Cytokines; The secret molecules of inflammation and practical advice to reverse persistent symptoms Transcript

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Introduction and Dr. Pattersonu2019s Background

– Welcome welcome uh we're gathered for another addition so to speak of overcoming long Covid and CFS and today it's exciting information packed time.

I have Dr. Bruce Patterson with me. And Dr. Patterson is head of I believe it's in cell D. X. I. and a company that I have been using for a year and a half and I have to tell you it's transformed you know my practice.

Dr. Patterson is a viral pathologist and was head of neurology at Stanford Medical School and has I feel been amazing um Trailblazer in understanding and also offering some treatments for long haul covid and also for a lot of the other chronic inflammatory diseases that we call chronic fatigue and fibromyalgia etcetera. So let me just start off and say Dr.

Patterson tell us a little bit about you know what was your aha moment in the beginning you know covid starts and you had some background in looking at viruses.

So what what got you going? – Well yeah after spending the better part of 25 years on virology and viral pathology starting with HIV. I mean it just so happened that was in the maybe the wrong place at the right time. I was in china in January 2020 and saw some of the first data I was actually at an immunology lab that did car t therapy for cancer.

And saw some of the immulogic data from patients with SARS Cov two and it was very clear obviously that you know it was heavily innate immune system driven macrophages disease, something that we really hadn't seen because frankly the infected individuals bodies hadn't seen these emerging in.

So, you know, it was a very unique profile with an unfathomable amount of inflammation. And then basically when I got back to the States, we started looking at a variety of different drugs in trials of acute covid.

And one thing that came out from our work, which we published in 2020 was patients with acute covid immune suppressed, highly, highly immuno suppressed.

They, there's CD eight counts were as low as CD four counts in HIV patients. CD being the partner with CD four's and fighting off viral infections and cancer.

So, it was that was probably one of the first aha moments that these people are immune suppressed. The second big one was that after the trials when we followed up with these patients, you know, they were better, they got out of the hospital. They didn't, they didn't die.

Early COVID Immune Findings and Long COVID Signature

But at 30 days, 60 days, 90 days, by no stretch of imagination was the immune system back to normal. It was in complete chaos and we used machine learning and ai to show that the chaos in these patients was very different than the inflammation and acute covid. So we identified the first signature of long covid, which was really vascular inflammation elevation of um, SCC 40 oh via Jeff TNF alpha. So, so clearly still driven by activated macrophages, but clearly using our algorithms, we could tell the difference between someone with long covid and somebody with acute covid.

We also published a paper at the time it showed that there was viral replication potentially out as long as as three months. So there's 34 month window there might still be some viral replication. But uh and you'll see a mixture of

acute and um long covid phenotype. But however you get into the 68, 12-15 months and it's clearly its own entity immunologically.

And and that's where we started. And then we started searching for why is there this vascular inflammation. – Yeah. Just to get people's attention that this low CD eight is a signature that we've often seen in you know, you know, a chronic Lyme and chronic fatigue.

People with Epstein Barr virus activation. So low C. D eight's our gateway in a way to chronic illness. And this is just – One thing we found that's in that paper is that these patients with SARS COv two infection, not only were the CD eight slow but they didn't make Grands I'm a which is kind of like their bullets for killing the you know, the virally infected cells of the tumor cells. So and we also found immune exhaustion which was later corroborated. So you know, it was a whole immulogic mess um immulogic chaos in the early days of acute covid.

And then like I said, when we followed these patients, they just they weren't their immune system was nowhere near normal in some of them in 60 to 90 days.

– And when you start to look at the people who became who we began to call long covid, What was the description? You know like the the discriminating factors that you saw between the acute and the long covid people, – We'll see, most of them were, you know, they're going to their physicians and they can't find anything. So this was the summer of 2020 and um these long covid patients were being ghosted by everybody because nobody could find anything.

And one of the reasons is they were using you know, crp which is actually a surrogate of aisle six, but the L six is indicative of acute covid and tends to be less prominent in long covid. And so you wouldn't see crp or they've used some other um test that like um said rate for a measure of inflammation.

