Introduction to the Parkinson's Solutions Summit

Welcome again to The Parkinson's Solutions Summit. I'm your host, Dr. Ken Sharlin, and today's guest is going to really expand your thinking, your universe when it comes to understanding complex, chronic neurological diseases.

We may get into other conditions. We're going to, of course, focus on Parkinson's disease. This is really hot off the press and formation, and I can't tell you how excited I am to interview Dr. Howard.

Urnovitz. And he's actually in, Germany right now. So we are welcoming him from across, the Atlantic Ocean. But he's going to talk with us in depth about the role that endogenous viruses play in these diseases that we all see, folks suffering from, including Parkinson's.

If you are affected by Parkinson's yourself. So he has many accolades. We'll talk about some of his background, his research, the companies he's developed and what's coming in the very near future for precision medicine in Parkinson's.

Doctor, thank you so much for joining us today. I really appreciate you. I'm looking forward so much to everything you're going to share with us today.

It's an honor. Thank you. As we kind of I like to set the stage for things. This is very scientific. You and I have chatted for a few minutes before hitting the record button, and I want to make sure that the folks listening or watching, you know, understand.

And, you know, this is sort of systems biology at the deepest depths is that it's really about how all of the various factors that we talk about mitochondria, the immune system, the microbiome, our detoxification systems and so forth all play a role not only in regulating homeostasis, but when homeostasis is disturbed, it may lead the person down the road to disease.

But it seems like what you have discovered in the focus of you are really.

Endogenous Viruses and the Origins of Disease

It sounds like a good part of your career has been the key role that viruses play in all of this, in disrupting that homeostasis and and driving us toward, in this case, Parkinson's?

That's correct. And so, thank you again for letting me speak. Here. We are, really excited about the fact that, we were able to, I've been working on this for half a century.

When my mom died of cancer, I realized I'm going to have to get in the game and figure it out. So age 18 years old, I got into what have we discovered at the University of Michigan.

My PhD advisor had discovered one of the most amazing things in the world. 1979 Nature paper. He discovered that he could create polio without the polio virus.

He used a harmless virus and mapped it down to using mice, mapped it down to one nucleotide, the the the letters that form DNA. And he could show he could create.

And it turns out it was an endogenous virus mutation that started us thinking. And in 96, we wrote the landmark review of human endogenous retroviruses in clinical medicine.

And one of our favorite papers. But I'll tell you, we've been working on this for a long time. There's so much data

out there. How do you even start? Well, that's when five years ago, somebody said, what do you know about artificial intelligence?

I looked at my Wikipedia and became a world expert right then and there. So we have incorporated artificial intelligence into our way of counting. And now we can count every ancestral gene in your body when they act up.

That's when disease starts at birth. They basically make you who you are a human, an individual. In fact, not even the identical twins are identical after they split, because then you have nuances on the ancestral genes that come up.

This is really something that is so important that now that we can measure it, we now understand that every disease starts by a panel of ancestral genes, which we can measure in the blood.

So, the use cases are early detection of Parkinson's. We're seeing that now in people that are recovering from Covid or long Covid is a group of people heading towards their.

We saw a 1918 Spanish influenza, a surge of pain, of Parkinson's disease come out of this. So what we have done is said that virology is not what we thought it was.

It's not following postulates that the one virus causes the disease. They are what we call inter active infections. And so when you get a virus like Covid come in if you're ancestral genes are activated, which would be most people in nursing homes and, and the like who are ill and frail.

That's why it went through them so fast. It was just basically we're ready for the virus because we have all these ancestral genes turned on. We don't want that to happen.

The for our, for where we come from. And I like your idea that a lot of neurologists like to treat early and treat heart. If we can give, the doctors the information that your patient is early stage, stage zero, no symptoms.

And then when you treat hard and treat early, we come back and say, did you quiet down the ancient genes? Because if you did, you cured the individual.

