Chronic Fatigue, Brain Fog, And Pain: Are Long COVID And Lyme More Alike Than We Think? Transcript

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Introduction and guest background

Hello everyone. My name is Doctor Richard Horowitz and I am the co-host of the Healing Lyme Summit 2.0 with Doctor Talks. It's my great pleasure today to introduce you, Doctor Bruce Patterson.

Bruce is one of the leading researchers in the world on long Covid. And today we're going to be discussing updates and diagnosis and treatment of long Covid.

So, Bruce, thank you for joining us today. My pleasure Richard. Yeah. So tell me a little bit, tell people a little bit about yourself how you got into this immunology.

How how did how did this whole thing start? Since you seem to have been playing a big role, kind of leading the way now for some of these new treatments.

You know, it's a really interesting, path. When I, when I take a look at it, because I started, in HIV in the late 80s and early 90s. And, you know, one of the major emphasis of my research was looking for hidden reservoirs of, the HIV virus.

At the time, they were even thinking that maybe HIV wasn't due to. Or maybe Aids wasn't due to HIV, that maybe it was a super antigen, a factor or something else.

So, you know, it started my journey on on sort of the reservoir search. And this being our fourth pandemic, my fourth pandemic, you know, I kind of knew where to look.

And I think that's that's where we've gotten to where we are. And, and now including, our newest data on, on Borrelia and Lyme, I think, you know, I'm going back to the old bag of tricks and, you know, looking for underlying mechanisms for chronicity and all of these post infectious conditions.

Yeah. So, you know, at this point, the reason I think our talk specifically is quite important is the Daptone protocol that I'm using for chronic Lyme is effective in the large majority of people.

There's no doubt that these persistent biofilm forms of Borrelia are causing a role in chronic illness. But we are finding long Covid specifically through, you know, in your lab already and diagnostics, we're finding it along with Mo toxins, with the busy and Bartonella kind of overlapping a lot of these patients.

And if they don't seem to get treated or they get a partial response from taps on partial response treating the mold, it seems like the viruses may be playing a role kind of underlying.

And years ago, you know, we would find some viral reactivation with EBV or HHV six. But now since long Covid has been hitting, we're seeing a lot more viral reactivation than we were from before.

And, you know, our new data, is now suggesting that it doesn't even have to be reactivated. We showed in long Covid that, the actual viral proteins and fragments of RNA, were sequestered in non-classical, monocytes through, scavenging, you know, or phagocytosis.

We found that with long vacs, if you will, where we also found, the S1 spike protein in their non-classical monocytes, despite the fact that they were never infected.

And then, you know, the clincher being just from the last, you know, for four weeks or so, we found Borrelia protein,

in one of the patients in our study was had Lyme 20 years ago.

We found Borrelia, USB, C, and, Flagler protein in their non-classical monocytes.

Persistent infections and immune activation

And we're doing the transcriptomics now, but it looks like there's no transcriptional activity of the bug. And the bug may not even be there. There's some really interesting data on, Borrelia in vitro, on monocytes where, they gave they, they cultured monocytes, in a flask.

They, they, they added in live Borrelia and got immune activation, as one would expect. But what's really interesting from that data that really wasn't pursued was the fact that they could put he inactivated Borrelia on these monocytes and got even more, immune activation.

Again, Liberation of IL six, interleukin one beta, TNF alpha, all the usual suspects in a monocyte activation, condition. And he, in fact, activated Borrelia proteins even did more activation than than the live bug.

And and that's what we're working on right now. We found that in chronic Lyme patients. So in addition to biofilms and some of the other mechanisms that you mentioned, I think there's a new one, and that's what we're writing up in our new paper, is that there's a new mechanism for, for chronic Lyme.

And that may be the fact that, you can, harbor, Borrelia proteins in your non-classical monocytes for years, if not decades in the absence, of replication, which may be.

A Bruce question. Question. How are you proving that you haven't scanned the entire body? I mean, look, most of these patients that I see, it is a chronic, persistent infection.

Now, they've published on peptidoglycan before that some of these proteins can obviously get in between the cell layers and stimulate immune reactions.

But at least from my perspective, it's not going to be an either or. It's going to be kind of everything all together. But, you know, just like they just published a couple of days ago that they found some of the spike protein sites on the bone marrow of the skull, right in the meninges.

I mean, they're finding these spike proteins. I don't doubt that you'll find some, you know, inactivated Borelli in some places. But honestly, in my case, because I've been doing this for 40 years with 13,000 people, it's an active infection in most of these people, but it doesn't mean it still can't be some of what you're describing.

