Introduction and Guest Background

Well, welcome again to the Parkinson's Solutions Summit. I'm your host, Dr. Ken Sharlin. I am very excited today to interview a special guest, Dr. Todd Levine, one of the co-founders of CND Life Sciences.

He is also acting as their chief medical officer. This is a very, very important interview. Listeners and viewers are going to get some key information that really may change the way that they think about Parkinson's disease from the diagnostic perspective and how the diagnosis is made, but also potentially from the therapeutic perspective, which I think is at least equally, if not more exciting.

So without further ado, welcome, Dr. Todd Levine. Thank you for having me today. Dr. Levine, before we dive into our discussion, you tell the viewers a little bit about your background and your company.

Great. Well, I am a neurologist and I did my medical school at Duke University. I then did an internship in medicine and a residency in neurology at Washington University in Saint Louis.

I then specialize actually not in movement disorders, but in diseases of the nerve and muscles. We call that neuromuscular diseases. So I spent two years after my residency doing that fellowship.

That was it. A period of time when a new technique was kind of brought to the world, and that technique involved taking skin biopsies and looking in the skin to look at the nerves.

So as opposed to studying the nerves through sort of indirect tests or physical exam tests, it allowed us to really visualize the nerves for the first time and in a very convenient way for patients.

And so I had started a lab to do that, which then began to receive specimens from all over the country, from doctors, neurologist, primarily to study these nerves.

Now, about ten years ago, while that lab was developing. My two other co-founders, Roy Freeman and Chris Gibbons, who are professors at Harvard Medical School, they were doing similar work as I was with the skin, but they began to ask the question, Could we look inside of the nerves?

Could we see what's happening? Not just with the nerves and looking at overall nerve health, but could we actually look inside of the nerves to see what changes were happening?

And they began to study this protein called alpha synuclein. And so they began to publish on the fact that, yes, using that same very simple skin biopsy technique, they could look inside the nerves in the skin and they could detect alpha synuclein in patients that had a group of diseases that we call synuclein opposites, and that includes Parkinson's disease, multiple system atrophy, dementia with Lewy bodies and pure autonomic failure, as well as a prodromal disease called REM behavior disorder.

And so about seven years ago, the three of us got together over a beer in Boston and said, hey, let's see if we can actually now start to bring this technology, which was being developed by many labs across the world.

Could we bring it to patients and neurologists in the U. S.? And so we founded CND Life Sciences, CND Life Sciences spent about three years perfecting the technique.

And so for the last 3 to 4 years, we've now been offering commercial testing so that patients and doctors outside of research trials could now have the ability to visualize this abnormal protein in patients that might have these diseases.

Wow. Yeah, it's very interesting because while this is a relatively new test, the the idea of alpha synuclein really goes back to the earliest pathological observations about Parkinson's disease.

Lewy Bodies and Alpha-Synuclein

Can you talk just a little bit about the Lewy Body and. Yeah. Absolutely. So you're absolutely correct. It's about 100 years old. So a pathologist began to study the brains of people who had passed away from Parkinson's disease.

And he found a very characteristic finding. And that finding was inside the nerves of the brain. There were these blobs basic, I'd like to call them blobs, because just think about a collection of stuff that is built up, and that was called the Lewy Body after him.

His name was Louie. So that has been the sort of pathological definition of the disease for a century now. No one really knew what the Lewy Body was until really about the 1990s.

And at that point, because of the development of a number of different techniques, it really became clear that that blob was, in fact, the accumulation of a lot of proteins.

And the way I like to explain this, because it's really unbelievably important in what we call neurodegenerative diseases, is that in our bodies, the thing that does what we need it to do.

So whatever function you need to do inside your cell, that is all done by proteins. And so we make proteins, the protein goes and does the thing it needs to do, and then you have to degrade that protein.

And so it's a continuous cycle. And so you can imagine that there can be some diseases where you're not making the right protein and therefore it can't do the thing that you need it to do.

And we call that a loss of function, or you make that protein and it doesn't degrade. And then that protein is just going to build up and build up inside of the cell.

And at some point, there's so much of that garbage in the cell that the cell won't be able to survive. And the term that we use for that now, we call it protein apathy. Yes.

