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Speaker 1: Navigating Parkinson's disease can be challenging, but we are here to help. Welcome to The Michael J. Fox Foundation podcast. Tune in as we discuss what you should know today about Parkinson's research, living well with the disease, and the foundation's mission to speed a cure. Free resources like this podcast are always available at michaeljfox.org.

David Kumbroch: Hello and thank you for joining us for the Parkinson's Science POV podcast. This is a resource for people with Parkinson's, [inaudible 00:00:48], and supporters, as well as researchers studying neurodegenerative diseases. I'm David Kumbrock, the host and a senior science writer here at The Michael J. Fox Foundation. I'd also like to welcome Dr. Jamie Eberling and Dr. Roger Gunn.

Jamie Eberling: Hi, everyone.

Roger Gunn: Thanks, David.

David Kumbroch: Jamie is the senior vice president for research resources at MJFF and Roger is the chief science officer at Xing Imaging. Now, imaging technologies or transforming how we understand Parkinson's disease, from what's happening in the brain to how the disease progresses over time. For years, researchers have relied on clinical observations to track Parkinson's, meaning what clinicians see as they're treating people with PD over time, but imaging offers a window into the biology behind the symptoms.

There are several approaches to imaging Parkinson's working their way through the pipeline. For example, with new tools and development, scientists are getting closer to visualizing the hallmark protein alpha-synuclein directly in the living brain. These advances could reshape how we diagnose, monitor, and even treat Parkinson's in the future, but it's not the only imaging approach [inaudible 00:01:53]. Let's start with a broad view. Why is imaging such a critical focus for the Michael J. Fox Foundation when it comes to advancing Parkinson's research? I'm going to look to Jamie for this one.

Jamie Eberling: Sure. Imaging is really the only way to visualize what's happening in the brain in a living person. It's really incredible that we can actually do this at all. You mentioned alpha-synuclein, for example. That's a pathological protein that accumulates in the brain in people with Parkinson's disease. Right now, we have no way of seeing that in the brain. We think that it's closely linked to the clinical symptoms and the progression of the disease. So to be able to track the disease by looking into the brain with imaging and being able to see the pathology, this would be a great advance and really accelerate clinical trials because they'd have a good marker of what's actually happening into the brain and how it responds to treatment.

There are different types of imaging. As you mentioned, alpha-synuclein is our biggest priority, but there are other types of receptors and neurotransmitters and even brain structure that we can use imaging to monitor over time. I think, for us, our biggest priority since I've been at the foundation for over 16 years has been to develop a way of imaging this pathological protein alpha-synuclein in the brain. While we're not there yet, there's been a lot of progress made, particularly over the last two to three years. We're hopeful that we're getting close. There's a way of imaging pathology in Alzheimer's disease and it's been really transformative for clinical trials in Alzheimer's disease, so we're hoping that the same thing will happen in Parkinson's disease.

David Kumbroch: Now, I'm going to look to Roger. From a scientific standpoint, what kinds of brain imaging technologies are proving most promising in helping us understand or track Parkinson's disease today? I want to start with where we're coming from in imaging, what technologies have defined it to date, but also where we're going in imaging.

Roger Gunn: Yeah, it's a great question. I think, as Jamie was alluding to, it's a very exciting time for imaging in Parkinson's, certainly the most exciting time in my career. Also, maybe taking one step back, I think recently in the last year or so, we've had the introduction of the neuronal synuclein disease, the concept of this, and the integrated staging system that goes with that. This comes down to highlighting the important biological targets that we need to be able to image. This neuronal synuclein disease looks to be able to understand it, one needs to image both synuclein and dopamine. What comes early in the disease is the accumulation of this alpha-synuclein protein as this misfolded protein, which causes ultimately neurodegeneration to subsequently happen, which is when people start losing dopamine neurons and this comes a little bit later.

Now, what we've been able to image today, we've been able to image the loss of these dopamine neurons that happens later in the disease. You can do that using DaT scan. This is dopamine transporter imaging, which can be done with SPECT systems. You can also image dopamine neuronal loss with PET imaging agents. They're equivalent to dopamine transporter agents with PET. And also things that are similar to that, agents that have their targets expressed on dopamine neurons. There's something called VMAT2. But all of those imaging agents and technologies I've just referred to, they all only allow us to image dopamine neuronal loss.