Like they could have a very low load of Borrelia. Is that like syphilis? Right. It doesn't have a high load. You may still find some pieces of the bacteria stimulating with only even a couple of life's barricades someplace else in the body, so I don't think it'll be either or I think it'll probably be both.

Yeah, I, I absolutely agree with you. The one caveat is that this particular cell type, number one becomes long lived, when it's presenting antigen and, and it also has a propensity for blood vessels.

And the one thing that I'm seeing that's common to long Covid, chronic Lyme, chronic fatigue syndrome, fibromyalgia is they have this pattern on our cytokine profiling, vascular inflammation.

You know, the Cd40 al, the Kf5, the VEGF, and other, inflammatory proteins that are being liberated by, vascular inflammation, platelet activation. And in particular, we showed in the paper last year that that these particular cytokines, can be correlated with symptoms headache, migraines, brain fog, ringing in the ears, you know, temperature.

If you ask some of your chronic patients, are you cold when you shouldn't be cold or hot when you shouldn't be cold, hot shot shouldn't be hot? They're going to say, absolutely.

And it's because their blood vessels are dilate and they can't regulate body temperature. Similarly, because they're dilated, they're periods where they drop their blood pressure and their heart races, so-called pots.

You know, to me, pots is just a symptom, but you relieve the inflammation. In fact, on on our, regimen, one of the first, symptom complexes to go away in 3 or 4 weeks is, is Pots and temperature dysregulation.

So I think it's this particular cell. Yes, absolutely. Can we find it in tissues for sure. But you know what? It's this particular cell type that has a density to spew out inflammatory cytokines.

And is propensity to bind to blood vessels that I think is particularly pathogenic. So so question for you because I'm using your paddles kind of all the time at this point.

And in fact, I was just discussing with you a few days ago a patient whose CCL five rants was negative, but the CAC 40 was positive, the VG f was positive.

How do we know in these patients that it's the Covid spike proteins that are actually driving it versus Bartonella or both, like, or multiple factors driving a have you able to come to any conclusions?

Because we use VG f as an indirect marker of Bart. It's been associated in the literature. But but obviously you don't know where the vascular inflammation is coming from.

Right. And, you know, at the end of the day, sometimes I tell patients I don't care. All I know is this is causing your symptoms, you know, if I take care of it.

But but in reality, you could have spike and you could have, you know, obviously tick borne, organisms. But you know what I see? I don't truly believe there's an overlap syndrome because what happens is when you have acute Covid, we published this way back in 2020, you are supremely immunosuppressed.

I saw patients dying in February 2020 of CMV, which I hadn't seen since the early days of HIV Aids. Their CD8 counts are as low as the CD4 counts in HIV, and even with the newer variants that don't seem to be as severe, you still get low CD8 counts.

So of course, as you, inferred, you know, the the chronic herpes family viruses reactivate if you had undetected or inadequately treated, you know, Lyme or co-infections.

They come out to play. And you know what they all calls the same symptoms. So it's very interesting. But we have a test, that detects the spike protein and, all the monocyte lineage cells, that's, that's now, commercial.

So I'll use that, if I see something unusual, in terms of the cytokine patterns and like, for instance, high il6, which is very atypical, I think in long Covid relative to acute Covid, then I'll go look for spike protein, in the monocytes.

And yes, I may see a spike protein in the monocytes, in somebody who has a past history of really a Bartonella.

Monocyte reservoirs and chronic Lyme mechanisms

But so, yes, I think there could be a multitude of factors going on, in fact, and islands, I call this cell type, the human garbage can, because we've only looked at three instances of, of, of, of sort of protein fragments in these cells.

But you know, I know these cells for a fact, since I worked with them, are infected by HIV, by, Zika virus, dengue fever. And truly, I think they scavenge, I think they're a garbage can.

And that leads to ongoing, long, long periods of immune activation. And at the end of the day, we got to empty the garbage can. Right? So, you know, by the way, regarding, the immune problems, we know that there's T-cell exhaustion with long Covid, and we we definitely see the CD4, CD8 counts drop.

Natural killer cells are oftentimes low in these patients. Their B-cells are also affected when they get lime and Bartonella. So I mean, essentially we're dealing with people with chronic variable immune deficiency subclass deficiencies and T-cell deficiencies.

And it's no wonder, right. They're getting so sick with all of this because they've got more toxins on top of the infections on top of spike protein. So yes. This yeah, there is a lot of things.