And so the most common neurodegenerative diseases, Alzheimer's disease, Parkinson's disease, multiple system atrophy, dementia with Lewy bodies, Lou Gehrig's disease, they are all related to this concept of some poor regulation, of the protein synthesis, the creation of that protein, and then the protein degradation.

It can be different proteins in each disease. So the protein that causes Alzheimer's disease we don't think is the same as it causes Parkinson's disease.

But when you look inside the brain, there are still these accumulations of proteins that shouldn't be there. And so in the 1990s, they discovered a a genetic mutation that caused Parkinson's disease.

That genetic mutation was in the gene that coded for the synuclein protein. And at about the same time, using newer techniques, we determined that that synuclein protein is the main protein inside the Lewy bodies.

So since the 1990s, the belief is that Parkinson's disease is what we call synuclein apathy. So protein apathy is all the different protein diseases and then synuclein apathy says there's something wrong with the regulation of the synuclein protein.

It's building up and is becoming toxic to these nerves. Yes, it's absolutely fascinating. And, you know, especially now with the FDA approval of Lacan and Mab, the pending approval probably of Donanemab with Eli Lilly just releasing its Phase three trial data, which was stunning to say the least, particularly for the most early stages of Alzheimer's, which would be mild cognitive impairment without impairment of activities of daily living.

So basically there's not as much destruction of neurons and therefore removal of this amyloid protein, the protein that we associate with Alzheimer's disease appears to have a huge, huge impact on disease progression.

And I hope we see the same with ALS, with multiple sclerosis, with ALS, where the TDP 43 protein and with M. S. is a new one, relatively new one for me, as I'm very interested in this overall subject.

But the bassoon protein that is at least associated with the neurodegenerative phase of multiple sclerosis appears to play a major role. And it you know, it's a little bit of a digression, but it's an interesting, you know, observation.

It makes you ask very, very big questions about what, you know, sort of who's in control here. And there's an old theory called the Selfish Gene theory, But maybe this is really all about the selfish protein, and it's the way that cells protect themselves against, if you will, mistakes or malfunctions.

I'm a big Star Trek fan, and I always say that, you know, the Enterprise and all those starships have various levels of defense, you know, the phasers and the photon torpedoes and all that stuff.

And the end, they're their shield. But in the end, in the end, you know, if all else fails, the ship is capable of self-destructing. And so maybe these diseases really are the end point of multiple attempts by the cell to manage various incoming signals that have either resulted in these misfolded or excessive levels of protein or in some other way the reasons that these proteins are produced, maybe partly from genetic influences plus environment, what we might call epigenetic influences.

But at any rate, this is so important and I call this 21st century medicine with my patients both.

Why Biomarkers Matter in Parkinson's

The reason that we call this 21st century medicine is that we are now in the age of biomarkers or biological markers and alpha synuclein and is one biological marker.

If we go back and look at the diagnosis of or how Parkinson's and other related movement disorders are diagnosed, it's largely been clinical. Isn't that correct?

Dr. Levine Yeah, and in fact, that really still is the gold standard. We're trying to change that a little bit. But one of the reasons it's so important is and I think you alluded to this, is that you may get to Parkinson's disease through multiple different ways.

Some patients will have a genetic predisposition. Some patients could have a toxic exposure that causes the Parkinson's disease, like the camels dune stuff that we see on TV now all the time.

Some patients may have reasons that we can't understand. The end result all seems to be the same, which is that formation of the Lewy Body. But but each person could get there in a very, very different way.

And I think until we're able to put people into smaller and smaller buckets and say this is what the problem is, we'll never really going to get the true homerun breakthrough.

So I agree with you completely. It's unbelievably exciting to finally have drugs that will affect amyloid and can slow the progression of Alzheimer's disease.

But on the other hand, again, there are many other reasons to have dementias. And we know, for example, in dementia space, if you take the best clinicians, the best neurologist that you can find and the neurologist says, okay, that's Alzheimer's disease, and then the person passes away and you do an autopsy, you find out about 25% of the time it wasn't Alzheimer's disease, and then you find out about 15% of the time it's Alzheimer's disease plus something else.

And so you can imagine now, if you're trying to use one medicine, you know, the big hammer hit all these little

teeny nails. You're never going to do a good job.

