It's been associated with inflammatory breast. So we know the virus have been associated with cancer for years.

Whether it's HPV or hepatitis C, hepatitis B, but it wasn't, I think, as clearly defined, H pylori may have been the, you know, the best one as far as B-cell lymphoma is.

But I don't think people have realized that Lyman Bartonella both been associated with B-cell lymphoma is right. and you can see barred associated. So it's interesting.

So the CL five really has a role with all of this, right? As a separate marker. And, Ccr5 antagonism directly. inhibits badger production. Right? So again, it lowers this pro tumor, you know, obviously Badger being a big player in tumor, angiogenesis, of course in Bart, and other, other infections that cause that of Jeff Brock's a perfect drug for that. So.

So again, how do we get it? Can you just tell people like side effects of that? Because people are probably wondering, oh my God, I think I have long-covid how do I get this drug? What are the side effects?

What's the efficacy rates? Can you kind of pull this out a little bit for us? Yeah, I'll just add in one point about statins is we use sub cholesterol lowering symptoms of statins and statins down regulate fractal kind which is the protein that these enshrinement pro-inflammatory monocytes bind to on on blood vessels.

So it's really a two pronged attack. Now in terms of access to drugs. We're starting our CT. We're making a single dose pill, for the RCT, which is a combination of Merab Rock and atorvastatin.

And, you know, the side effects in ten are probably close to 18,000 patients, out there that we've used. It, is very, very mild. We haven't had to take one patient off of Morava Rock because of, side effects, or liver.

And, you know, there's a black box warning for for liver, which was done in HIV patients. So 12 years ago. And then since then there's been a five year safety profile of Merab or Ox have been well published.

And that is a great, great drug and very well tolerated. and, you know, it's, you know, it's a really exciting, opportunity because here we have in a pill form, a very, very potent, immune modulator that, I mean, Ccr5 antagonists are being used in cancer in addition to the checkpoint inhibitors.

And you know, and now we're using it in post viral infections to, remodel, a, you know, damaging immune response. I mean, it's, we're really excited about the future.

And and frankly, that's why we we're not a long Covid company. We are a chronic inflammation company just because of the widespread, you know, finding of vascular inflammation.

And in this, in this great drug combination that, that, that treats it. Right. How long does it take. So again, getting a coverage that you have to have a positive profile from your lab from inside.

And, and how long does it take before people would see for example, a response. So so so what we do is number one the the test is covered widespread coverage, including Medicare.

so we're excited about that. The drug still has, spotty coverage. Some payers pay it, some people don't. You know, we we we write it for inflammation.

Obviously not. You know, for inhibiting R5 isolates of HIV and and and and frankly, we're, we're we're honest about it. We're, we're writing it for this mechanism and we're writing it as, for inflammation.

And it's a way of getting around having to use steroids. So, and, and again, what we're doing in parallel with our RCT is now, going to the payers, setting up our own, you know, drug ID, making our own pills.

and then, we'll be applying for, reimbursement. So by the time our RCT is, done, you know, in the latter part of 2024, we should have, all that covered that.

That's great.

Treatment strategy and CCR5 antagonism

And and by the way, I'm also, if I can get it going, planning on trying to get a randomized trial by the end of this year on that some combination therapy, because I've been I've been having such great results, you know, speaking of DApps, and I was curious when I looked at the acute Covid literature, especially with Ards, and of course, they were using dexamethasone in the hospitals, right, to kind of push people through.

There was a study in 40 patients where they had it, and it was a blinded study, and they put 20 of them on DAP on at 100mg a day, 100 didn't take it. And they found that it cleared and stopped the Ards from actually happening.

Have you have you seen any of this? Because it's the only study I really saw in Covid where they were using DAP shown. because they are obviously is one of the worst consequences.

But it was the earlier versions of the virus that of course were causing this. We don't see it as much in the later version. Yeah. You know, we didn't we didn't get too much into that.

I mean, we're trying to push Ccr5 antagonists in, in acute Covid. And I definitely think they have a role. I mean, in particular, by repolarization monocytes, macrophages, the Il6 comes down in three days.

Right. And if you remember, like everything when we talk about the cytokine storm, you know, the, the, the hallmark of a cytokine storm was this really high level of interleukin six.

And they're, you know, there I think Tuscaloosa Mab, the IL six monoclonal antibody got approved for acute Covid. Well, you know what. Nothing brings out six down faster than CCR five antagonism.

