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The Michael J. Fox Foundation:

Navigating Parkinson's disease can be challenging, but we're 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.

Maggie Kuhl:

Welcome to this podcast edition of our fall year-in-review research roundtable, where we gather with expert panelists to delve into the state of Parkinson's research and drug development, the incredible steps forward we've taken this year, and what to watch out for over the next 12 to 18 months. I'm Maggie Kuhl, director of research communications at the Foundation, and I'm here with our Co-Chief Scientific Officers, Dr. Brian Fiske and Dr. Mark Frasier. Thanks for joining me.

Mark Frasier:

Good morning.

Brian Fiske:

Good morning.

Maggie Kuhl:

Thanks for doing this today. So, as you know, given COVID, not everyone who normally would have joined our roundtable in New York was able to be with us this year. So we are sharing highlights from the event in this podcast. Rest assured that what you'll hear about represents just a small fraction of the work of the Foundation, and others, in Parkinson's research. We think some of these are the most important stories for patients, families and researchers to follow. So to kick us off, I'm actually going to play a clip from our moderator, from the New York roundtable, Dr. Katie Kopil, who is vice president of research partnerships at the Foundation, carried us through the conversation in person, and we're going to hear from her on where that conversation started.

Katie Kopil:

We're going to start where we always want to, how close are we to a cure?

Maggie Kuhl:

So, Brian, we'll touch on some specific targets in our conversation today, but curious your thoughts in general. Where are we towards this urgent need to slow or stop disease? How close are we to a cure?

Brian Fiske:

No, really great question. Of course, the billion, trillion-dollar question for people with Parkinson's. And probably, the thing that has been a big driver of that, over the last 20 years or so, is really the power of genetics. And as a reminder for those who haven't maybe had a biology class in a while, genes are basically the instructions in the cells that tell the cell how to make particular proteins. So when you

change the underlying genetics, you essentially change the recipe for those proteins. And so when we look at that in Parkinson's, ultimately, it points to biology in the cell that when altered can lead to Parkinson's disease.

Brian Fiske:

And with that knowledge in hand, we can actually look at everybody with Parkinson's and start asking the question, "Okay, is that same biology altered in those people as well, even if they don't have those specific genetic changes?"

Maggie Kuhl:

So it sounds like genes teach us where to look. And some of what we find might be shared from people who don't have those genetic changes, but really applicable across the broader population. And, Mark, that is the story of a key target called alpha-synuclein that we talk a lot about in Parkinson's research.

Mark Frasier:

Yeah, alpha-synuclein is, if not the most important target, one of the most important targets in Parkinson's. And the reason researchers are so interested in it is really for two reasons. One is everyone has alpha-synuclein protein in their body, whether you have Parkinson's or not, but in people with Parkinson's Disease, alpha-synuclein protein becomes sticky and misfolded and clumps up in the brains of people with Parkinson's, in particular, brain cells. And the hypothesis is that if you can reduce this clumpiness, this stickiness in alpha-synuclein, you can actually slow or potentially even stop progression of Parkinson's disease. The second reason that researchers are really interested in alpha-synuclein is a story that Brian described, where it was a story from our genetic understanding of Parkinson's Disease. And there's rare families that have a high prevalence of Parkinson's in multiple generations in their families. Just having too much alpha-synuclein in these rare families actually causes Parkinson's disease. And so this is why researchers really are interested in alpha-synuclein as a target.

Maggie Kuhl:

At the roundtable, MJFF special advisor and president and senior scientist at the Institute for Neurodegenerative Disorders in Connecticut, Dr. Ken Marek, recapped the state of alpha-synuclein therapies right now.

Ken Marek:

It's a very exciting time, where we're seeing a number of different ways of reducing the amount of synuclein in the brain in individuals with Parkinson's disease. I think we have an enormous number of trials and opportunities in front of us.

Maggie Kuhl:

So, Mark, tell us a bit about the trials.

Mark Frasier:

Sure. There's a real diversity of strategies that different researchers and groups are taking. Some of the recent news, we've had some successes and some failures. So earlier this year, there was a report from a company called Biogen that actually reported they were not successful in their therapeutic approach. It was an immunotherapy approach that did not seem to change Parkinson's disease, and they've halted

their program. But within the same month or so, there was a report out of the company Roche that is developing a similar immunotherapy that reported some positive findings in their phase two clinical trials. And so they are moving forward with their program.