Now, that's important because that's a valuable marker to look at, both for diagnostics, for use in clinical trials, but the ability to image alpha-synuclein would be truly transformative. That comes early in the disease. At the moment, the only way we can assess that is with a synuclein seeding assay that's using CSF samples. But that, in conjunction with dopamine imaging, has formed the foundation of this integrated staging system, which is really valuable in helping us to understand where somebody is on the pathway in this disease.

Synuclein imaging agent development, very exciting. We're taking one of these PET imaging agents into humans in our facility in New Haven this year, so we're very excited about the prospects of that based on the preliminary data we already

have. I think it feels like we're on a cusp to have a successful alpha-synuclein imaging agent in the next 12 to 24 months. If I had to put money on it, that's where I'd say.

David Kumbroch: Well, that's really encouraging. I do want to highlight one of the nuances you brought up there, the neuronal synuclein disease and the integrated staging system that goes along with it. Can you explain a little bit about what that is, that new thinking about the disease definition, and how that relates to imaging?

Roger Gunn: That new definition really comes down to say it's actually a biological definition rather than a clinical one. This is why molecular imaging techniques such as SPECT and PET are so important, because they allow you to image molecular targets. We're able to image dopamine neurons right now, the concentration of them, and we hope to be able to image the concentration of alpha-synuclein within the next 12 to 24 months. That would enable a very accurate biological characterization of an individual. I think what this staging system has brought to the fore is that this is a more accurate way of understanding where somebody is on the pathway of the disease. It's akin to what's being developed in the field of Alzheimer's with amyloid and TAV forming similar molecular targets to characterize patients.

David Kumbroch: Really helpful. Thank you. I want to make sure that we trace the foundation's efforts to support some of these technologies. Jamie, can you tell us a little bit about the foundation's portfolio and how it includes some of these ambitious imaging initiatives? Walk us through how MJFF supports the development of those technologies.

Jamie Eberling: Yeah. For PET and SPECT imaging, we use radio-labeled drugs essentially, or tracers we call them, but it's essentially a radio-labeled radioactive drug that's injected into the bloodstream. It accumulates in the brain where the target is. So if the target is alpha-synuclein, it would accumulate in the brain where alpha-synuclein is located, and then you can use a PET scanner to actually make an image. We have a portfolio of many different targets that we're working on, but by far and large, the biggest priority is alpha-synuclein. Most of our funding and effort has gone to developing a way to image alpha-synuclein in the brain.

We've also, though, focused a lot on dopamine imaging and I think it's been more useful than we thought it would be. Our big biomarker study, PPMI, collects various different types of biomarkers, including fluid markers from blood and CSF, but also imaging using DaT scan so that we can image dopamine in the brain. In this study, the patients are very well characterized because they get all these different types of tests done, and we can see how their clinical symptoms relate to different aspects of biology, including dopamine in the brain.

What we found is that you can actually measure changes in dopamine in the brain over at least a couple years, which hopefully a clinical trial is over about a year. To be able to see a change in dopamine over a year means that you could actually track the progress of the disease and hopefully response to treatment using dopamine imaging. Most of the clinical trials that are focused on disease-modifying therapies now use DaT scan in their trials. So far, it hasn't paid off in

that none of the drugs has shown a change in DaT scan over time, but we think it's possible.

Over the past few years, largely due to the work of Roger and his colleagues, there's been a big improvement in how we can measure dopamine using DaT scan and it has to do with the way we analyze the data. It seems to reduce the variability that we see so that, if you look over time, you would have a better shot of seeing a change over time and perhaps a response to treatment. I would say right now most of our funding has gone to improving dopamine imaging and developing alpha-synuclein imaging, but there's also other targets that we can look at. One that comes to mind is neuroinflammation. We know that this is important in neurodegenerative diseases in general, including in Alzheimer's disease, but also in Parkinson's disease.

What we don't really understand is where do we see inflammation in the brain? When do we see it? What does it look like over time? Does it get worse over time or does it plateau and then decrease and maybe it's worse earlier in the disease? We don't know that, because right now we don't have a great way of imaging inflammation in the brain. We do have some tools that we can use, but they're not great. They need improvement and there's probably other targets that we could look at that would enable us to do this.