That's our hypothesis. And we're ready now to enter clinical trials to see if we are right or not. And if we're right, then we'll be very excited to get this thing into the hands of every doctor around the world.

That is fantastic. Now, Dr. Urnovitz there's a kind of an analogy. I use it not a lot of analogies as as a clinician, as a doctor, I always think that, you know, a big, role that we play in the clinic is to teach, to educate our patients, and we have to bring that information in in a way that they will understand and ultimately embrace in a heartfelt way, because that's, of course, on the on the functional and integrative side of things.

That's when change tends to occur. But that being said, one of the analogies I've always used is the old, story for children about Fort. And here's a who, you know, because if we look in the mirror, we see this, you know, a head and a torso and arms, legs beaten heads don't.

That's us. Right. But in fact, we are much more than this sort of big picture that you see in the mirror. Now, when I thought about that analogy, I thought of that analogy.

I was really thinking of, say, the microbiome, those microbes that live in our gut on our skin.

Early Detection, Interactive Infections, and Clinical Trials

We now know that there is a kind of a brain microbiome. But if I understand correctly, what you're saying is it's not just the sort of microbes in total, it's fragments of these microbes that have then integrated into our genome over hundreds of thousands of years and are passed along.

But we're still in that, again, if I understand correctly, we're still in that mode of it's not just your genes, right? It's that genes plus environment.

It's that, you know, there's an expression that genes load the gun, but the environment pulls the trigger because then you have to have some environmental factor.

And in your case, you're talking about the role that Covid 19 plays. And we certainly saw this epidemiologically that when the epidemic hit, it was very clear that we were going to see a major rise in Parkinson's and Alzheimer's and ALS.

So we acquire this virus. But then it's how these viruses interact with our own genome, with that ancestral genome that then drives these diseases. It's that is correct.

That's a great analogy. There's you're going to we're going to focus on Parkinson's, but there's been there's a couple of areas that are, you know, have been are particularly, well studied.

And, and, and published where we're a discussion of the role of virus, is a major trigger for disease. We have Epstein-Barr virus and multiple sclerosis, and we have herpes simplex virus in, Alzheimer's disease.

Ruth, it's a if it's akis, we're at Oxford. I always use, paper that came out of Taiwan. It was just, epidemiological, retrospective study, but it looked at something like 32,000 subjects, of which about 8000 were infected with herpes.

And the, hazard ratio of developing Alzheimer's was about 2.6. And then when they looked at individuals who had been treated with Valtrex or a cycle of beer drugs that are anti, herpes virus drugs, if you will, the risk of developing Alzheimer's went down dramatically, that there is an ongoing prospective trial, that's registered under clinical trials.gov to further evaluate this.

So we don't know for sure yet, but I do personally treat my patients testing positive for HSV and the earliest stages of cognitive impairment, due to Alzheimer's or who think they may be at risk.

Maybe they're ApoE4 positive things like that. I then treat them with at least 30 days of Valtrex, but I wonder if we can use this as a launching point and then talk about how these same types of viruses play a role in Parkinson's.

And yes, your thoughts on how we can use interventions. And ultimately what we're looking for, of course, is disease modifying therapy for Parkinson's.

And we don't have that yet. Yep. No. That's great. When I was asked to review the science paper on the study, I think it was, 8000 is huge number of ami, recipients.

And then concluded that Epstein-Barr virus was the agent. I pointed out that the testing. I'm not sure what you're testing is, but usually the clinical test is for the virus antibody, not the virus.

And so what they showed was there was a very good correlation of the Epstein-Barr virus antibody, not the virus. What I'm proposing is that we know now that Epstein-Barr virus is part of our genome, maybe 90%, but not the whole thing.

We know that people in Africa are almost devoid of human herpes virus six, another herpes virus. But everybody northern European has it because they had to adapt to the environment.

And I'll give you a, point for how many years it was, it was 150,000 years ago when the first male walked out of the cave in South Africa. And then as they moved north, they collected viruses to help adapt with their environment.