And and so what we had done, we we ran over 100 and 50 to 200 biomarkers during acute covid. And then like I said, followed these patients up at 60 and 90 days and we found that um um the signature of those markers changed and we had machine learning and ai choose which ones were the most powerful in our model. And what was indicative of long covid, – Which are the ones that show this vascular quite vasculitis or at least a vasculopathy.

Yes. – Well you know they like the new term is which actually probably the most accurate which is endothellitis. So and then we started looking at funky named markers like fractal kind and fractal kind receptor. And we started looking at monocytes because in HIV towards the end it was I mean not towards the end but towards the end of the nineties were starting to realize that you know they don't it doesn't just infect t cells.

It infects monocytes and thesemonocytes are long lived. And it was the intermediate monocytes. And we I published a paper in 2009 on the these intermediate monocytes also carrying hepatitis C.

In patients who are infected. So you know I regarded them as as as basically a viral garbage can and the Vegas it ties which means they eat up you know everything including virally infected cells that are dying off. And you know sure enough we found that s one protein in those cells by one technique. And then we sorted those cells and used mass spectrometry which is highly highly specific to identify that these cells indeed held fragments of S. One and fragments of RNA.

And this was two years ago people are talking about every day now we found it two years ago and published it. And then we did whole genome sequencing and showed that it was indeed just fragments.

There was not enough of these viral fragments or viral um nucleic acid to make a replication competent virus. – Can you amplify that a little bit because this is something that's really important these days.

And people you know we do know that the D. N. A viruses and retroviruses can be can stay in the body and still be replication conflict meaning you can they can reproduce and make more of themselves. – They have true latency.

Right and so RNA viruses typically don't. But you know the fact is You know we we used a technique whole genome sequencing to answer the question is are these fragments and persistence of fragments?

The answer is yes we found that at 15 months.

Viral Persistence vs Replication Competence

But we went the extra yard to say are these capable of being replication competent? And I think that's the only place. So a lot of people are corroborating our data right now with you know you know residual virus viral fragments, fragments of protein fragments of RNA and tissues and cells which is very satisfying. I think the only thing they think um is that that's different is they think there's actively replicating virus at least our data hasn't hasn't shown that now we published a paper showing replicating virus out to three months.

So if you had a long hauler it said oh I've I've been a long hauler for 90 days or 120 days. Yeah I bet there's a proportion of them where you could find replication competent virus.

But you know when we were looking at 12, 15 weeks, 18 weeks. Yeah we could use PCR and even artistic chemistry and in sexual hybridization. Some of these techniques that detect small segments and find residual segments or fragments of protein and RNA.

But when we actually sequenced both in cells and in tissues from long haulers we found that less than 5% of the genome was represented, meaning in our minds you can't build a building with 5% of the bricks. And again we still are adding more and more um tissue samples to our registry.

And will soon come out with a paper showing our sequencing results using whole genome sequencing. – So getting noises here. Two questions about that one. So in other words there can be remnants of of RNA that that that can be transcribed but there's not enough but there's not enough to make a virus, there's enough to make a protein.

– Yeah. Some of these groups talk about negative strand which means that they're in the process of making a new viral particles that could be done on fragments and like I said, you know the whole world sequences every tumor that ever comes out of anybody. And for the life of me, I don't know why every single tissue from long covid patients.

They're not doing sequencing to show if there's any um full length replication competent virus. And you know what if I were doing a trial with um you know, packs livid which again of our 36,000 patients really doesn't work uh in a lot of long covid patients. But I'll be interested to see the results of that trial. But the fact is no one's really shown a there's actual replication competent virus being made here either by sequencing or growing it.

You know, we we came out in 2020 and said, Oh we found uh SARS COv two RNA in plasma and we were actually doing plasma viral load before and after uh the therapies we were using in trials and the fact is we could detect plasma viral load.

And I remember discussions with blood bank directors etcetera and saying, hey, is there a risk here? Are we at risk of transmitting you know, SARS Cov2 to through the blood supply. That was a big topic.