We need much more targeted therapies. And to do that, we need the biomarkers and the analogy that I use. I think most people sort of get here is if we think about horrible diseases like Alzheimer's disease, Parkinson's disease, another disease that you would say you and I think are old enough, is is HIV right?

So when HIV came to the world, everybody died of HIV. But two things happened so that now nobody should die of HIV. People can live with HIV and live a completely normal life.

And those two things were a bunch of drugs that we could cycle people through and see what's actually working for them. And then the second was a very fast way of measuring the level of the virus, because, again, if people get to Parkinson's disease in multiple ways, not everyone may respond to exactly the same type of therapy.

So we now have 37, I think, different molecules that are in development to treat alpha synuclein in patients with Parkinson's disease. So imagine a world now you've got 20 of those drugs and you can rapidly put somebody on those drugs within, let's say, a month or two.

You could say, are you really affecting synuclein in the way that you want to? And if not, go to the next drug, go to the next drug. And that's what the HIV world did.

They just got a bunch of drugs. They cycled people through very quickly and they could measure the viral load. And as soon as the viral load was zero, they said, oh, that's the right combination for that person.

But everybody has different drugs, right? And so I kind of think of these as being the same thing, which is it's highly unlikely that we're going to find one drug that cures everybody with Parkinson's.

But if we had 20 drugs and we had a way of measuring quickly whether those drugs were effective, now we probably could stop Parkinson's disease. Yes. Yeah.

I'm going to steal a phrase from another podcaster I heard recently and she said, If you've seen one patient with Parkinson's disease, you've seen one patient with Parkinson's.

So it's perfect, you know. But that being said, you know, it's so interesting that we come full circle on these big, you know, chronic neurodegenerative disorders where we have the descriptions of the original cases of Alzheimer's or the original cases of Parkinson's.

And then we go through all of these years of relying solely on clinical observation, which still has huge value. And for folks who are listening, to be clear, as you know, it's been stated that it still remains sort of a gold standard.

You know, we we certainly and we will have a discussion that preclinical disease, that is disease before it's fully manifested as the rigid a kinetics shuffling gait may be UNILAG, lateral tremor, change in facial expression, etc..

That's when when when I see that in my office, I always say, you know, I make the diagnosis as I'm walking into the exam room. I don't even have to really, you know, go through a detailed history and examine the patient.

I do, of course, but it's a very visual diagnosis. And the response to leave a dopa therapy becomes sort of what clinches the diagnosis in general. But that being said, we've now moved from knowing about Lewy bodies and then finding out that Lewy bodies are primarily composed of this Alpha synuclein protein.

And now there is data that suggests that alpha synuclein is not just in the brain, it's in fact found in the digestive tract. And there are some people who think that Parkinson's, at least in some cases, may start in the gut and then migrate perhaps to the brain by way of the vagus nerve.

That's one theory. And it's found in the skin and it's found in the skin. I guess what you're saying, because our skin has nerve endings and this is really a systemic disease.

Correct? Yeah. So the gut connection is fascinating to me. We are doing some studies now and actually there was just an article published, I think in the last 30 days that shows that you can see these synuclein accumulations in the gut in people with Parkinson's disease.

And we also know, for example, that if you have if you're over the age of 50 and you have idiopathic constipation, that your risk of developing Parkinson's disease is four times the normal population.

So then another great example of that is if you take a mouse and you create a transgenic mouse that's going to develop Parkinson's disease, the first place that you see synuclein accumulate is not the brain of the mouse, it's the gut of the mouse.

And that's why the lot of this gut brain axis story in Parkinson's disease is probably not too far afield. So, yeah, so I think that that's really interesting.

And then we've known for a long time that Parkinson's is more of a systemic disease. So not just the gut. So we know that there's trouble regulating the blood pressure in the heart rate.

Gut, Skin, and Preclinical Disease

We know that bladder symptoms can occur and everyone has looked for a convenient, accessible biomarker to say this is the synuclein that's building up.

Therefore, we believe this person does have Parkinson's disease in the right setting. And then also as we work with pharma companies and our lab is now working in about ten different research trials, if you're that pharma company, you want to take someone who has synuclein and then you want to treat them with your drug and then you want to prove that there's less synuclein.