And you know what the other thing is, you know, we no one does it clinically. But if you look for exhaustion markers, PD1 one Tim, three Ctla four on, T cells, I mean, they're there.

These cells are exhausted, you know, so, you know, and we published in 2020, there was T-cell lower T-cell numbers, especially CD8. There was, T-cell exhaustion, and the T cells weren't making, grand Zima.

So, you know, there's three reasons for them to be completely exhausted, not doing the job that they should be. Right. So so question. So in the testing that you're doing through radiance when we get it back.

It's interesting because a lot of my patients, when they've done some combination therapy, we know that when you turn on TNF alpha, when you turn on NF kappa B and you get T and alpha L1, l2 il6 on your panels, it's true.

I'm not finding a lot of TNF alpha and IL six on your panels. What we're mainly seeing a lot of times is the CCL five wrenches, the VG app, the CC 40.

You finding that in a large number, are you seeing some of these active Borrelia patients with the IL six and TNF alpha. You're seeing a mix of this when you're looking at it. Yes.

And in fact, in the paper we published in August on, a long Covid diagnostic with, differentiation from chronic Lyme. In fact, the Lyme index, which will be launching, clinically, here and probably in the beginning of the new year, the numerator of the Lyme index was interleukin four plus TNF alpha.

So yes, TNF alpha. In in in the truest sense is, is a marker that leads us more towards a Lyme diagnosis than, than a long Covid diagnosis. But, you know, you have to also differentiate between, you know, a Lyme diagnosis and acute, Covid, where you do see the interleukin six and TNF alpha is because of the, you know, immune activation.

But we don't see that much anymore because usually people have been either exposed to the vaccine or exposed to the virus multiple times. So your, your, your adaptive immune system has kicked in and you're no longer, you know, the the first wave was just all innate immunity.

So of course you saw the interleukin six anti-TNF alpha. You know, really taking a dominant position and and doing a fair amount of damage in addition to trying to fight off, the virus.

And, you know, this. Is this begs the question, and I don't want to get into vaccine controversies too much, but it does beg the question whether the Novavax vaccine, which is a different type than the messenger RNA making spike proteins, if we're now finding spike proteins in, you know, bone marrow of the brain and meninges and all over the body, should we be going back to more of a classical?

I mean, obviously I've gotten vaccinated in my patients gotten and not everybody gets sick. I've seen some patients reactivate after the boosters. Fortunately, not that many after they've gone through that zone.

But it's a question. Do we have any way of like knowing which people are going to be more sensitive to getting spike proteins, whether from the vaccine, whether from the virus itself?

Is there any way to differentiate? I don't know that we can at this point, but it's it's a question. Other people have even brought up other retroviruses that even transform some of this. Right.

And that's a big question that no one's answering that, you know, it's a big one for the literature. Yeah. It's a, it's a

it's a big leap. You know, again, like I said, one of the reasons we developed a spike protein, not a spike antibody, but a spike protein test, and found obviously this reservoir, which now serves as a great marker, is to be able to answer those questions.

You know, are you retaining spike and and certainly, you know, we see it when when patients have symptoms, we do see patients who've never been infected by Covid who just got vaccinated and and have spike protein in their in their monocytes and have long Covid symptoms in fact, it's one of the things that I rely on to say you don't need active viral replication, to have long Covid and and I've said this all along, and there's no way you can prove with patients getting infected five, six, even 7I1 patients seven times.

It's a Covid I have. I have one also, by the way, seven times. How can you tell if you find active virus. Oh that was the one that caused long Covid for years ago.

Or it's one from last week. And we did it with not with, whole genome sequencing. You know, in our first persistence paper where we showed there was no complete genome.

I mean, yeah, you can get partial transcription and everything, but you know what? You never could make a new viral particle with you with 5% of the genome represented.

We showed that in tissue of long Covid patients as well. So you know our our what we would say is you can't make a building with 5% of the bricks. And but nevertheless we found this mechanism where you don't need, active replication.

And that's what we're pursuing right now. There. New paper umbrella is you know what? We don't need active replication, to to cause symptoms and to cause, and, chronic inflammation.

So, so question on this regarding the virus with because there have been studies showing that sometimes the virus right can persist like causing inflammatory bowel.

Right where they found active virus in parts of the body. Your cytokine panels and chemokine panels have a way of telling how many are just pieces of the virus.