We let the therapies guide our imaging strategy. There's a lot of therapies that target inflammation. We need a better way of imaging it in the brain. There's a lot of therapies that target alpha-synuclein. We need to be able to image it in the brain. We have a portfolio of probably 20 different targets, something like that, that we're looking at, but as I said right now our main focus is alpha-synuclein and continuing to learn more about how dopamine imaging can help us.

Roger Gunn: Maybe I can just jump in on the end of Jamie's comments there, because I think they're really valuable. I think inflammation is incredibly interesting and important, but it's actually very difficult. It's multifaceted neuroinflammation and also applies to many different diseases, so there's a lot of complexities in trying to develop a successful neuroinflammation marker. That also highlights why alpha-synuclein is so important because it's much more specific to Parkinson's disease and relevant in that space. If we can be successful there, that's certainly the top prize, as Jamie and I both know.

David Kumbroch: That's really excellent information. I appreciate the breadth of it. I do want to drill down a little bit more on alpha-synuclein, misfolded alpha-synuclein, the hallmark of Parkinson's disease. Detecting and tracking it over time would reveal a lot about progression. Now, Roger, you've been deeply involved in the science behind imaging tracers. What are the technical and biological hurdles in developing an alpha-synuclein PET tracer and how close are we to overcoming?

Roger Gunn: Probably the biggest challenge to develop an alpha-synuclein PET tracer is the low density of the target. The higher the concentration of a target, the easier it is, because what do you have to do to develop a PET tracer? You have to design something that's going to specifically bind to that target. The lower the concentration, the higher the affinity it has to have to this target. That can

become difficult, particularly as you go down to lower concentrations. The other challenge you have if you're looking to image something with a small concentration is that many of these drugs also bind to other things, other misfolded proteins, sometimes amyloid tau, those kind of things. Those exist at a higher concentration, so you need something which is called high selectivity over your other targets; otherwise, your signal will be lost in a sea of other things.

One other thing as well is non-specific binding. Most drugs will bind into fat and lipid in the brain as well, so this gives you a background. You want a low level of non-specific binding. You want very high selectivity and high affinity for alpha-synuclein, and then you want various other factors that are common to development of any brain tracers. You need good delivery to the brain, so plasma clearance into the brain. You also don't want any radio-labeled metabolites forming that, again, could contaminate your signal. There's a range of particular criteria one needs to meet, but the biggest challenge is the lower density of this particular target with synuclein.

David Kumbroch: Got it. Now, Jamie, once these tools get developed, how do you envision them being used in research but also in real world situations like clinical trials and maybe even clinical practice?

Jamie Eberling: I mean, I think clinical trials for sure. Once we have a PET tracer for alpha-synuclein, the companies that are developing treatments, we'll use that tracer right away. Oftentimes, what we've seen in the past is when developing new PET tracers, the first one isn't often the best one. We learn from it. Sometimes it's the best one, but oftentimes it's not. And so we build upon that and we optimize and we come up with something better down the line. But I think that the companies will be all over anything that we develop and they would use it right away. Specifically for any trial that is developing a therapy against alpha-synuclein, of course, they want a tool like that, but any trial that is developing something that is meant to change the progression of the disease would likely be interested in using an alpha-synuclein PET tracer as well. I think clinical trials, clearly it'll be a game changer.

For diagnostics, not so clear that it's as important there. We do have a diagnostic where we're able to measure alpha-synuclein and cerebral spinal fluid. We're hoping to develop something where we can measure alpha-synuclein in the blood and, of course, that would be much cheaper and easier than imaging. But without imaging, we don't really know what any other marker is actually telling us about what's in the brain. If we measure alpha-synuclein in cerebral spinal fluid, we think we know what it says about what's in the brain, but it doesn't tell us anything about where in the brain there's alpha-synuclein. We don't really understand the link between what we're measuring in cerebral spinal fluid and the amount of alpha-synuclein in the brain.

Right now, what we can measure in cerebral spinal fluid does not track with the progression of the disease. So it's a way of saying, "Yes or no, you have alpha-synuclein in your brain, but that's about it," which is a big game changer. Clearly, that was a big step forward just a few years ago, but we need something better. I

think in order to really understand what you're measuring with any kind of peripheral biomarker, you need the imaging piece as well.