Mark Frasier:

We also just, last week or so, had an announcement from company called MODAG that we supported the early development of their alpha-synuclein drug program. And they recently partnered with Teva, which is a larger pharmaceutical company, that's going to take their development forward. So there's actually around 13 different clinical trials that we're tracking all developing different alpha-synuclein-based drugs. We are optimistic that several of them will look promising.

Maggie Kuhl:

Yeah. And I think it's important to add, too, that we learn from every unsuccessful trial. I don't even like to use the word failure because you take something away, whether it's crossing one approach off the list and devoting more resources to others or finding something about the biology that informs your next generation. So important to say that a discontinued program is not for not. And the company that you mentioned, Biogen, that is no longer working on that particular therapy in alpha-synuclein, is doing a lot against another target LRRK2 or L-R-R-K-2. And we had on our panel as well, the senior director of the neurodegeneration research unit at Biogen, Warren Hirst. Dr. Hirst did share an update from some of the deals that Biogen has had in the LRRK2 space and the work that he's doing there.

Warren Hirst:

Last year, we partnered with Denali. So we're working with them and basically ramping up to start Phase II studies in the very near future with the small molecule. And then the program that I lead behind that, which is the ASO program, we're in the Phase I studies.

Maggie Kuhl:

So, Brian, I'm going to toss it to you to tell us more about LRRK2, and Warren mentioned another abbreviation, ASO, that stands for Antisense oligonucleotide, I believe. So maybe you could explain that as well.

Brian Fiske:

Sure, sure. Yeah, mutations in the gene LRRK2 are actually really exciting. Two independent groups discovered those in about 2005 in two back-to-back papers. And it really opened, I think, the genetic vault in a way, because soon after those initial publications, people started looking at lots of other families and other populations and found that mutations in LRRK2 genes seemed to explain about two to four percent of Parkinson's overall, but in certain populations, actually people of Ashkenazi Jewish descent and people of North African or Berber descent, anywhere between 30 and 40 percent of their Parkinson's cases might be linked to mutations in this gene, LRRK2.

Brian Fiske:

So really powerful genetic discovery that opened the door. Now when they started to look at the biology of the LRRK2 proteins, the protein that the LRRK2 gene instructs the cell to make, what they found very quickly was that the LRRK2 protein is a type of protein called a kinase. This is actually a common cellular mechanism that the cells use to essentially signal changes in the cell.

Brian Fiske:

Once they discovered that LRRK2 is one of these types of kinases, it allowed drug makers very quickly to start thinking about how to make drug against that type of protein.

Brian Fiske:

What we know about the mutation that is linked to the LRRK2 is we think that it increases its activity, so it makes the LRRK2 protein more active. Most companies then are focused on the idea, can you try to dampen down the activity of the LRRK2 protein in people with Parkinson's with this mutation?

Brian Fiske:

That's where a lot of the leading programs have started, and so Denali, are leading a small molecule program right now and clinical testing of a so-called LRRK2 inhibitor. And they're one of several companies that are approaching it that way. You mentioned Biogen also has this other interesting program using a so-called antisense oligonucleotide. It's that RNA that then the cell to actually make the actual protein that the cell needs. Researchers have discovered over a number of years that you can develop approaches that can actually target the RNA if you want to say, block the production of the particular protein. In the case of LRRK2, the idea that Biogen also approached was can we actually use a so-called antisense oligonucleotide, which is just a fancy term for an approach that can go in and target that RNA and block it basically, can we use that as another way to reduce LRRK2?

Brian Fiske:

So in effect, reducing its activity. They're currently testing this approach as well, in addition to partnering with Denali on the small molecule approach.

Maggie Kuhl:

I love that about science. There are so many different targets for each single target. There's the instruction manuals, the production factories, what actually happens after the thing's made. And that increases our likelihood of success. Just a timing update, the Denali and Biogen trial, their next phase is slated to begin soon, so we're all anxiously awaiting that. There's a lot of momentum in that department as well.