And one of the big pluses to that, again, to go back to the HIV analogy is if I want to see that I can change the clinical course of Parkinson's disease, I might have to treat a patient for a year or two because Parkinson's disease doesn't change in a month.

But if there was a test or I could rapidly see if I'm affecting that protein now again, I could run my clinical trials faster. I could make sure the patients are responding faster because we probably had some drugs that have been tested in Parkinson's disease that have failed for really two main reasons.

One is the clinical diagnosis of Parkinson's disease, as I referred to before, and Alzheimer's disease is often wrong. So Chuck Adler and Tom Beach are both here at the Mayo Clinic in Phenix.

They published ten years ago, but 230 patients that came to autopsy who had been diagnosed by expert neurologists and the expert neurologists were only right 68% of the time.

So I tell people all the time, if I tried to get through med school with a 68% average, I would not be talking to you right now. And that's that's the best clinicians that we have.

So we were certainly we're enrolling some people into Parkinson's disease trials that don't have the disease. So that's number one. And then number two, we may not have treated them long enough because Parkinson's disease doesn't change that rapidly.

So if you have a six month trial, maybe you don't see the effect, but maybe you would have in three years or five years. So that that test, you know, again, you know, think about another analogy I use is think about trying to treat glucose in a patient with diabetes.

The nice thing about managing diabetes is you can check the glucose conveniently and easily 20 times a day and the patient can adjust their medicine and adjust their food intake to try to compensate for that.

That allows us to very rapidly treat diabetes. But we don't have that in Parkinson's or Alzheimer's. And so we're hopeful that this the cinnamon test gives us the ability to visualize that protein.

And so we can tell hopefully, you know, over the course of a few months, is the protein going up or is the protein going down? So what we're saying, folks, is that these biological markers can not only facilitate the diagnosis, but they can actually help to determine if there has been a treatment response, which is profound, because we we have you know, there are scales, there are clinical scales to evaluate Parkinson's that are widely used in clinical research.

But this is one other measure that's very important and is being used. If we look at some of these other major diseases, Alzheimer's, multiple sclerosis, we do a lot of trials in both of those areas.

At Charlotte Health Neuroscience Research Center and we're doing MRI as we're doing PET scans, we're doing lumbar punctures. Here we have a test that is so simple really for any clinician.

You don't have to be a neurologist. I don't know if you guys are necessarily, you know, what you're and perhaps you could talk about that, what positions you are sort of accepting samples from that you're engaging in contracting with so that, you know, can any physician do this?

But I would say and I'd like to know that, but I would say in general, this is a very simple test, and I when I explain the test to my patients before we even have them in the in the room for the procedure, I say, look, you know, here's a pen and the actual piece of skin that we remove is about the size of the tip of this pen.

That's I said, you've had a cut in your life, way worse than this biopsy. Right. And we do three of them. We do three skin samples. And after the numbing experience, which most people know is a little bit uncomfortable in general, whether you're having, you know, a dental procedure or otherwise, really this is an entirely painful test that the actual, you know, harvesting of the skin probably takes 5 minutes, you know.

Yeah, Yeah. So really any clinician can do them. So we have about we're mainly talking to neurologists, obviously, because we're talking about dementia and Parkinson's disease, but we do have geriatricians that do it.

We have primary care doctors that do it and we have some neurologists, unlike you, who just say, I don't really want to learn a new technique, and then we could even send it to a dermatologist or anybody.

So it is very simple. We say it takes about 15 minutes. That's on the long end to do all three. And your and your comparison is exactly right. I tell patients when I put it in the light, a cane, it's going to burn like crazy for 5 seconds.

And all they have to do is breathe through it for 5 seconds and then they're pretty much numb for the rest of the procedure and they don't feel anything.

It doesn't really hurt. The next day it heals up like a little scab. There's no stitches and it can be done anywhere in the country. So it's shipped to our ship, to our lab in Arizona.

We've also touched on some of the preclinical features of Parkinson's disease. And it's important to to remind folks that when we're looking at alpha synuclein, alpha synuclein can be seen in diseases that are in the same family as Parkinson's, multiple system atrophy, prognostic failure, Lewy body dementia.

And I'd really like you to talk about some of the recent presentations that you've been making. Your company has made public that your research and even distinguish it using this test to actually distinguish between these different hyperkinetic movement disorders.