And I would say February and March of 2020 when we were using, you know, digital droplet R. N. A. I mean PCR to detect RNA in plasma and we were finding it. But everybody under the sun took samples from us and as did we and tried to grow it, we couldn't.

So, you know, it's a big stretch to go from, okay, there's viral persistence. Yeah, absolutely. We published it, you know, a year and a half ago. There's nucleic acid persistence, there's protein persistence. But it is a big stretch to then say I can make a replication competent new virus that is capable of causing disease.

– Yeah. Of infecting someone. And I guess you're the the holdout that people hold say is that if we biopsy different tissues, we might see find a replicating virus and that's your challenge to the world to say, well you're getting

biopsies, why aren't you doing this?

– Right. I mean we're looking all over the gut. I think a gut is a good place to start. And I think you know there's been some papers that come out using immuno autistic chemistry which only tells you that there's protein there doesn't tell you that all the proteins there. It says proteins there.

Right. Great, beautiful, nice stain, you know. No surprise there. So but the fact is then extrapolating and saying in the discussion that oh this shows that there's you know that there's persistent virus meaning there's replication competent virus.

That's that's a long stretch. – Yes. Well yeah, but thank you because this is this is an extraordinarily confusing subject and I thank you for helping clarify at least what we know, you know, at this at this point in time. And so now talking more about um just a little bit about the monas sites because they again, monocytes which become macrophages and tissues. So people give some idea because most people have heard of macrophages in our in our world. So tell us a little bit about you know quick maybe a quick overview of the three different types and like what what you have found.

– I think it's important because um number one like I said we found other viruses in these monocytes we found excuse me my lab we were able to infect intermediate monocytes with HIV we found hepatitis E in intermediate monocytes, Zika and dengue fever are carried in intermediate monocytes. So you know this is something that is really I think underlying chronic inflammation and you know all the unmet needs out there in terms of treating in in in managing chronic inflammation whether it's post treatment Lyme disease fibromyalgia, chronic fatigue, long covid. You know all of these are we found similar patterns and then patterns that made them unique.

So what we were able to do is develop a diagnostic that said I can distinguish between post line chronic fatigue long covid which I think is invaluable because they're all treated differently and when we're about to launch a clinical trial and to think about mixing those you know and having you know a placebo have E.

B. B reactivation when and get symptomatic when in fact they're on a placebo. I mean these are types of things that can really damage a trial. You know because placebos are not just you know, healthy individuals. I mean you don't know what's happening in them if you don't rule out, you know whether or not they have uh line, you know, Epstein Barr the other herpes family viruses um when you're designing the trial as exclusion criteria. So I think it's extremely important.

So we divide the diagnostic then only not only said, well you have a long long hauler index of X, Y and Z. You must be a long hauler.

Monocytes, Endothelial Inflammation, and Biomarkers

But also we did another study which showed that if we mix samples between post line um chronic fatigue, long covid post fax patients with long covid symptoms and acute mild and severe acute covid that we could tell the difference. And that is enormous.

And then we use that on this last trial which I presented at CAssie on over three 300 patients on our treatment regimen um for 6 to 12 weeks pre we measured being complete sided kind profile before and after therapy and came up with a p value of less than 10 to the minus sixth. So we are ready for our clinical trial.

– Yeah. That that is very, very, very exciting just for I want to go. So I want to go to places but let me finish one first. So at this point you also have a test, you know that looks at whether a remnant of the s one protein is in the monocytes and these are usually found in what you call the nonclassical monocytes because you…

– And the intermediate sites. – Okay. – But what's interesting is um we first came out and said, hey these nonclassical monocytes carry S One. Well why is that important?

Well it's the nonclassical monocytes that their sole job is to patrol the blood vessel. And so when they bind to fractal

kind through the fractal kind receptor that they express, they bind the blood vessels and cause blood vessel inflammation.

Just like we've been talking about this endothelialitis and they're long lived because they're expressing an again, so they're a pathetic pathway uh is short circuited and they don't die. Everyone is like oh Dr. Patterson, they have a lifespan of one week. That's true.