How many are active virus. Any way to tell at this point? Our first paper, which I don't think people quote enough, is that we showed with two algorithms, a completely separate population, of patients who were in D long Covid, and we did it without any, any bias towards symptoms.

And then we found the ones who had either mild acute or severe acute, Covid through what we call the severity score. So yeah, we, we know which ones have a immune response that is, consistent with, active replication.

But the fact is, how are they proving active replication? Not by PCR, not by in situ hybridization. Only way to do it is to sequence or to grow it. Because we caused a stir in our first paper when we found using droplet digital PCR, which is supremely sensitive on in plasma of acute Covid patients, we could find virus and of course, all the blood banks in the world called me and said, do we have to worry?

Much like this reminded me of the late 80s and in HIV, do we have to worry about Covid being transmitted by blood? And so we collaborated with a number of different groups.

We took the plasma from, from these patients who had virus detectable by digital droplet, and we could not grow it.

Cytokine patterns, vascular inflammation, and symptoms

We, the best virologists in the world couldn't grow it. We couldn't grow it. And you know, what whole genome sequencing was, was spotty. We didn't get whole genomes.

And that's the kind of work that has to be done to make that huge, leap of faith from persistence to replication

competence. The problem is, I think the world interchanges those.

And that's wrong. Okay. Yes, I agree there's persistence. You can find fragments of S1, you can see fine fragments of RNA, any tissue you look at, you know.

No, no questions. Ask whether or not it can actually make a new viral particle. I have supreme doubts that it can. So that unless it's a branch. Right.

So that part is not proven at this point, even in the ones you get all of a sudden inflammatory bowel with Crohn's and ulcerative colitis, it's not clear whether there actually is as again, just pieces of the virus are active.

I mean, we are finding a lot of EBV. PCR is in the blood, HBV, PCR. I mean, that would definitely seem there's no there's no doubt, no doubt. No doubt.

But you know what? Now that we're showing this new mechanism, I think it's a new mechanism in immunology where the fragments of protein and maybe even the fragments of nucleic acids are sufficient to cause an abnormal and a deleterious, immune response.

I think we're into a whole new arm of immunology where, you know, maybe autoimmunity is caused by these retained proteins. We never found it because we're doing genomics and everything.

Right. So you know, we're I think we're only at the tip of the iceberg in terms of chronic inflammation and what those underlying mechanisms are. But you know what?

From now until I'm done doing research with I don't know if I'll ever be I'm I'm looking for non replicative, reasons why we have chronic inflammation from past, past infections.

You know, it's interesting because you're warning that there was my studies that were done years ago when they were looking at xeno diagnosed, and they found in some of the mice with Borrelia that they had what looked like initially non replicating forms.

And then eventually they found that some of these forms actually were replicating later on, but they were dormant in the body. And in these cases they were probably biofilm persistence they were looking at.

But as I said, I think it's going to be all of the above. I have no doubt, because peptidoglycan has been talked about years ago. I think, you know, from my perspective with the inflammation, the the bees, for me, the brilliant but easier Bartonella are on the top of the list with toxins like mold and metals driving inflammation.

And then they get leaky gut and food sensitivities with mast cell activation and microbiome issues. They're not sleeping. So their il6 is high. They got vitamin mineral deficiencies.

And then they have all the downstream effects with these free radical oxidative stress, with mitochondrial dysfunction and hormone dysregulation and HPA axis like you're talking about.

They can never get, you know, they're cold. They're hot because their hypothalamus is affected by. So there's a lot of there's a lot of causes of why the inflammation is there.

I think what's important and we'll see this because I'm about to start a randomized trial, hopefully also on that soon. By the end of next year, we're applying for NIH trials.

And by the way, not that I have to speak to you today about it, but we plan on using your profile in studies we're going to be doing because we have to figure out when I'm going to be looking at Adapt.

So in randomized trial, the things that are interfering most with the success of what I do is active. The busier day sweats, night sweats, chills, flushing, air hunger, unexplained cough, Bartonella pain in the bottom of the feet,

these that are changing colors and moving around the body.

Severe. Neurosci. Psychiatric symptoms with pots. We we find a lot of active bartonella species. But then we've got long Covid kind of following right behind with mold.

And so we're dealing with all these immunosuppressive things. And I have to figure out in a trial to look at chronic Lyme how I'm going to separate out these different things.

I don't I don't think it's easy because we see so many overlaps. I just have to figure out what what I do in the trial to eliminate the worst long Covid cases.