But that being said, you've touched on REM sleep behavioral disorder. We've talked about constipation often for decades potentially, although that's not to say that everybody who's constipated should have a skin punch biopsy, you know, and even mood or personality changes that may precede the actual movement disorder.

So I probably the take home message to borrow from a colleague of mine who said that everybody who's turned age 65 and goes to their doctor for their preventative exam should have a cognitive ask AP.

Doctors should be sort of going through that what we call that review of systems and see, you know, how is your sleep, how's your digestion rate, how's your mood been?

And if we start to see a pattern, the possibility of then thinking about preclinical Parkinson's becomes very valid. Yeah. And especially we, you know, once we get into the and fingers crossed because I believe it's coming soon.

The age of disease modifying therapies for Parkinson's, can we prevent this? Data released by Eli Lilly and company on their new Alzheimer's drug and Mab showed that individuals who had just mild cognitive impairment but they had the pathology of Alzheimer's, meaning if we image the brain because they primarily used PET scans to look at amyloid into our proteins, but those individuals are highly functional individuals, we wouldn't really look at them and say that they had dementia when they received inanimate, they had a 60% slowing in the expected progression of disease.

Using Biomarkers for Early Detection and Trials

That is amazing. So early identification and intervention, I think is going to be the mantra across the board, including Parkinson's disease. Yeah, yeah, yeah.

So that's this what you just mentioned is probably the thing that I get the most excited about when I think about our test. And the reason is, and I think you probably would agree with me if you wait until somebody has dementia for 20 years and then you think we're going to have a drug that's going to reverse that, that is, I think, highly unlikely to happen any time soon.

But what the biomarkers allow us to do is to detect people before they've lost a lot of neurons. So if we know that you're at risk for developing Parkinson's disease, what can we do then to slow that progression and prevent these diseases from ever occurring?

And I think that's that's really where I get excited. So we talk about sort of two levels of early detection, if you will. So one is what you were sort of alluding to, which we will call prodromal.

So the prodromal symptoms in Parkinson's disease could be REM behavior disorder, could be constipation, could be dizziness, could be depression, could even be bladder symptoms.

So those types of symptoms could prompt us to say, should we look and see if synuclein is starting to build up Now, that then leads to what I call when I talk to neurologists the so what?

Question Okay, so if I found out today that I was building up synuclein, what do I do with that information? We don't have an anti synuclein drug yet the way that we have an anti amyloid drug, but we also know that diet, exercise, sleep all of these things actually slow the progression of Parkinson's disease.

And so I talk about that as wellness. So just like if I found out today that I was pre-diabetic, what would I do? What I sit around and do nothing and let myself become diabetic or hopefully I would exercise and diet and really focus on wellness with the hope that I can prevent ever becoming diabetic. Right.

We're really in an era now, at least with these diseases where we can detect these people in that prodromal stage and at least in the short run, focus on that wellness, those things that do have I think there was just a paper that said 75 minutes a week of exercise slows the rate of progression of Parkinson's disease.

That's not a 10 minutes a day. That's not a lot. And so if you if that were you or your brother, would you really be able to say, okay, now we have to do that?

So that's the prodromal stage and that's what I'm really excited about. And then again, once we get an anti

synuclein drug from one of these 37 molecules that farmers companies are developing now, the So what questions answered?

Well if you're developing synuclein and there's an anti synuclein drug, don't wait until you have Parkinson's. Take it five years before your Parkinson's and hope you never get Parkinson's.

And then there's a further stage which we call Presymptomatic. And this is more complicated. So this would say the analogy here would be, for example, we know there are certain genes that are related to developing Alzheimer's.

So let's say me at 56, think I'm still an okay neurologist, still thinking, okay, but I get my genetic test and I turn out I have two APOE4 genes, okay, I don't have any symptoms, but I now know I'm in much higher risk of developing Alzheimer's disease somewhere down the road.

Now you have the same. So what question What do you do with that? And some people don't want to know that, but if you just go out and biopsy, let's say a thousand people that had no symptoms of Parkinson's disease, we believe somewhere between two and 5% would already be accumulating alpha synuclein.

So one of my dreams and actually it's a study that we're doing right now, most people in the US at 50 go get a colonoscopy, right? Because if you get your colonoscopy every ten years and they remove the polyps, then you never get colon cancer to completely change that disease.