Maggie Kuhl:

Another thing that Warren had said at the roundtable, it's great that we started those studies, those human trials so quickly after the discovery of LRRK2 tied to Parkinson's. But the Fox Foundation has really been leading innumerable efforts to better understand and measure LRRK2 and other targets while these trials are happening. So Brian, maybe you could just give us a little color on what Warren meant by that?

Brian Fiske:

Yeah, it's really interesting, LRRK2, there may be other ways we can think about it, and even though the drug development around LRRK2 happened very quickly, there's still a lot of biology we don't understand. For example, there's evidence that suggests that LRRK2 might have some role in the immune system. What does that tell us about targeting the immune system for Parkinson's?

Brian Fiske:

Scientists are really looking at the biology of LRRK2 and what that's helping us think about is different ways we might be able to target LRRK2 directly or the LRRK2 mechanism.

Brian Fiske:

So there's a lot of work that's still happening. But we don't want to wait until we know everything before we start making drugs. We need to start making drugs and trying out these ideas as fast as we can and as fast as we feel like we can reasonably and safely do so, but at the same time, continue to explore the biology.

Mark Frasier:

I think it's important to also appreciate that these clinical trials are really experiments, and not every experiment works and has a successful outcome. But what we really want is for every experiment to be informative. So we learn from even the trials that may not demonstrate improvement of Parkinson's disease. And what we've seen, I think, particularly recently, is the sharing of information from these experiments that I think historically sat in the vaults of companies, is more widespread. So companies know that Parkinson's is hard and they're actually incentivized to share data and even potentially samples, bio samples that were collected in these trials, to really learn as much as possible about these trials, whether they were successful or failed.

Mark Frasier:

And I think that's one role that the Fox Foundation continues to play, is to convene these groups together to share information about their experiments regardless of the outcome. And it's exciting to see the willingness to share.

Maggie Kuhl:

Absolutely. Let’s get these trials, these experiments started as safely and as smartly as we can, but let's keep learning and evolving so that we continue to increase our likelihood of success and the speed towards those new treatments.

Maggie Kuhl:

To pivot a bit, the way to curb the numbers of people with Parkinson's might not be only to deliver a therapy after or close to diagnosis, but really remove the triggers that lead to Parkinson's disease in the first place. Can you talk to us about the environment’s role in Parkinson's and some of the Foundation's work in trying to mitigate those risks?

Mark Frasier:

Sure. We know that environment can contribute to many disorders, including Parkinson's disease. We know that it's been shown that Parkinson's is more common in rural areas than in urban areas. We know that certain pesticides in animal models, in rats and mice, can contribute and cause Parkinson's disease in those animals.

Mark Frasier:

And so there is an environmental component to Parkinson's disease. The challenge is that understanding the role of different environmental factors are really hard studies to do. They require hundreds, if not

millions of people, and exquisite amount of data that really tracks what toxicant or environmental pesticide was used and when. And Parkinson's is a chronic disorder that may be influenced by an earlier exposure to a pesticide, but one may not develop Parkinson's for many, many years, so it requires a really different type of dataset to understand these environmental factors.

Mark Frasier:

We recently embarked on an effort to fund more of this research and actually uncovered, through our funding mechanisms, some really interesting datasets that could be used to answer the questions about the role of environment in Parkinson's disease. We're funding a couple focused on military exposures, so we know that traumatic brain injury and head trauma is a risk factor for Parkinson's disease. There also are some datasets that track exposures of toxicants to our veterans and so we're supporting work on that. We're supporting a project out of Finland that is looking at air pollutions and air pollutants and the risk of developing Parkinson's disease. And then also, one in California that's combining the role of genetics and pesticides. It has a really unique database where we have information on individuals with and without Parkinson's. We have their genetic information and pretty exquisite information about which pesticides were used in different farming communities. And so we can start to peel back the onion and ask these questions about what's the role of the environmental factors in Parkinson's disease that will then allow us to potentially remove those environmental factors and advocate for removal of those factors that could actually eliminate or reduce the prevalence of Parkinson's disease.