So this is a case where it's good where you get all these evolutionary things. So in the case then of Parkinson's, we've identified what viruses would turn on and turned off as far as the RNA system.

But we haven't really found a classical virus, if you will. So when, the the gut microbiome is indisputable. But when we talk about a brain microbiome, those papers need to be reinterpreted, that they may have actually been genomic mother and father derived and that have got you there.

But when something reactivated, radiation or chemical exposure or something that just went bad is if you reactivate an endogenous virus. Yeah. I don't I don't think I would call it the microbiome of the brain.

I would call it ancestral genes. So if that's true, then you obviously are showing, clinical results by putting people on anti herpes drugs.

Parkinson's Biomarkers, Personalized Prevention, and Variable Regions

Then I would ask the question, are you in fact inhibiting these endogenous viruses. And we should start from a positive. We need to dissect why your, your system is working.

Your protocol is working. Because if it works I want to know why at the molecular level. So that's why these breakthroughs now in concepts of how we how we look at viruses, a concepts of evolution.

But most importantly is how do we cure the patient of Parkinson's. And I think the only way to do it is with the scientific method. And we start with a hypothesis.

You got good data. We'll be happy to work with you on why is it working, and then how do we make it work on 100% of patients? That's our mission. So some of these diseases Parkinson's, certainly Alzheimer's.

We now recognize they have a long what I'd call sort of incubation period or pre morbid, in Parkinson's, there's loss of smell very often. There is REM sleep, behavioral disorder, mood or personality changes long many, many year history of constipation.

And those should be, you know, warning signs to clinicians. But too often we're not asking the patients to rate the stress genes in the primary care clinic.

So clearly something is happening before the tremor appears. And, so my two part question is one, what is the what are the viruses of interest or what is the ancestral genetic, you know, viral fragments of interest when it comes to Parkinson's?

And then what are your thoughts about, you know, where do we do this? Almost sounds like childhood vaccines to me. I mean, it's and I, you know, in other words, what I'm saying is we have the opportunity to prevent before anything happens by identifying who's at risk and what we can do about it.

Yeah. Well, you know, let me start with, in my, golden Age of Virology on our YouTube channel, it's FB, which is Frank brother brother bio on YouTube.

Our golden age of virology is one of the things we want to do is personalized prevention. Because it's just too hard to administer herd immunity. And, so why don't we then say, if that's the reality, then why can we personalize, the prevention so I can find in who is going to be susceptible to an interactive infection.

Those are the people we need to vaccinate and, and because they really need that protection, do we need to vaccinate everybody else? The clinical trials will tell us.

But I do believe that we can combat flu or any coronaviruses at all by identifying what percentage of people are susceptible now, which viruses cause Parkinson's.

We haven't found that virus yet because we measure all of the ancestral genes, transfer our RNase, microRNAs, everything but messenger RNA because up until now it's been called junk.

So our contribution to science is we have we measured the junk and it turns out that a set of RNA, ancestral genes define Parkinson's, a set and it's not a virus.

It's just all of this, myriad of all of the functions the wonderful world of RNA is we measure them. And when they work in concert, they define a disease.

So for us, when we got these two Covid patients in, they have no symptoms at all, but they're showing half, roughly half of the ancestral genes being activated that we see in Parkinson's patients.

We're seeing the earliest event. And why? Because RNA is the cause of these disease. They can be reverse transcribed in you can make multiple copies where they land could be deleterious.

So we knew that 50 years ago because we had plenty of experiments showing that the ancestral genes, when activated, would cause neurologic disease. What we're showing.

So that's why differentiates our approach is we're trying to measure the cause of these diseases and, and work in concert with many the tau proteins and everything else the the clinical observation, loss of smell.

You should have been able to detect that with our test a year earlier. And so because they slowly reproduce, they reproduce themselves. They then package themselves and go out to other cells and pseudo infect them so that now you got a problem in another cell.