So if they're in, they're already doing a colonoscopy and biopsies all the lesions that they biopsy, what have you could biopsy the colon in every human being at 50 and see who is accumulating synuclein, who's accumulating amyloid, who's accumulating tal.

And now you have sort of a neurodegenerative screening test and then again with the hope that there are drugs so that, as we say, you're developing TDP 43.

So we're going to put you on an anti TDP 43 drugs so that you don't get frontotemporal dementia. That's where I would get now that's that's the Holy Grail and you probably never actually get to hold the Holy Grail, but you can imagine a world where that actually happens, say, in the next 10 to 20 years.

And now we know that the incidence of neurodegenerative diseases by half. Well, I mean, now you can take a little swab out of the inside of your mouth, send it in and get a digital record of your entire genome.

So perhaps it's really not that far fetched. And there will be these screening panels for these major diseases that we have early interventions for. Yes, I'm not not farfetched at all.

Now, the same one person, but part pardon me, sir. Not too Star Trek. No. Right. But the skin, the skin punch biopsy is not the only test out there that we might use in a setting where we do need to distinguish Parkinson's from other disorders like essential tremor or drug induced Parkinson's.

And now we know that that with your particular test, that we can even distinguish the different subtypes of movement disorders that fall in the Parkinsonism family from each other, the degenerative Parkinsonism family from each other.

We've had the that scan, for example, which I've used for a number of years, but I must admit a lot less common for me to order, and that's SPECT scan these days because I can do a scan punch biopsy right in my office.

And they are different tests. They're really not testing the same thing. Right? Yeah. So they're complementary in a lot of ways. So if you think about the Syn one test, the Syn one test is looking for the accumulation of the abnormal protein.

Our data that we just presented at the American Academy of Neurology shows that we have about a 95% sensitivity and a 96% specificity. So very specific and sensitive test.

But there are many other diseases that cause those same parts of the brain to deteriorate over time. And not all of them are associated with synuclein deposition.

So two of the more common are associated with the accumulation of a protein called Tao, which we also see in diseases like Alzheimer's disease. So if you do a dad scan, the dad scan gives you a broader look.

And it really says, is there evidence that these nerves involved in movement are dying? And then the Syn one test says, is there evidence that the synuclein protein is accumulating abnormal in this person?

So you can imagine if you have an abnormal dead skin, but a normal syn one test, then what that tells you is, yes, those movement disorders, structures in the brain are dying, but it is not due to synuclein and therefore it's very likely it's due to Towle.

Now, again, that's one of the reasons we'd love to have that Tao test, which we're working on, because then we could specifically answer all but so you can end up, as you said, with Parkinsonian diseases, diseases that look like Parkinson's disease, but are not related to synuclein.

And the reason, again, that that's so important these days is because we are developing these targeted therapies. So you can take a symptomatic medication like cinnamon, like levodopa, and give it to anybody whose basal ganglia is deteriorating and hope that you see some response.

Clinical Access, Test Performance, and Future Directions

But when you start to think about targeted therapies targeting synuclein and targeting how targeting amyloid, you've got to start with, well, I'm not going to give an anti synuclein drug to a patient that as a Tao disease, that doesn't make sense.

But the wrong antibiotic. Right? Exactly. Yeah, exactly. So the two really do work together nicely to provide a lot of information even in advance of just what the one test tells us.

Excellent. As we start to wrap up our interview, I want to bring up the thin one clinician network that may be very helpful to folks who are affected by these movement disorders because they want to find a provider or who is able to perform this test.

Yeah, So we we have on our Website a network of doctors across the country that have agreed to let us put their information up because they are comfortable with this in one test, they're comfortable doing the biopsies.

And so that's one way to kind of reach out. We have a very good patient services team at the lab. And so if if there's not somebody locally, they're available for you.

I would call here. We probably know somebody close, even if they're not in the network right now. And then we've done a lot of work. Even again, if you're if you would talk to your primary care physician who would say, oh, that that makes a lot of sense to look for this, but I don't really want to do it.

You know, I don't want to think about bringing something new into my practice right now. We've done work even with in-home nurse practitioner groups, which is a great way to get someone to come to your house.