Non pathologically, you know? But the fact is when they're carrying an again, you know, they can be long lived, they were long lived in in HIV as well and considered a long lived reservoir. So the fact is the if you look back in the early two thousand's in the afro sclerosis literature, believe it or not, there is a lot about CCR five which we use the more abstract the CCR five antagonists um S. C. D. 40 L.

Being the first protein expressed in the throne biotic pathway um CCL five and SCC 40 L. Are expressed by activated platelets. So that's going on. So yeah we came out and said these nonclassical monocytes, you know, are elevated in long covid and by the way, you know, a significant proportion of them may be carrying S.

One. And that was corroborated in that recent Yale paper that they also found elevated nonclassical monocytes. So again, I work from a year and a half ago is starting to be corroborated.

And I think really the only, the only issue I have is this definition between persistence and replication competence. That's it, you know. So… – Is there a difference between the, I mean because the nonclassical monocytes carrying the S One and is there any difference in cause or origin and result?

If we find a lot of them in the intermediate monocytes as well. – The bottom line is by treating them, they tend to die off because you short circuit their pathetic pathway. That the problem with those two cell types is of course they travel through the blood brain barrier and carry information all over the body.

So that's why you see the neuro inflammation that people are identifying in long covid and and some of the narrow changes because the blood vessels in the in the brain are inflamed as well. So when you treat that um that's when you see, you know the deal and a lot of symptoms are caused by vasodilatation.

Because when the blood vessels are inflamed, the vessel dilate so long Covid patients. I always ask this because no one else ever ask this. Do you have hot cold insensitivity invariably they say yes.

How did you know? And I said it's because your blood vessels are inflamed, they're dilated, dilated blood vessels drop the blood pressure. What happens when your blood pressure drops?

Part goes up? Wow, that pots, you know, So everyone's talking about sort of the end result when in fact we've been looking at the cause and then treating the cause the same thing with micro clots. I mean literally we haven't had a single patient with clots in our 36,000 patients. And it's because we're treating the elevations in the expression at S. CD 40 L.

And platelet activation using statins and CCR five Antagonists. So, you know, we we stopped that rather than sucking the clots out, we stop the fact that they're even being made. And I think that's that's the strategy that we've deployed successfully.

– And I think that's the exciting part has been the treatment options. You know, looking looking at the different sub fractions, you talk a lot about, I mean, the CCR five which is and the s the sizable cd 40 ligand and veg F. That's a trio.

I said that when I've been doing these tests I've seen in almost everybody. – Yeah, that's it, that's the vascular inflammation triad. So obviously our goal is to get those three markers down to normal and then the key differentiating markers for instance are um you know violate we see that in post treatment Lyme disease and patients post vaccination who have long covid symptoms but you rarely see I'll eight in true long covid. So our ai and our machine learning use that as a differentiating um hub. We have this cytokine hub paper that's out And we

have the cytokine hub algorithm. We have a long hauler algorithm.

We're in the process right now getting FDA approval of the algorithms by partnership with another company. And the hope is that by getting FDA approval on the algorithms that we'll be able to really be a lot more successful with reimbursement. That would be that would be good.

– But I said but these you know I guess in my because I treat chronic illness and I treat the people who as you say have been ghosted for basically 30, 40 years now because their regular blood tests are normal.

They have learned that they unfortunately have to pay out of pocket to get the tests that actually are going to show them something. But it's a hard sell for most Americans who have been you know until they got long covid have been able to you know operate successfully with conventional medicine and um more or less and but when you when you have something where your blood count and your chemistries are normal, unfortunately you have to start looking harder. – Again, but we also we also have a responsibility to keep pursuing better reimbursement. I'm not saying they don't get some Canadians, don't get reimbursed for the tasks they do because of one insurance or another.

But the fact is I think an FDA approval. And then we can go to the basically the payers and and and look for for a code to get those reimbursed for long covid. So that's been our plan all along exciting.

– I hope that happens. That will be quite wondrous. The but the post vaccine now you see your what it's interesting that you're seeing um you said I. L. A. Because I said I've seen I. L.