Brian Fiske:
Just to say, this concept of gene and environment, I think one of the really important parts of this is appreciating that, although at the extremes, there might be fairly pure genetic forms of Parkinson's and at the other extreme possibly fairly pure forms of Parkinson's driven by some of sort of environmental toxin exposure, that they're not mutually exclusive and that there's likely this sort of mixture of gene and environment in the middle that explains possibly a good percentage of the cases of Parkinson's. What's exciting about some of this more recent work, is that we can really start to explore those two components in a more sophisticated way, and I think really uncover more meaningful understanding of the causes of Parkinson's.

Maggie Kuhl:

Absolutely. And while we are building some of that data, we do have some information on some toxicants that do raise risk of Parkinson's disease and our policy arm is supporting legislation to limit or ban use of in particular one herbicide, paraquat, that has been linked to increased Parkinson's risk. If that is of interest to you and you want to learn more, encourage you to check out the facts and take action through our website, michaeljfox.org.

Maggie Kuhl:

One thing that we also know about Parkinson's is that people have a range of symptoms that come with the disease. And Brian, there's been a number of therapies approved over the last two decades, I think nearly 20 or so, against a range of symptoms, the motor, the non-motor effects that come with the disease, and the Foundation has supported a real catalog of innovative approaches over the last year.

Brian Fiske:

Yeah. We can think about those ultimate cures, fixing the underlying damage, repairing the brain, even preventing Parkinson's altogether. That's really our long game, but it's critical, of course, to be thinking

about what can we do about the symptoms today that people suffer today? The cells and one of the key types of cells that degenerate in Parkinson's disease are brain cells that make the chemical dopamine.

Brian Fiske:

We've figured out over the years, different ways of targeting that dopamine, either through giving an early precursor version of dopamine called Levodopa or targeting the breakdown of dopamine. But as the disease progresses, that becomes a little more complicated. Obviously the brain continues to change. Cells continue to die. Drug makers have looked at different delivery mechanisms. For example, two relatively recent drugs that were approved, one is a way of delivering levodopa through an inhaler. And then another approach uses a different chemical that mimics the actions of dopamine delivered as essentially an oral strip, almost like a breath mint type strip. And both approaches really are intended to help people at those later stages of Parkinson's.

Brian Fiske:

A couple of companies out there that are focused on subcutaneous, under the skin type of approaches that can deliver dopamine a little bit more continuously. And so we're looking forward to seeing the outcomes of those trials.

Brian Fiske:

Focusing on dopamine and the motor symptoms, of course, aren't the only progress we want to see. It's really the non-motor features, cognition and dementia, constipation, and sleep, and a whole variety of other features that are really, I think, problematic for people with Parkinson's, especially as the disease progresses. Another big problem that especially in later stages is our problems with gait and balance.

Brian Fiske:

People with Parkinson's will often, especially as the disease progresses, start having issues with their gait and balance and can lead to falls. And so what we've seen there is actually a lot of innovation as different companies try to develop different types of devices and assistive devices so that people can hopefully walk a little bit better, walk a little bit more confidently with their symptoms. And that's another area that we're seeing some real promise and progress happening.

Maggie Kuhl:

Great, great. I think a theme of a lot of the work here is trying to help people with Parkinson's and our roundtable panelists, Patient Council member, Bryan Roberts said it best.

Bryan Roberts:

The thing that excites me with the Parkinson's research is that we're not just following one thing. We keep looking at different areas, but where does this come from? It comes from a patient's voice and that's really important.

Maggie Kuhl:

A theme of a lot of biomedical research, our Foundation as well, in the recent past has been listening to more patients with broader inclusivity. We had on our panel at the roundtable, managing director of the Aligning Science Across Parkinson's initiative, Dr. Ekemini Riley, which is supporting, her initiative ASAP, is supporting a program to broaden Parkinson's understanding.

Ekemini Riley:

There's a lot we don't know. And so thinking about how best we address those gaps so that we get concrete answers in a shorter amount of time. And we're fortunate to be partnering with the Fox Foundation. They bring both implementation prowess, but also thought leadership and expert scientists on staff.