David Kumbroch: I want to dig deeper on that with you, Roger. When we zoom in on those clinical trials and we think about the ways that imaging biomarkers interact with the other biomarkers that Jamie mentioned, what kind of unique opportunities do we have for improving clinical trials by improving our imaging tools?

Roger Gunn: I guess it's a couple of main ways they used in trials. One is enrollment. To ensure that you're actually enrolling the right subjects in your trial, i.e., those that do have Parkinson's disease, and also to recruit the optimal cohort. If you're developing a particular therapy, it may actually be designed to work at a particular point of the disease, so being able to actually enroll subjects who are at a particular stage of the disease is probably critical as well.

Then it's the ability to actually monitor that signal longitudinally. It's taking images, whether that be every 6, 12 months, and then actually doing the analytics to carefully calculate whether those molecular targets have changed over time with therapy. One would look at that versus a placebo arm. If we were doing this with dopamine imaging, we'd look at a placebo arm and we'd see dopamine neuronal loss happening over 12, 24 months. And hopefully with a successful therapy, what we'd see is actually that would reduce that change over time.

The similar opportunities with synuclein, that's what we're seeing, as the disease progresses there, is likely an accumulation certainly early on in the disease. Being able to modify that accumulation, reduce it using some therapy would be really valuable endpoint to be able to directly measure that in the brain of a Parkinson's subject. This is something you can't do any other way. That would be a real game changer if we could do those kind of things.

David Kumbroch: Incredible opportunities there. I do want to note that this kind of imaging advancement can't really happen in a vacuum. Jamie, can you tell us how the foundation has worked with academic, biotech, and pharma partners to push those tools forward in a unified way?

Jamie Eberling: Yeah. I think when I first started at the foundation 16 years ago, there wasn't a lot of activity going on in the field to develop an alpha-synuclein PET tracer. There were a few groups working on it here and there, but there weren't really the tools needed to even start working on it. We saw our role then as that we had an opportunity to drive what was happening and to recruit people to work on this. Part of what we did was just to reach out to great radio chemists that we happen to know and they're usually disease agnostic. They work on different targets in the brain, but they usually don't work just on a single disease. We had some workshops and got together the real experts in radio chemistry and begged them to work on this quite literally. That was part of it.

We also, early on, put together a consortium that included academics and companies to work together on this. We see this as a pre-competitive space. The companies should be competing on their therapeutics, not on the tools that they need for the clinical trials, not on the biomarkers. I think most of the companies

that we work with agree with that position, which is great. So just being there in the beginning to lead the way and bring the groups together... We organized meetings where we could talk about the latest research. In the beginning, it was hard to come up with an agenda for those meetings because there wasn't a lot to talk about yet, but I'm happy to say now that there's a lot of activity in the field, both in academia and at pharma companies, biotechs that are interested and are actively working on this.

A lot of what I'll say my job is just to know what's going on out there and to talk to the people even if we're not funding them and to come up with opportunities where we can bring people together to talk about the progress that's been made and share results. I think it's been pretty successful in that regard. Things really started progressing more quickly. I would say over the past five years, we've just put in a lot more funding, our own funding into this, and we're happy to fund both academic groups and companies that are working on this. I think just the collaboration has been helpful and we've seen the fruits of the labor of all these different groups that are working towards a common goal.

David Kumbroch: Fascinating. Roger, I want to get your perspective there, too, because you've worked across industry and academia. From your viewpoint, what's the landscape like for turning a tracer from the lab into something that can be used across, say, multiple clinical trials?

Roger Gunn: I mean, yeah, that's a great point. If we think about the different phases of drug development, clinical ones, a phase one study is a small study that can be done at a single site. If these traces are being developed, you can run phase ones fairly easily because they exist as a single site, so it's just then getting the subjects there. But phase being able to set these things up to use them successfully for phase two and phase three trials, these are the sorts of trials that we'll have a couple of hundred subjects in phase two and up to thousands in phase three. That requires one to set up production facilities for these imaging agents such that one can run larger phase trials using them. That's something that Fox is supporting with agents as we speak right now.

Looking to set that up for a platform, really, for the most relevant Parkinson's imaging agents so that they can be quickly deployed with dopamine imaging agents in the first place, but getting that network ready for alpha-synuclein agents so that they can quickly support phase two and later phase trials. Yeah, it's something that needs to be typically done commercially. As you're setting up a large network like that, academics would tend to work in the single site setting.