I think everybody at this point is going to have some exposure. Would you at this point say they should not have, you know, a CCL, five rants or an SDC 40?

Like, how would you if you would just wanted a pure trial for active replicating Lyme active, what would you leave out of the side? What would you not want to see on your panel in my trial?

Well, funny you should ask because we're doing the exact opposite for our trial. Yeah. So, you know, we're enrolling that. So to answer your question, long hauler index has been so, robust for us in determining who's got long Covid and who doesn't unless it's over six, where that's almost 100%.

Lyme. Okay. And and and so we're doing the same thing to rule out Lyme and, EBV reactivation in our long Covid trial because we want to cure population of long Covid.

Everybody's lumping everybody together. And they're saying, oh, well, 28% of my long Covid patients have EBV. That's not long. Covid, right. You can call it Pasc, which is post-acute sequelae of Covid.

That's fine. But it's not long Covid because the SARS-CoV-2 virus is perfectly capable on its own without EBV, without line of causing a post infectious condition.

Right. So we're doing the same thing. I'm ruling out Lyme. So if I see elevations that in my, you know, my three bullet points for Lyme elevated interleukin eight plus interferon gamma, interleukin 13 plus interferon gamma or a long haul or index greater than six.

They're getting excluded from our trial. And because of something else, we're doing EBV, DNA, PCR for EBV, not serology, because I think it's difficult, if even possible, to say whether or not there's reactivation of EBV on serology. But, you know, we are doing the exact same thing from the other angle.

And I think I think it'll work out well for you. And all I would look for is a long hauler index. So. Okay, so so now there's just if the long hauler index is high.

Yeah. So you talking about interferon gamma IL for, you including VEGF there.

Testing strategies and distinguishing long COVID from Lyme

Then when you do a. Long hauler index is IL two plus interferon gamma over CCL four. So you can imagine in in line which is extremely high interferon gamma that you're going to have really, really high long hauler indexes.

So when I see a long hauler index greater than six or even nearing six, I reflex to, to borne diagnoses. So that's what that's what I'm, I'm, I'm looking for and and long hauler index actually interleukin two plus interferon gamma over CCL for you know what rants.

Yes is important was it was extremely high early on in acute Covid. But you know what Mahavira Kf5 antagonist targets so much more than than Randy's. That's the obvious right?

But there's papers direct, lowering of VEGF by Ccr5. Okay. Antagonists. You know, direct lowering of S cd40 L by atorvastatin, which is our combination direct lowering of of, interleukin six and ten of alpha by Morava because it re polarizes the proinflammatory monocytes.

So there it's so much more than just kind of blocking CCL five, which is obvious. So I, I don't that's not a criteria for me to choose therapy. My criteria for choosing therapy is number one this triad of vascular inflammation as cd40 LKL five and fed Jeff.

Or you know, signs of monocyte macrophage activation interleukin six TNF alpha. So that's that's my criteria. So going back to going back to Bart for a second.

So you know, we can always find most of our patients have some Bartonella immuno blot showing prior reactivity there. Bart fishes aren't always positive.

We don't even bother with if I could do direct droplet PCR from Galaxy, I would love it. But in New York, I can get it. How do you know when you're using Miraval Rock and you're going after VEGF in that point?

If, let's say, Bart is hanging around in the background and it's tricky because there's so many species, you're you'll be lowering down the vascular inflammation.

But if the cause of the vascular information is still there, right, the Bart may just still be there and cause it to come back months later. You're going to you're going to account for that in the trial?

No, you're absolutely right, Richard. And, you know, we, you, as I tell my patients, I'm like, you have to treat the bug and the immune system. It's the immune system, as we showed last year in our publication that's causing the symptoms.

But at the end of the day, the bugs driving the symptoms. And if the bug is still active, you're you're running your headache, you're hitting your head against the wall.

So, you have to treat both. And, you know, we'll, I mean, again, we're going to do some screen. We'll probably do some, some if we see even a suggestion or an elevation of Jeff, we won't just pass it off.

We would probably then go on to screen those individuals for for Borrelia. Bart, the easier before we enroll them. Yeah, you'll probably find, I mean, Bart the marker for Bart for the VHF being apart from cancer things, of course.

God forbid anyone came in by accident. But the. Jeff, I would say it's probably not more than maybe 25% of our patients with Bart at some point. You know, the Jeff goes up, but we have seen it as a marker and then we treat it in a and it does go down and there better but but but definitely the long-covid pieces, you know, it's created a, a monkey wrench for me in the last couple of years because we were having such great.