So we're kind of going on that angle. And then as you say, you know, we moved quickly from the alpha variant into Delta and some of the other variants.

And we weren't you know, we got away a little bit, because what happened is we and I got back from China, we started an acute Covid trial very quickly with CCR five antagonists.

And we found, you know, when we followed the patients 30 days, 60 days, 90 days, that's when we found that, hey, you know what these patients were better.

They're out of the hospital. They didn't die. But, you know, at 90 days, their immune system was nowhere near normal. And that's when we used machine learning and AI to say, okay, at 90 days, the immune system's abnormal, but it's abnormal in a different way than acute Covid.

And so we used, obviously machine learning to tell us at different ways. And we got this great separation, this of cluster analysis where long Covid was a distinct separate cluster from a few Covid.

And, we're able to model that. And that was really the beginnings of, that long Covid diagnostic, which we've since, obviously refined with additional, algorithms.

And now, of course, we have the Lyme index to go along with it. Right? So we're coming. Boy, that was a fast. Our you and I promised an hour and went like nothing.

So we're almost actually at the end of the hour. do you have any final comments? I mean, the only comment I would want to make on it is. Congrats, because you've done an amazing job on this.

And and really, I actually I learned a lot every time I speak to you on this, but, any final comments you want to make about, where do you think this is going about the overlap of the tickborne and, and long Covid?

And again, remind people how to get in touch with you and your lab because I know you're going to probably get a lot of calls after this interview. Well, thank you, Richard.

And, you know, I always learn something from you every time we talk about the, you know, Lyme and the tick borne organisms. I'm being a pathologist. You know, I knew superficially, but getting up to speed on, you know, on the bugs themselves, different treatments, etc.

and I think I'd like there to be more dialog between kind of, you know, as I've always said, there's there's the bugs and then there's the immune system, the immune systems causing your symptoms.

Let's take care of that. And then, you know, we need Lyme literate doctors to take care of the bugs. But I think there needs to be more, discussion like we're having about how do we attack it.

you know, maybe at the same time and, and what's the approach? We treat the bugs first. We treat the immune system first. I, I hope that this opens up much greater dialog.

on that front. I'm really excited about it. because it is rampant. And, like I said, I've seen the numbers in the UK and Europe in terms of, you know, Borrelia, other species.

It's it's incredible. and we have to these are treatable. We have to know the difference between long Covid and and chronic Lyme. And, I think we just nailed that with our, Lyme index. So Covid long haulers, is, for right now.

we'll have an announcement later on this week or next week for, our new enterprise. So, thank you so much. It's been a pleasure. Like I said, I always learn something when we talk and, I hope, hope this can continue.

You know, I think, look, this is going to be one of the most important talks in the summit because of how big of course, Covid is at this point. And, you know, from in my world what I learned that when I take home from all of this is I've been developing the 16 point map with, you know, six overlapping courses of inflammation town ten downstream effects, which, by the way, are exactly the same, and Lyme and in Covid because I did the literature search.

But what it says to me is, although the vast majority of my patients are getting better by addressing the underlying immune effects and the downstream effects, there's always going to be a certain subset of patients in this day and age who just doesn't recover to the same extent.

And this really the question mark for all of us then, is, hey, because Covid even could be asymptomatic, right? In some of these people, did it affect your immune system and is it having an effect.

And we're now finding, for example, the mole toxins, which in the last paper we did, it was 84% glial toxins or immunosuppressive. So, you know, as we expand this out, the virus is creating havoc with the immune system.

The environmental toxins are creating havoc. The lyme and Barts creating havoc. We've got all of these factors now causing inflammation causing havoc with the immune system.

And you've got to get to the underlying effects. And this is a really important one that I think everyone needs to be aware of, right. Yeah. Yeah. So Bruce thank you again. This was a fabulous talk.

I look forward for you and I even possibly collaborating at some point in the future because we're seeing so many, you know, overlaps with our. But I just want to thank everyone again for for attending today.

this was the healing from Lyme summit. You're speaking to Dr. Bruce Patterson, and I'm your co-host, Dr. Richard Horowitz. we're talking about differentiating long-covid from chronic Lyme and possible differential diagnosis and treatments.

Fabulous conversation today. thank you again, Bruce. I look forward to speaking everyone in the near future and we'll see everyone soon. My pleasure.