So that's this chronic progression of disease that can all be detected in the blood, because we found a way to measure the activation of ancestral genes.

And the ones that get activated are the ones that will tell you which disease you're starting. And it sounds like it would be potentially different for different people, even with broadly the same disease we know.

We do know there's that expression. If you've seen one person with Parkinson's, you've seen one person with Parkinson's, and that applies to all of, you know, the names and.

Yeah, and July. Well, let me tell you why. So I'm sitting here in Germany, I'm going to look out the window and there's Paul Ehrlich laboratory. 100 years ago, Paul and Erlich discovered the variable region of antibodies, which is using rabbits in bacteria.

I am proposing in one of my videos, I proposed, you know, 20% of the genome has never been mapped. And do you know why? Because I propose it's the variable region.

We know there are five super human groups North China, South China, Africa, North European and South America. Everybody has their own variable regions.

We have the common genes that make us Homo sapiens, but then we have the genes to adapt to our environment. Those are the variable regions. So like you said, we can find common Parkinson's disease genes all across the spectrum.

But then we can see individual ethnicity associated with these because they have a different set of ancestral genes. Some are more sensitive to be activated, some are less.

And so when we say we're going to go down to the personal level, it means that we're going to respect how evolution brought you here, and we're glad that it did.

And then we're going to treat you according to all of the all the genes, not just your favorite one. That's what artificial intelligence and machine learning did for us.

It discovered that the variable region genes means no two people may not be treated the same. There'll be a

general, treatment in protocol, but then you'll have to come in with just knocking out those, variable region genes with all of these new RNA silencing pharmaceuticals.

They just need somebody to tell them where to apply it. And that's where I see the the eye. That's where I see this going. And we're terribly excited about it.

You know, sometimes they talk about cigaret smoking. And of course I always start with I don't I'm not recommending cigaret smoking to anybody. It's terrible, terrible deadly habit.

But you know, we we know that cigaret smoking sometimes leads to lung cancer, sometimes leads to heart attack. And stroke sometimes leads to COPD. It's associated with renal cell carcinoma.

And then there's the person who's 105 years old, and you ask him what was his secret to living that long. And he says, you know, a shot of whiskey and a cigaret every day.

And so we have this common environmental exposure, and yet many manifest many consequences because of the bio biological or biochemical uniqueness, you know, of everyone.

And to be clear, what we're saying, I think, is that, these viruses have helped us get us where we are. This is not all a bad thing. And we need to understand that very, very deeply that we really are, we and I, you know, you heard that 150,000 years when we since we have left the cave.

Evolution, RNA Operating Systems, and Homeostasis

But I think you're I'm wondering, you know, how much you embrace the concepts of Darwinian evolution in the sense that, you know, these viruses probably played a role in, in the evolution of, of and the alien species and all the way down to single celled organisms, you know, and not just human evolution and and so what I think, at least it strikes me, is when I think about, as a neurologist, some of the big diseases that I, that I have to, you know, help people with.

There's interesting things like why is it, for example, that primary progressive, non fluent aphasia targets the language system of the brain? Why is it that ALS targets the motor system?

Why is it that Parkinson's targets the programs for the motor system, among other things? So it's not just the cortical spinal tract they als, but it's how we eloquently combine, you know, muscle movements into, you know, I think the, the conductor, conducting the orchestra.

Right. It's those fluid movements that that have to play out as software. They do all in the, you know, Alzheimer's memory decline, the hippocampal, deterioration of Alzheimer's.

They do eventually all have, you know, come in, you know, everyone ends up sort of rigid in with pneumonia and sepsis in the end. But, these diseases start out different ways.

So it kind of tells me, too, that almost from a teleological perspective, that maybe these viruses, the positive aspects of these viruses that have supported evolution are things that gave us language, are things that gave us movement, are things that gave us memory, are things that, you know, all the things that when we when we see the disease manifestation, there's there's the other side of the coin as well.