And by the way, what's interesting about capstone, which I have to prove, it's an anti-inflammatory, right? It decreases Milo oxidation. There's a whole bunch of anti-TNF use for autoimmunity.

There are people that will argue, well, how do you know you're just not lowering inflammation like with Miraval Rock, for example, working on specific pathways.

The only reason I think it'll show it separately is because we're going to use other persistent drug combos, like we'll use rifampin and methylene blue and other persistent drugs without dapt shown in one arm of the trial, to see if we can see a difference in the results.

But yeah, just be kept. Bart, Bart is showing up in the vast majority are just look. And by the way, I'm happy to look over before you go forward with this.

Yeah. You want to send me over and just brainstorm with me? I'm just like, I'm going to brainstorm with you. But

before I get complacent. Absolutely. Because, I mean, you get one shot at these things and you certainly don't want to make that mistake.

So, Yeah, I mean, I think, as our trial comes together, I mean, our, our primary outcome is, fatigue score. And the secondary is, disorder. No Mia score.

But, so we'll we'll start having presumptive enrollment based on a certain fatigue score, then we'll start ruling those patients out. And that's that's when your phone's going to ring for me.

Asking if, if if we've gotten rid of those. But you know, what can it's conceivable that we could get a couple, but we don't know. Yeah. So why don't you just tell people I mean, you you and I, of course, know about norovirus and atorvastatin or pravastatin, but just for the group, why don't you just talk about there was an article that just got released about, I don't know, maybe two weeks ago with hundreds of variables that they were finding in long Covid in these chronic, fatiguing illnesses.

Meribel Rock was one of the ones on there. So is glutathione, by the way, that I did see the p value actually look pretty good. Because we published the first article on low glutathione and Covid in April 2020.

But why don't you tell people about the dosing, what you're seeing using this and what they might be able to expect? Yeah, I was I was really excited about this article from, I believe it was from Stanford and Harvard on on patient based responses to, various interventions for long Covid.

There's probably like 15 of them. And and yeah, Mahabharat had the highest rate of sickness and improvement, in patients of of all the different interventions.

And it was also the only one that didn't have like it didn't make patients significantly worse in some instances. So yeah, we were very pleased by how that came out.

And of course, it was compared to, you know, pacing and IV fluid, which is just, you know, you're just just putting a bandaid on symptoms and but ADHD medicines, LDN, gluten file, I mean, everything under the sun.

I think it was really well done. And, you know, it came out as a leader in terms of significant improvement. And, you know, we stick with the 300mg, twice a day of Maverick, dosing, because that allows us to go through the 500 5B2 path, of the FDA, since the drugs are approved for, other indications.

And then statins are extremely important. As a, as I mentioned, it directly lowers a cd40 l and, it also lowers a protein called fractal kind, which binds these pro-inflammatory monocytes, to the blood vessels and to the blood vessel wall, where they then extrapolate into tissues and bring their inflammation with it.

Treatment approaches and trial design

So, it's a it's a really great combination. And on top of it, moreover, OC of course, re polarizes the proinflammatory monocytes, keeps them from migrating, all over the body.

But the other function Morava act which is key is the Lymphopenia post infectious Lymphopenia is a CCR five dependent pathway. And when we noticed, early on and in 2020 that Morava OC was increasing lymphocyte count, patients who had low lymphocytes and increasing Cd4+ T cell counts in patients who had low CD8 T cells, which was almost all of the long Covid patients. So, it's got a really exquisite, mechanism of action.

And but there's still people who think it's an antiviral antiretroviral and nothing could be further from the truth. So, that works out. Well, you don't need a lot of stat.

And I think that's the other important thing about our protocol is we're not trying to lower cholesterol. We're trying to lower, inflammatory, proteins related to vascular inflammation.

And so, you know, we'll go with low levels of, of statin. So we don't have patients who have a lot of side effects. Sometimes we'll add in colchicine when patients aren't progressing fast enough.

And by the. Way, what point would you do that's normally at least a 12 week protocol. Correct. It's a 12 week protocol. If, if, if if they've plateaued, you know, at eight weeks or so or they're not progressing as quickly as we would want them to, then right around the eight, 6 to 8 week mark, we'll add in, 0.6mg of colchicine and that that is, a real kicker to get them across the finish line.

Great. Right. And of course, for those of you who don't know, colchicine, it's a very old drug from 50, 60, 70 years ago for gout. But but but essentially has anti-inflammatory properties.