Jamie Eberling: Maybe I'll just add there. I think this is a really important point, because if we develop a PET tracer and nobody can get their hands on it, it's not going to be very useful. The deeper you get into the development of a tracer, the more expensive it gets. It costs millions of dollars to get to the first in-human study, but after that, it costs many, many millions to get additional subjects at additional sites, to set up sites that can actually produce the tracer, and deliver it to the clinical sites so that they can use it. This is where the real big bucks come in.

We're really trying to look forward and put into place a plan for how we can do that and how we can fund it. I think the foundation can play a really unique role there by identifying sites across the Northern America, Europe, other places as well globally that we know have access to Parkinson's patients who do good work, who have the imaging facilities and resources available, so that we can set up the best sites for success in clinical trials ultimately. We're putting a lot of work into doing that now and we have a lot of great supporters available to us who are interested in this effort. I think once we identify something that looks good in the first in-human study, we can quickly expand upon that and prepare to deploy the tracer so that it can be used by companies in their trials.

David Kumbroch: It's so encouraging to hear that we're poised to step into action here. Clinical trial is, obviously, a huge way that this is going to impact the Parkinson's landscape, but I don't want to skip entirely past diagnostics. Jamie, can you tell us a little bit about the outlook on that front? What ways are we close to a big breakthrough in diagnostics and imaging? What ways are we still far away and how does it fit into the landscape overall?

Jamie Eberling: Well, I mean, I think the biggest thing that's happened in the past few years for diagnostics is this seed amplification assay that is able to measure alpha-synuclein in cerebral spinal fluid. As I said before, it's a yes-no answer. Do you have it or do you not? It doesn't tell you anything about the severity of disease and it doesn't tell you anything about the progression of disease. There's still work to be done on that front.

For imaging, for diagnostics, I mean, we use DaT scan to confirm a diagnosis, but still it's clinical symptoms that neurologists use to diagnose a subject. Oftentimes, they can be missed diagnosed early in the disease, especially if it's something that has symptoms in common with Parkinson's disease but maybe doesn't even involve alpha-synuclein at all. We know there are diseases that involve tau that can mimic Parkinson's disease and it's often difficult to make a diagnostic early. So being able to image dopamine in the brain and say, "Oh, this person has these symptoms and they have reduced dopamine in specific brain areas, it's likely Parkinson's disease," it's been helpful in that regard.

If we can image alpha-synuclein in the brain, potentially we could see alpha-synuclein very early even before there are symptoms. I don't know how easy that will be to do since, as Roger mentioned, there's small amounts of alpha-synuclein in the brain in diagnosed Parkinson's patients. As you go earlier, it's probably lower levels. I don't know if we can image it very early in the disease, but that's something that's an open question. Potentially, it could be used to diagnose patients before there's any symptoms of the disease at all or, else as I mentioned, also to confirm that another biomarker that we can measure in the blood, for instance, is telling us what we think. It's telling us about what's happening in the brain.

David Kumbroch: Really helpful. Thank you. Roger, what excites you most about where imaging science is heading in Parkinson's? What breakthroughs could we see in the next 5 to 10 years that really have you feeling enthusiastic?

Roger Gunn: I think there's three really important things that define imaging in most spaces. But if we focus on what they are in Parkinson's, it's the scanners that acquire the data, it's the imaging agents that allow you to image particular biological targets and their performance, and it's the analytical methods that are used to analyze the scans you acquire. It's those three things. Improvements in each of those three areas can substantially increase your signal.

If we take those in order, the first one. Scanners has been a remarkable step change in brain PET scanner technology in the last year with the development of the United NeuroEXPLORER scanner. This is something that's been led by Rich Carson at Yale in conjunction with Simon Cherry and Ramsey Badawi at UC Davis and the United Imaging team. For the first time in 20 years, it's the first time that somebody's developed a dedicated brain PET scanner. For those last 20 years, people have been distracted by oncology and whole body imaging.

This machine is fabulous. We just have one now at Xing and this is thanks to Fox. I think we really see the value in such a machine. It has transformative specifications. It images at 1.4 millimeters. Most brain scanners before this were imaging at around 4.5 millimeters, so it's a bit like looking at images through an old box Brownie and a modern high resolution digital camera. It's remarkable, some of the structures you can see. Why is this so important for Parkinson's? Because some of the key areas where alpha-synuclein accumulates and where we see dopamine neuronal loss, they're in small structures like the substantia nigra. These things are half a centimeter across, so you actually need really high resolution imaging to be able to actually develop alpha-synuclein tracers in the first place and to image those changes over time. Transformative changes in scanner technology and that's fabulous.