Yeah. Well said. I'm smiling because I'm going to tell you what I told the audience when I stood on the stage at the Royal Society of Medicine, where Charles Darwin used to give his lectures and say, and it was on the evolution of cancer.

But the fact of the matter is, you've hit it right on the head. This goes down to a single cell organism. What we discovered is, for example, what is what's, an operating system.

We always have to update them. It's two things. It's code that controls files. That's the definition of an operating system. We have discovered the operating system of life, its code DNA and its files. RNA.

But the RNA can also be the code and the files. That's what makes us so unique. So when the first single cell organism started it, it was had to bring in more information to get us all the way here.

And so all animals have this RNA operating system. I've worked for a long time in veterinary institutes on mad cow disease as a way for neurologic disease. CJD.

And so what we discovered was it's the RNA that folds the prions, not the prions, folding the prions. And so when when these things are made to develop, you want to fold proteins.

So you turn something into a liver cell and maybe something into, a reproductive cell. But the fact is, our simple, logic is don't anger the ancients, because when you call on them, they will respond and so this is really the basis of all of it is we have this, you know, I'm shivering right now, having stood in Watson and Crick slab, and there was only 70 years ago that they discovered the structure of DNA.

If you think we're taking too long, step back and look at. We're moving at amazing speed. So from the structure of DNA, I have to throw in Franklin and Wilkins.

To today, we've gone from what? DNA is a double helix till now, discovering the operating system of life. And so when it started, I also have a YouTube of the Berlin Symphony playing in tune.

And as a way of explaining to the public what we're doing when we see the cello player is a little bit off. We, you know, tap on the conductor and say, you may want to get that one back in tune again.

Yes, it's a symphony of life run by a biological operating system that's so exciting to us. And, you know, that's it's funny, I use the same a very similar analogy with this, with the symphony orchestra and to talk about homeostasis, ultimately, that's what this really is.

It's the balance of all those different parts the cellos, the violins, the horns, the percussion, you know, everything that makes a symphony. And when one is too loud, it's going to either have to soften up or everybody is going to have to play louder.

But where you haven't, you know, until now, incorporated this idea that ultimately we have all of these environmental inputs and folks, this is still sleep, this is still nutrition, this is still, you know, movement as medicine.

But the idea is when we do all of these things, we have to understand all the systems that are being affected by these therapeutic lifestyle factors. And those systems incorporate our our ancestral genes that make us uniquely vulnerable.

Like the ten different scenarios of cigaret smoking that I, I mentioned earlier. Yes, correct. That's absolutely correct. Well, I want to bring back into our discussion.

I know we're probably going to run out of time soon, but I want to bring back in the discussion something that we talked about before the recording, because, ultimately folks are going to have I mean, this is, first of all, this is, you know, just mind blowingly fascinating.

But, folks are thinking about solutions for their Parkinson's. And we still want to emphasize that if you want to respect your ancestral genes, you have to follow those therapeutic lifestyle factors.

But we were talking about multiple sclerosis before the recording button was pressed because, there is a drug that's used in and that's been used actually a very long time, to use in cancer therapy as well.

It's called Collider, been historically, it's been given intravenously in a variety of doses. And, and it ended in the

setting of cancer therapies. It'll wipe out your bone marrow at all. Yeah.

Basically kill the white blood cells, by disrupting your DNA, but in, oral form and low dose oral form in multiple sclerosis. It's been proven to, deplete enough of your overactive white blood cells that theoretically, the idea and this is I don't it's not just theoretical because the drug is approved under the name, Marvin Clyde, but it then that sort of reboots the immune system.

You make new immune cells. We refer to these as naive cells. And the idea with NAD in Clyde is, you know, you basically take it's a, it's a one big treatment and may be repeated the following year and potentially in the United States, for a second year.

But the point here is that it's not an ongoing treatment.