Absolute. And it does that has a little bit of gi side effects. But the Moroccan statins really don't, don't do well. Now, we haven't really seen a lot of side I mean, I have occasionally sensitive patients, but for the most part I'm even though it's a low dose of statins, like ten of atorvastatin or 10 or 20.

Do you still use CoQ10 with it? Or you don't even bother? I don't, you know, I don't care. Yeah. I mean, I usually have these patients come in with a laundry list of supplements and and really, there's not a I'm fine with them taking the supplements, especially CoQ10.

And you know. That statins have been shown to lower it, that the reason I'm asking is they've been. Shown for sure. For sure. But, you know, in 6 to 12 weeks with a low dose, I don't think it's necessary.

But, you know, I I'm also not going to try and dissuade anybody from taking them now. So what happens? So if someone gets to 12 weeks, and they have not improved the way they would or you would expect to do, you then go back and go, well, gee, if it's not just long Covid, maybe there's other factors driving inflammation.

Like how does it work as far as the protocol, the way you're looking at it. For sure. And, you know, if they get to 12 weeks and, and and they aren't, progressing, with pure long Covid, that really doesn't happen.

But but certainly that's when I start looking for other things, looking for, you know, Tick-Borne looking for some chronic herpes family viruses. And now, you know, I've been trying to get this study with Neil Nathan off the ground.

We're going to look at mold. I start looking for other things in these patients. And that brings up another point, in terms of mold is, you know, with our 14 biomarkers that span kind of, many different arms of the immune system, there's 89 billion different combinations of those.

Am I biostatistician? So the ones who've developed all these, these algorithms for long Covid for chronic Lyme and I think, you know, I think we're anxious to develop an algorithm for, mold.

Funny thing happened when we were developing an algorithm. For me, CFS is they did more or less like a tree, you know, basically like you do with with genes or gene sequences.

They did a tree of immuno types. And me CFS, we found, is at least five different diseases, five very distinct, different diseases. With. Immune profiles completely different.

You're looking. At them. We have we're working right now on what defines these five immuno types. And it may be more than five immuno types. But to say me CFS is one disease is is is now, not part of our or our thinking.

No. And and of course, the problem with patients is the symptoms of me CFS and fibromyalgia and long Covid and chronic Lyme overlap as does mold, in fact.

So the difference with Lyme is usually the symptoms are coming and going with good and bad days and migratory joint pain, migratory muscle pain, or migratory nerve pain.

The tingling, numbness, stabbing, burning that's classic for Lyme. It may also be an overlap with Bart, because

when we first did our study and published the statistics in 1600, people, when we did this in 2017 with the State University of New Paltz, there was no way of knowing how many people really had Bart, because we didn't have good Bart fish testing at the time.

But, you know, so they made me a part of Bart also. But that's the only way I can differentiate, honestly, whether Lyme is there versus these other diseases and whether it's being driven by other bacteria, viruses, parasites, even fungi, I mean, the inflammation can come from multiple sources.

It can. And, Richard, I'm with you. Those last three, conditions that you mentioned really steer me towards, you know, Lyme or the co-infection, which is joint, muscle pain.

You know, paresthesia is, you know, and certainly the striae in, Bartonella are. Still other questions. A lot of researchers and doctors out there who were talking at this point about, platelet activation, they're finding, you know, the virus in, in the platelets or pieces of the virus with platelet activation.

They're using natto kinase. They're saying these micro clots, we've got to open up the micro clots to be able to get to it. There's vascular inflammation where they're using or curious all or other things, you know, for the vascular inflammation.

And you mentioned LDM, mast cell activation. People using par dialing. There's a lot of things out there that people are trying at this point. When you looked at that study, I still need to go through it because there was so many p-values all over the place.

I need to look at it. Is there anything else you're finding when you've looked at the literature about this idea of there are these micro clots holding on to pieces of the virus with the and the fueling dysfunction, you know, with the mast cell activation.

Are you finding anything in there that is showing up in your research? You know, the great thing is it's all in this paper. If you want to look at the efficacy of Neda kinase, it's right there and in, in living color.

So, and we'll stand by that data. But, you know, we're really not and you know what? The more time goes on, out of 12,000 patients, we haven't had a single one with a clotting event.

And, you know, it's because we immediately get them on, statins and lower as cd40 L, which is the first protein in the thrombotic pathway. So and we we essentially relieve the, platelet activation, using a combination rivaroxaban, statins.