If we talk about the imaging agents, I think we're on the cusp of alpha-synuclein PET traces that are going to be successful, particularly when you combine those with the NeuroEXPLORER and then the advancement in analytical methods, which continue to abound and make substantial differences. We've just recently re-analyzed, as Jamie mentioned, all of the PPMI DaT data. This is from the Parkinson's Progression Markers Initiative study led by Ken Marek. This looks at DaT imaging over time. What we were able to show is that we substantially increased the performance, so we were reducing the variance.

To put it in context, if we were using this data to power a clinical trial compared to the previous analysis, we need half as many subjects. It's a dramatic change. It's really trying to push on all three fronts, the scanner technology, the imaging agents, and the analytics, because if you combine that together, you get a dramatic increase in signal-to-noise, and that's likely what we're going to need for successful synuclein agents.

David Kumbroch: Jamie, I want to give you a chance to answer the same question. What excites you most about where imaging science is headed in Parkinson's?

Jamie Eberling: Well, the NeuroEXPLORER that Roger just talked about is one of the most exciting advances, maybe the most exciting advances that I've seen in my career, I mean, at least in Parkinson's disease. I think in Alzheimer's disease, it was the

development of the first amyloid tracer and the first tau tracers. I think the same thing's going to happen in Parkinson's disease, but that scanner, it's just unbelievable the images that you get with that scanner.

I did PET imaging research for many years before coming to the Fox Foundation and there were incremental advances in PET scanner technology. This is not an incremental advance. This is a huge advance and it's taken at least 20 years to get to this point. I remember some of the technical aspects of the scanner that had been improved. We were talking about them over 20 years ago and nobody could make much headway. Super excited about that.

I think we have to consider that maybe the levels of alpha-synuclein are to the point where we need that advance in technology in order to be able to image alpha-synuclein in the brain. It's possible. We don't know. We hope not, but it's possible. At least now we have that technology that could enable the first successful alpha-synuclein PET tracers. Super excited about that.

But the other thing I'll point out is that, up until a few years ago, maybe three years ago, we saw one alpha-synuclein tracer being tested in humans at a time. Oftentimes, there was nothing being tested in humans. It was sort of one at a time and nothing worked all that well. Now, there's over half a dozen new tracers that are being tested in humans. That's super exciting and it tells me that we are headed in the right direction, we are making progress, and it gives me great hope that we will get there soon. Maybe this new tracer that Roger and his group will be testing on the NeuroEXPLORER will be the breakthrough that we've been waiting for.

And then the analytics as well. I mean, I completely agree with all of Roger's points here. The dopamine imaging has really shown me what a huge difference the analytics can make. I mean, if you're cutting a sample size in half, that's a pretty big deal. Companies are going to think it's a pretty big deal if they don't have to spend money on twice as many subjects. I think that it's a really exciting time for imaging in Parkinson's disease in general for all of those reasons and I'm very excited and optimistic that, for alpha-nuclein in particular, we're going to get there very soon.

David Kumbroch: Thank you so much. There's a lot to be enthusiastic about here and I appreciate you both for outlining it. I want to say thank you to our guests, Dr. Jamie Eberling and Dr. Roger Gunn.

Roger Gunn: Thanks, David. It's been great talking to you. Some great questions and really pertinent. It highlights how exciting imaging is looking at the moment.

Jamie Eberling: Yeah, I agree. I'm glad that this podcast is taking place now, because I think we can end on a more optimistic note.

David Kumbroch: For those of you listening, we do have a number of resources available on our website that might be helpful to you. If you are interested, for example, in the Parkinson's Progression Markers Initiative that was referenced a few times, that's

available. We also have the Fox Trial Finder that will help connect you with recruiting studies in Parkinson's disease. Also for researchers, we've got our researcher resources page that will help outline all of the different ways that the Fox Foundation helps support Parkinson's research and could potentially help you in your efforts. You can find the links to all those resources in the show notes and on michaeljfox.org. But for now, thank you for joining us for the Parkinson's Science POV podcast.

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