Multiple Sclerosis, Epstein-Barr, and Precision Treatment

It's an opportunity potentially to sort of reboot the immune system in multiple sclerosis and then just not have that mess anymore, put you in a remission or achieve a cure however you want to define it.

But the caveat here is this idea of Epstein-Barr virus that is an ancestral gene that now, you know, is related to an ancestral genome, and that we know Epstein-Barr virus plays a major role in multiple sclerosis.

And so the question then is that I posed to you before the recording is perhaps what we really should be doing with folks with M. S. is new diagnosis. Treat them all with oral clouds of being or maybe even I. V.

under certain circumstances. But then combine that with something that specifically targets Epstein-Barr virus. Maybe it's a drug like Valtrex, or maybe it's a specialized stem or RNA vaccine that Moderna is developing.

But could we really you know, we already know that oral being works for mass, doesn't achieve a cure. About 80% of the people who take the drug never have another relapse.

Right. So could we bridge that 80 up to 100% by then going after these viral DNA particles? We have to and, as I said earlier, before our recording, I was asked to review that, marvelous paper that showed the correlation of Epstein-Barr with Ms..

But I pointed out one simple thing you were measuring the antibodies. And so what we have to do is these have seen bar viruses. They're not total viruses. They're fragments of viruses.

When they make protein you see it as an antibody response in the test. What I said we published this in acronyms last year with Doctor Walls, that we could follow three patients over three years and showed that one one patient got I. V.

steroid, that two weeks later, completely turned their operating system upside down and came back again. And so what I'm saying is we've got a tool to ask the question, can we predict in advance whether somebody needs I. V.

or, or not? And that's what we need to do in advance so that you're bringing it up 100%. Does this person need anti-HIV drugs? Does this person also need to do this and and then ask the lifestyle questions?

Do you live near a toxic waste dump that you're constantly, always turning your ancestral genes on? This approach will be what has always been the hope of personalized medicine is to go in and say, we were all created equal, but you're falling apart uniquely, and here's how and here's what we're going to do about it.

We need to build our tool to be in the hands of clinicians like you. Because when I give you this new data, the new perspective that these are viruses, they're in fact endogenous ancestral genes, that's a whole new way of thinking about it.

New targets for pharma, a new target for, lifestyle changes, all of these different things. And so if we do that in advance, we will prove your theory and then bring 80 up to 100, because we have to.

We have to, and again, and I apologize because, today, you know, sitting down and preparing for our interview is really the first time that I've really, gotten into this particular topic, in this depth.

Some ask questions. I hope all makes sense. But this makes me wonder about something else, which is sort of the mirror image of everything you're talking about.

We recognize that there are really a few prominent factors that seem to lead to the development of Parkinson's disease. The Parkinson's, sort of from a more public health epidemiological perspective, seems to be a phenomenon of the industrial age.

And I, I'm presenting it a couple of weeks at a big meeting. And certainly we've recognized cases of Parkinson's going back several hundred years. But if you look at the, you know, the rising, the numbers of new cases of Parkinson's, it really goes up as we've industrialized our society.

Now we all live right. You know, however many billion people now on this planet, 8 billion or whatever. And yet, you know, a little over a million have Parkinson's right now.

So obviously in, you know, in the US. So obviously not everybody gets Parkinson's. But I'm wondering if is it possible, does this even make sense that it's not?

And since we don't yet have that genome for Parkinson's, could it be possibly. And I'm going to kind of really put it out there that it doesn't exist, meaning that the problem isn't that certain people have this ancestral genome that create a susceptibility to Parkinson's, is that many people have an ancestral genome that protects them from Parkinson's and that, you know, what we need to do is sort of infect people with the right viral DNA so that they can live in this toxic world and not get sick.

Well, let me tell you something. If you can't guess that one of the things I've said are quite disruptive, and I have a way of walking into a room of scientists and hearing, oh.

When I built the blood test for mad cow disease, which picked it up 15 months earlier, and we, confirmed it with the Canadian and German governments. They were not in a position to say that I know what caused that mad cow epidemic.