And you know, those all those markers tend to go away. But you know, I don't all right. And being a laboratory, I'm very cautious about micro clots. I mean, you take blood from anybody who has inflammation, you put on a slide, it's going to clot if you put it, if you run any blood through microfluidics, where most of the diagnostics these days are microfluidic, you form micro clots.

And then when I saw pictures of them, some of them look to me is pathologists like these epithelial mesenchymal transition cells or EMTs. They're they're common in cancer.

But I think they're also common in chronic inflammatory diseases, especially ones where there's tissue damage. And basically what happens is epithelial cells from from organs binds to, monocyte macrophage lineage cells and forms this kind of glob in the blood.

And we could see them when we were detecting, circulating tumor cells. But you see on there is EMT, they stain for everything because the monocytes just Vegas.

It has an antibody. So the nonspecific staining is ridiculous. So I, I have a really hard time believing in that. And like I said, none of the anticoagulants ever worked for us when we tried them.

3 or 4 years ago. And like I said, our our, protocol keeps working. So, you know, I don't know that any of these things, you know, we we described our mechanism for endothelial.

It is now almost three and a half years ago.

Closing remarks and contact information

And I'm not we haven't really ventured far from that because I think, you know, for for everything we can see it's working and we have a non subjective means to, to show that it's working.

So, the only reason, by the way, I don't think we have seen micro classes because everybody in my practice has been on an acetylcysteine with good advice.

And it turns out in C 16 lowers von Willebrand factor. I didn't realize it at the time. And and that was of course an issue with the early Covid viruses, with the variants, right, where people were dying of those clots.

It's true with the latest versions and the variants, I don't know if we're seeing it as much. It's very difficult to compare. Yeah, some of these people had long Covid from the initial variants, from Alpha, from what's going on right now.

Exactly. And, you know, I mean, we saw a lot and there's no doubt clots were happening in the acute phase. There's no doubt about that. I just think that, you know, 90 days later and in long Covid, we just we just didn't see a lot of it unless they got reinfected, of course.

So, and that's always the specter is this, this reinfection. And, and yeah, there was this window of lung that this, you know, someday when, when the books written, you know, between February 20th, 20 and June of 2020 was a hotspot for long Covid.

And, someday when we have time, we may go back and look at our samples from then and try and figure out why that was. I know, in our persistence publication, we showed that in some severe acute Covid cases, we started to see, spike protein and, in, in intermediate monocytes and, and some in non-classical monocytes.

So we're starting to establish a reservoir of of protein in, the monocytes and some of these severe acute cases, but we haven't really pursued it to date.

With, now that we're on the precipice of this trial. Great. And, how many people are you planning on enrolling in this trial that's going to go forward?

So it's, 252 patients, three sites will be making an announcement here in the next few weeks. And, everything's ready to go with, the drugs being made as we speak.

And, you know, I it should take, right around 28, six weeks. So some sometime, you know, by by the beginning of summer and we have an eight week, interim analysis.

So, we'll have a good idea of how it's going. That's great. Well, congratulations on getting this done. And and, so for Bruce, for people that want to contact you, that may have questions or want to use, include radians, how do they get in touch with you?

What's the best way to do that? Yeah, I think the best way is, the, health, but, yeah. Helped by our i.com and, of course, obviously Bruce at in south.com is how most people go and I can announce it because everybody uses it anyway, but, yeah.

You know, that's like me trying to keep my private email private at this point. Yeah. Yeah, I'm well aware. Exactly. Yeah. No I have yeah. Another family email.

But and then of course the chronic Covid treatment center, which is Covid long haulers with a Nasscom, is how patients can enroll and and and visit with our team of physicians and and we'll manage them.

So, that's how to get Ahold of us. Great. Well, thank you, Bruce, this was a great talk. I think people are going to get a lot out of it because this is a real it's an overlap we're seeing in our chronic Lyme population.

I, I, you know, I think you're doing really essential work here. So I want to thank you for being on those frontlines and exploring it. Just like I've been exploring the biofilm for a while.

You know, everybody's got and all of this is going to come together at some point in time. I enjoy these conversations because we can kind of synthesize kind of, the first attempts at putting this very complicated issue, together.

So I really appreciate it. Yeah. So everybody, again, you've been listening to Doctor Richard Horowitz and the co-host of the Doctor Talk Healing Lyme Summit 2.0.

You've been listening to Doctor Bruce Patterson. Bruce has been one of the experts on the frontlines with long Covid. Bruce, again, thank you for taking the time today.

And we'll see you again for another episode soon.