A. Again in my chronic Lyme patients. And I think I think even some overlap with some of the chronic fatigue people. But you're saying any anything else that helps stand out in the post vaccine people.

– Well again, I think they have the classical, you know, blood vessel inflammation. But you know, I do think you know, IL8 is the key thing for post facts long haulers.

And I think you know, whenever I see IL8 you know and other markers that are in our hubs, you know, we we know how to treat them. And you know, I had some person rebutted interview of mine one time saying, oh we don't need a diagnostic for long Covid. Well, you know what when you have all these very prevalent conditions with the same symptoms. Yes.

You do need a diagnostic for long covid. That's like saying, oh you have chest pain. I'm gonna treat you for a reflux because you know, you don't have a heart attack. – Yeah. And to be fair, is that many people who, you know, present with long covid when we talk to them, you know, and and we do the testing. We find that there's also the element of the recurrent you know, D. N. A viruses, whether it, you know, most commonly. – Yeah. – The herpes family viruses that, you know, their immune system had been dealing with, you know, effectively until they got Covid. – Great. And then they became immuno suppressed and then reactivated and they got these symptoms that's now all over the press about what are the symptoms of long covid.

And the fact is, yes, they are the symptoms of long covid, but they're also the symptoms of post treatment Lyme disease, the symptoms of chronic fatigue and the symptoms of fibromyalgia.

Right? So, you know what maybe if they have lined, you know, we've found since we launched the hub algorithm in the spring, we've found between 30 and 50 patients who had line who never knew they had line. Okay, just from their immune profile and then they get sent to be tested for line. So they have untreated line but they have all the symptoms that someone would long covid has And they came to the physician because they thought they had long covid because those symptoms are all over the press.

I think, you know, it has been a huge benefit to patients with chronic inflammation across the board because now we're recognizing that people with chronic line, chronic fatigue syndrome are suffering just as badly as patients with long covid. And they've been searching for answers for for many, many years. – Yeah. No.

Diagnostic Algorithms and Treatment Strategy

And it helps so much because for, you know, for 2025 years we've we've been treat some people, for, you know, like the chronic, as you say, the chronic endothelial inflammation, you know, with various various remedies.

I mean, low dose heparin was something like we used a long time ago and there would be subsets of patients that would respond beautifully to this. Except, you know, it's not good for long term use.

But the point is is that, you know, we just haven't been able to measure really well. You know, who, who's who and I think this is really, really important.

Are there any when like, you know, what else? I mean, we also see some people who look, we think have mast cell on top of this with very elevated IL fours and sometimes IL tens. Are you are you seeing that or is that just or…

– We have to be careful because I think I'll four and IL 10 are also compensatory. I mean they're immuno suppressing the cytokines. And and if you have inflammation, invariably the body's reaction is gonna be to try and, you know, increase those two sided kinds fact is, you know what?

We have no qualms about somebody taking Zyrtec and Pepcid with whatever we're treating them with. But on their own, they just don't seem to be working very well. – Oh no, their there band aids, they're there, they're just going to quiet down uh irritated mast cell population, but they don't make anything really better until you deal with the underlying triggers. – Exactly, – That's keeping it going.

So are are you you know, um in your therapeutic approaches, you know, how quick, you know, are you using much in the way of the platelets stabilizing medicines?

Are you sticking more to the the statins and mara brock? – No, we're treating the underlying endothelialitis. And that's the bottom line, because frankly, if you do that, then, you know, you don't need the plan. You know, I can maybe I had maybe five patients feel better on Plavix out of 5000 or more that I've seen personally. So I just don't see that it's helping that much and we know there's studies out there where they just tried that and and that didn't work? So you know what it's all about?

Are you going to treat the cause? Are you gonna treat the result? And I think any kind of clotting as a result of what the underlying pathology is and those are the pathways that we were targeting. – Okay, so going back then too, like you mentioned fractal kind and practical receptors and I'm fairly certain that our listeners have no idea what those are. So just a little bit of like how the statins and more of a rock work in that would probably be helping these, – These monocytes populations have all kinds of receptors on them and that basically tell them where to go. I mean that's what randy's or CCR five and CCR five are all about telling immune cells where to go in the body to sites of inflammation.