It was industrialization. It was Chernobyl. Chernobyl happened in 86 at the end of the year, when it rained for a week on, from the Irish Sea. On on England, 20,000 cows die.

So it's my position that Chernobyl caused mad cow. Why was there an epidemic of surge of Parkinson's after the Spanish flu? My hypothesis is, that you had hundreds of kilotons of mustard gas and chlorine gas in the air from something called World War one.

When they started turning on those canisters. And, in fact, that guy went back to Kansas. Everything else you had just, submitted to the world the most toxic gases in the world.

I can there's books on military campaigns and infectious diseases. Going back to Attila the Hun. You hit it. Parkinson's. We see it. More polio now. That one really got me.

Polio was always around. I mean, FDR started this, place in, Georgia for other polio victims, and then it went through the roof and it was Rachel Carson who identified it.

It was DDT, the most deadly neurotoxin to kill mosquitoes.

Industrialization, Toxic Exposures, and Mitochondrial Genes

But when you gave it to people that had ancestral genes, like my adviser's model, you turned on the epidemic of polio. And it wasn't until JFK banned the use of polio.

Excuse me, DDT. We saw it go down to its its regular levels before then. Parkinson's is an industrialization, a result of industrialization. That's my feeling.

Do since mitochondria have their own genes. I'm just curious how this plays a role because we we know that mitochondria are really at the center of the Parkinson's story as well.

You bet. Oh, well, we measure the mitochondrial, genome as well, because it also it also has the ancestral genes in it. In fact, the whole mitochondria, his ancestral because it was a purple bacteria that climbed into an amoeba is that, there are cells.

Is that, we never came up with the mitochondria. It was there to help us crawl out of the soup and onto land. So we walk around with the symbiont relationship.

And the mitochondria also have their own ancestral genes. So everything has to be measured. Everything. So our future family practice preventative medicine.

Doctor, we're going to go in. We're going to have a simple blood test. So just like, you know, 23 and me on on turbo. Right on on nitro, not even turbo. Right.

And we're going to come back and they're going to say, okay, here's all the conditions your risks were and, we're going to have to find some solutions.

But I do think that while we eventually will get more and more comfortable with manipulating DNA, we're already doing it. There actually, a couple of.

Gene therapy. Treatments in. Testing. Yeah. Terminally ill, als, from Biogen as well as, spinal muscular atrophy, as well, these antisense oligonucleotide therapies, but, you know, probably for the broader general public, if I tell you, I'm going to give you, an injection, probably using a viral, some kind of, viral mechanism that.

You're. Using in a vector, that's going to scare a lot of people initially. So we have to change belief systems. We have to change cultures. But we really can kind of make, you know, it's like making the 6 million.

I'm old enough to endure, I think, to, Lee majors in the $6 Million Man rally. Rebuild him. We have the technology. Right. Well, I I'm working with NASA.

I've worked with this before. I am a rocket scientist, and I. They need longevity. And so, they'll need to go, where nobody's gone before, and they're going to have to bring stem cells.

And when they see that the radiation is turning on all these things, they need to turn it off. All these oligonucleotides are chicken soup for the genome.

Just fantastic. Well Dr. Urnovitz this has been just an incredible conversation and I, I for one I'm very grateful for you know Brett with DrTalks to have connected us and have brought to you on both as a speaker and as a sponsor of the Parkinson's Summit.

I'm so grateful for the work you're doing. I know that my research center, looks forward to clap. You know what we can do to find some common ground for collaborative research?

I'm going to be launching a podcast. Am I going to have you back? This is such important information for folks, and I hope you guys have gleaned, you know, a good chunk of what we're talking about and go back and listen to it again, because this really is the future of medicine.

Yes, it is. And I look forward to working with you because we need to make tools for for the clinicians. And your work is fantastic. So let's let's change the world together.

Thank you so much. Thank you very, very soon.