They could do the biopsy in your living room. It's incredibly, incredibly simple. And then they shift the biopsy to us, just like your doctor would, and then your doctor can get the results.

So we have a lot of ways to help patients get access to this, but we have right now over a thousand. So there's about 15,000 neurologists in the country right now.

About a thousand of those have used our lab over the past four years. So we are growing. So we should have pretty good coverage across most of the country.

Talked a little bit about your research, and I know you've been a presenter at some major meetings recently presenting on the sensitivity and specificity of your tests.

Are there some major upcoming events we can look for for CND Life Sciences? So most of the major meetings for this year are over, except for the Movement Disorders Society meeting, which is going to be in Copenhagen in August.

So we're going to be presenting our data there as well. We're working with a lot of investigators across the country on what we call investigator initiated trials.

So now that this test is available, I'll give you just one pitch for what I think, again, how smart people are. There's an amazing movement disorder. Neurologist at the University of Michigan and he believes, as many people do, that Parkinson's may really be sort of a spectrum.

Right. And so if you think about, you know, what you mentioned earlier, which is as a neurologist, we could often be across the parking lot and see a person walking and we'll say, oh, that's Parkinson's disease, right?

But then you see older people and this is my dad is 92 that are kind of hunched over, that they walk really slow and they're not Parkinsonian, but they're just sort of developing one of these gait disorders of the elderly and know how many people in 90 walk the way they did when they were 50.

So Dr. Bonin in Michigan believes that that may be actually a different form of Parkinsonism related to synuclein. So we're bypassing a large number of folks, kind of those over 80 year olds that have developed kind of slow gait, hunched gait, but not the clear features of Parkinson's disease.

We've also uncovered we say there's five synuclein apathy, which we labeled already, just this year there was a publication of a disease called Lovebug, which is actually a disease that includes Parkinsonism, but many other features like dystonia and other movement and dementia, and it's confined to the Filipino population.

So it turns out that's a synuclein apathy. So in five cases, all five had synuclein. And even though it's not really Parkinson's disease. So I think as we start to measure this protein, I think we're going to find that, yes, PD is definitely the biggest and dementia with Lewy bodies and multiple system atrophy, but there may be many other diseases where the accumulation of this protein is just never really been thought about.

And so that would be fascinating. I think it's so important. Dr. Todd Levine, to what do you share the website for your company so that people can look that up and read more?

Yeah. So the company is called CND, so Charlie, Nancy, David, Life Sciences and the website is just cndlifesciences.com I think the website is pretty good.

It can give you sort of a good background. As I said, I'm, you know what, what patients think about when they think about the past talks a little bit about insurance coverage.

And again one of the nice things there is that this test has been very, very well paid for by Medicare. And most of the patients that we're talking about because of these diseases are of Medicare age.

Commercial insurance is always a little more hit and miss just because there's so many rules, out-of-pocket deductibles and so forth. But we can work with patients on that as well.

And all that information is on the website. I would say on a personal note, we've had very, very little trouble getting coverage for our patients and the turnaround time also important to a lot of folks.

How quickly will I get my results is very good. So we usually schedule their follow up visit 3 to 4 weeks after the biopsy is complete. They have their answers and we move forward with a diagnostic or therapeutic rather plan.

So it's excellent, etc. And the contact information for your company is right on the website as well. I might add that there is a fascinating that a one minute video or so, if you want to see what outfits Nucleated looks like in sort of almost three dimensions on the video, it's nicely done.

So I would encourage folks to get a visual by going to the website as well. Great. Well, thank you so much for spending time with us today on the Parkinson's Solutions Summit.

I know that viewers have learned a tremendous amount from you. And again, please keep up the excellent work. I know we're very committed to your company and the type of work that you're doing. It's absolutely changed my neurology practice when it comes to movement disorders.

And I'm excited about how all the puzzle pieces are coming together. We're just a hair behind the Alzheimer's folks, but there's even a lot of work still to be done in that area, of course, as well.

But I see us converging on disease, modifying therapy, hopefully in the next few years as as the research and clinical trials evolve. And this will be central to treatment, diagnosis and treatment of patients.

Well, appreciate it. Thanks for your support. Thank you. Have a wonderful day. Thanks. Thanks. Bye bye.