So the other cool thing about cross linking CCR five with mara brock is that re programs monastery to macrophages away from being pro inflammatory, so away from making TNF alpha which we found is the number one side the kind associated with fatigue IL2. And and the p values are 10 to the minus fifth.

That TNF alpha and interleukin two are associated with fatigue. And so um the morale of Iraq does two things it keeps itself from migrating and and potentially binding binding to blood vessels all over the body.

And the statins decrease fractal kind expression so it decreases the receptor by which non classical mono sites bind to blood vessels? So and when we block that then the apoptosis pathway of these cells re engages And they die. So and we've shown that in serial um serial samplings and serial testing using our new S one S that's available. – Okay.

So basically when the when the monocytes isn't able to stick to the blood vessel wall, it then tends to follow its normal lifespan. – Correct. – It doesn't, okay. When it sticks to the once it sticks to the blood vessel wall it has a little tendency towards maybe not immortality but a lot longer life. Okay. – And also you know basically getting

through the wall and causing inflammation. – Right?

And so you by using the statins you're short circuiting that. – Right. – Right. – And we're also looking at new non statin drugs that do the same thing because obviously there's a there's a group of patients who can't take statins can't tolerate statins. So we have some alternatives that we're currently working on right now. – Okay. Anyone you want to mention to people or..

– No. – Okay. And and as far as the Morava rock that's always been that's been a hot button for a lot of people because of the you know the supposed black box warning on it and things of that sort. But I know in our experience it's been benign.

– But a single liver enzyme elevations. Thousands and thousands of patients, not a single liver enzyme elevation. We had one patient during acute covid who had taken almost half a bottle of Tylenol and got huge elevations of A.

S. T. And A. L. T. If you ever want to compare the labels on more of a rock and Tylenol you pretty much see a xerox copy. And so yes it's been a very safe amazing drug and there's actually a recent study out of U.

C. L. A. That mara brock increased memory in mice. So I think it also has a very positive neurocognitive effects – Lowering the inflammation – And it's not a steroid and it's not immuno suppressive.

There's a lot of people early on and said Oh uh you know it's just you know it's a immunosuppressive and it's not it's a new modulator um and it's not an immunosuppressant.

– Yeah and its effect as far as you know it was really pretty limited to the CCR five receptors. – Yeah it's very specific. So we have a number of different um receptor occupancy assays that we've used for different clinical trials over the years. In fact we were involved in With fighter in the early 2000's in the original para Barack trials doing essays for them.

So yeah it's you know I've been working on CCR five since I think the mid nineties in in HIV and then obviously have expanded that lately. It's very interesting. It's sort of like you you were the right person at the right time. – Yeah someone you know in in the right place at the right time because I was in china. So someone else told me that the other day, – Yeah, it lined up a lot of, you know, uh you know the right the right knowledge and the ability to to look at the problem because you know, again, not many people unfortunately, you know, immunology seems to be hung up on you know, B cells and T cells and and we there's the stepchildren, I don't think people appreciate enough how little we understand of immunology.

– And then the other thing I was saying early on in the pandemic is um we have to use this knowledge of of the early days of Covid as we go forward and we face uh new emerging infections that will also engage the innate immune system.

So we need to know find out ways in CCR five antagonism was one of them that we can address some of the harmful elements of the innate immune system when other emerging pathogens come out because the time until you get pathogen specific therapies, whether it be vaccines or drugs or what have you is an extended period of time.

And so we're gonna have to do something that's broad and broad spectrum in general. And I kind of used early on to the early interviews when I was doing acute Covid, I was using the example of cancer where we found PD L one, you know, which is now the you know wonder drug of immuno oncology, the PD L one inhibitors. Because by blocking PD L.

One they bring the immune system back to fight the tumor off. Right? And that's the most powerful tool that we have. What is that? Well that's manipulation of our immune system to um let's call it a favorable light.

Right. And I think the same thing can be said for um CCR five is expressed on um you know, T regulatory cells. So dousing is important. So tier egg cells shut off the immune system.

They turn the light off PD L one brings it back on and then you use you know then then the T rex come in and and basically turn that off so that you don't get auto immunity.

Well now we're using combinations with the PD L. One inhibitors to inhibit the T. Regs to prevent auto immunity. So these are ways of manipulating the immune system in cancer. Why aren't we thinking about this in infectious diseases? And like I've been saying for the last two years since we came out with these tests, why aren't we looking at covid and long covid with precision diagnostics, you know that tell us what to do.

You know when we do telemedicine, we look at follow up labs and we see what's going down what's going up, we know what the target and you know that instead of just throwing a wet towel against the wall and trying the next, you know, the next uh faddish therapy, we actually see what's going on with the immune system and we can target different side of kinds. I mean, TNF alpha, interleukin six CCL five veg F, S cd 40 L. They're all lowered by uh CCR five antagonists that's in the literature and has been for a long time.

You know, TNF alpha is the target of Humira for, you know, inflammatory bowel disease. Well, you know what, TNF alpha also causes fatigue. I mean, not that you're going to use, you know, something as powerful as Humira in long covid, because frankly more of a rock does a great job on TNF alpha because it's made by pro inflammatory mama sites. So there's a, you know, there's this is this is the path that in Saudi X is focused on, both on the diagnostic side and on the therapeutic side.

And we're very excited. We got our first regulatory approval with the C E I B D mark on the, on the side of kind kit. Next up is um, FDA approvals, um, you know, on the kits in the United States.

Post-Vaccine Cases, Reimbursement, and Clinical Trials

And then, you know, we're anxious and aggressively getting our clinical trial organized for mara brock and and and and staff and sort of staff and substitute. – This is so exciting and so important.

And I just want people again to remember that, what we're seeing here again is that it's not so much the bug that got you sick. It's your immune response to the bug that's keeping you sick. – That's my message.

When I'm talking to a Lyme patient, I'm talking to a chronic fatigue patient. I'm telling them, listen, you know what, especially crime, I don't know what bug causes, but you know what your symptoms are being caused by what you see on that piece of paper. And what I'm gonna do is I'm gonna make those proteins get better and you have your symptoms go away.

Then we can deal with the bug is a bug activist bug not active. You know, same thing with line, you know, does it still need more treatment? Doesn't need treatment. You know, I think um, all of that comes into play, but that's why it's so important to say what it is.

You know, their immediate response. I guess what, you know, when they hear about what we're doing, trying to parse these chronic inflammatory diseases apart is, oh, it doesn't matter. You know, and they're lumping E B B positive patients in on um, on long covid, you know, investigations and, you know, and then I was like I said, I was happy to see that Duke, you know, took, um, I don't know if it was, you know, whether they heard me when or where it was in the paper, but we've always said, you know, what you got to exclude line from doing any investigation on long Covid because they are almost identical.

So – And that's just so important because once again, it's, you know, treating, you know, chronic fatigue and lyme for 20-30 years, you know, in the beginning, you know, you think you have to treat the bug and you know, I always say there's about a third of people that when you treat the bug they get better, but there's two thirds of people, it doesn't matter how hard you treat the bug until you get their immune system working appropriately.

They're not getting better. – Right. – And right. And, and so once, once you get the immune system and in our world we often do a lot of detox and things of that nature, which will help, but you know, it's just, but when you get the immune system no longer kicking you to the curb and actually functioning normally, then you often get these

bugs to just quiet down because we co evolved with a lot of these things. They're not all our death to, you know, to death enemies. You know, we can live with them, but we have to be able to control them and that means we have to have a functioning immune system. And for some reason, um, bugs like Covid really can throw a wrench in it. I mean that that I think we – We know they dropped the cd 8%. We know they cause immune exhaustion.

We know we show that that decrease the amount of grands I'm a and then in the cd eight sir magic bullet. So you know what, it's, it's, that's all out there right now. – So it is, yes, this is just such important information. And I really hope that, you know, people listen to this and share it with their friends because um, you know, I can only, I just want to mention, I mean there was the, the article and there was an article, I don't remember which journal, not journal. It was a magazine that I was just appalled by, um, that we just took such cheap shots they at your work.

– And yeah that was a year and a half ago, – And it was, it was just this is the ignorance that under the guise of medical, of investigative medical journalism that I just, I just appalls me where people, they just wanna be insulting, they don't really understand what they're, what they're writing about.

And I just think that I just beg people to, you know, when you read something, you know, don't believe everything you read, but at least, when people are, when medical, when medical journalists are talking more about the price of the test, um, and how nice the doctor was and not looking at the science, then, you know, okay, you can listen to that, but don't, don't let that sway you buy what therapies you're gonna try, I mean you really have to look a little harder and that was just like yellow journalism and you know, – It was at its worst and I think you know, we didn't comment on it really, but our patients were obviously very vociferous about it.

I mean nothing about our credentials. Like we were some doc in a box and you know, in you know rural America, you know, telling, we've solved long covid, I mean it just wasn't the case. We published everything.

So it was they were peer reviewed, you know, um because but they never mentioned our peer reviewed articles, right? So um you know, again there is a lot of that early on I think now you know, we got a regulatory approval in europe for our test will soon go on to get regulatory approval in the United States.

We're gonna, we're quickly taking the drug through clinical trials were doing everything by the book and we're not afraid to because we are doing things the right way and we are using precision medicine. So, you know, at the end of the day, you know, that was early in the game. And yeah, we get out to a really fast start in terms of figuring it out and you know, it's one of those, whoa, you know, let's let's take a look at what's going on.

And there really wasn't much information that they really knew, but like like we were writing the, we've never written a single prescription or we were doing the test in our offices, know they were being done by licensed reference laboratories that fall under Clia. So you know, there was just so much that was in there and then to say that, you know, I was just some middle aged white man in a in a lab coat. I mean then it gets personal.

Then you really wonder what the motives were and you know, we later found out who commissioned it. But um, you know, that's neither here and there there. We have a we have a job to do.

– Well on just one more note, I don't want to keep you too long. But um, just as far as I could totally off subject but packs love it.

Paxlovid, Blood Vessel Protection, and Closing Remarks

What what are your thoughts on that just done on, you know, where do you, – I think, you know, frankly, and I've had, you know, literally once things opened up, I started giving um talks at conferences in person and my first two conference talks in person. I got Covid and I hadn't had Covid.

So, um, but I was very much I took Paxil did. It's worked for me both times. Then I follow it up myself with more of a rock and statins for a month to protect my blood vessels and I'm now nine months out of my first um infection that not a single sign of long Covid. I think one of the keys to acute covid and potentially even preventing long Covid is protect the blood vessels to protect the blood vessels, protect the blood vessels.

– On that note I think that's we'll wrap up I think that's the important thing is how important the blood vessel inflammation is and how much it has to do with a lot of chronic illness and just one more important thing that is why you don't have to have the infection in your brain to feel bad. Okay I always want to remind a lot of people who we panic that they have um these you know severe infections in their brain and those can happen but they're rare. Most likely you have inflammation that's affecting the blood vessels in your brain and it's gonna look quite the same and you know and so again to thank you and if you know just for people to understand is that in cell D.

X. And is you have a website. Right? – So the website for our program is Covidlonghaulers.com with an S. And that includes once you're on there you can say your Lyme or chronic fatigue or fibromyalgia or post backs whatever.

You have that option. But it's Covidlonghaulers.com – You know and I do feel that getting that test really gives you a leg up on what to do and how to treat yourself. And just remember again nothing works 100% of the time. I mean I thought that clear is that I I have found the in cell D. X. Has to be really helpful and the treatments really helpful. But there are still some people who need more work. I mean it's I I just want people to understand that there's uh you know for some it's a panacea and for some it's just a step up and there's still more to do. There's more to look at.

So Dr. Patterson thank you so much for your time. – I really appreciate it – And again for your work and I'm just so glad that you were thinking about monocytes at the right time.

– They're my favorite. Thank you.