Mark Frasier:

Yeah. I mean the ASAP initiative is really exciting. It's a large initiative. That's providing significant amount of funding around open science and sharing and collaboration across research efforts. There's really three main activities within ASAP. One is to fund some basic understanding in laboratory to understand what causes Parkinson's disease, understand the basic biology and fundamental biology contributing to Parkinson's disease. The second is a large scaled genetics initiative to expand the number of individuals for whom we have genetic information to really increase our understanding of the genetic contribution to Parkinson's disease, particularly to individuals that may not have participated in research in the past. And then the third major activity is to fund a study that is focused on measuring and diagnosing Parkinson's more precisely. This is the Parkinson's Progression Markers Initiative study that the Fox Foundation has supported for some time. And The Michael J. Fox Foundation is an implementation partner with the ASAP initiative. We're really excited to be involved with it.

Maggie Kuhl:

Brian, Parkinson's genetic understanding from a population of European descent. And as Mark mentioned, ASAP's effort to change that and really expand this pool of samples and thereby our understanding of the disease.

Ekemini Riley:

Right now about 95 percent of the genetic data sets that have been produced on Parkinson's disease and several other diseases are concentrated in people of European descent. How do we expand that? How do we really understand what we're going after?

Brian Fiske:

The Global Parkinson's Genetics Program, or GP2 for short. It's a huge, massive five-year effort to try to essentially collect genetic information from more than 150,000 people around the world. They'll be looking at genetics really in two ways. They want to obviously look and explore in a large number of people with and without PD, look for a common genetic signals and signatures of Parkinson's disease. But they're also going to be looking at a number of families with Parkinson's. There probably is a higher risk genetic cause of their Parkinson's, but yet where that signal has yet to be identified.

Brian Fiske:

Also a global collaborative environment, people around the world who are all involved in the GP2 effort, investigators from for many, many countries, including many underrepresented populations and countries. And all this data ultimately being made available is a genetic resource for the Parkinson's research community to uncover and hopefully identify new therapeutic targets for Parkinson's disease.

Maggie Kuhl:

Just over the last week or two ASAP and MJFF as its partner announced $132 million in funding over three years to 14 teams looking at bringing circuitry and brain-body connection, what's going on in the brain and the body in Parkinson's disease and how it's all connected. Mark, you mentioned that ASAP is also partnering in our PPMI study. Ken Marek, who we heard from before is the principal investigator of PPMI.

Ken Marek:

The Parkinson's Progression Markers Initiative or PPMI is a project that began about 10 plus years ago, was really an enormously innovative project that Fox developed with the idea that it was important to think ahead. The information that's been acquired as a result of that project has been used to really accelerate therapies. And that's been great, but what is even better is that now with the support of both Fox and ASAP, we can do so much more.

Mark Frasier:

PPMI has found individuals, volunteers, both with Parkinson's and without Parkinson's and asked them to contribute to a study by going to clinical centers, usually academic hospitals and contributing imaging, brain scans, bio fluids, blood and spinal fluid and urine, as well as undergo some clinical testing by a neurologist to test memory and motor symptoms. And the study's happening all over the world in about 50 different sites globally. And to date in the last 10 years, there have been about 1,500 individuals with and without Parkinson's that have contributed information and data to this study. And as Dr. Marek mentioned, it's already really accelerated our understanding of Parkinson's disease. But more importantly, the data has been used to inform clinical drug programs and design of clinical trials in a way to make those clinical trials more informative. We are expanding, as he alluded to, expand from 1,500 individuals to about 4,000 individuals globally that are participating in this study. And I should mention that anyone that's interested in learning more about the study, whether you have Parkinson's or not, can learn more and see if they're eligible to participate. You can visit the michaeljfox.org website slash PPMI. But this is a really exciting opportunity because we are finding and increasing our ability to find individuals that are at risk for developing Parkinson's before symptoms even develop. And the study can enroll individuals to really understand what changes, what measurements are changing in the body before developing Parkinson's disease? We can use those indicators as ways to identify individuals and potentially even treat before symptoms develop.

Brian Fiske:

There are also are some clinical features that might be early indicators of Parkinson's — loss of smell function is one particular robust, early clinical sign of Parkinson's. It's not very specific, of course, a lot of reasons why you might lose your sense of smell, including unfortunately, over the last two years, people who experience COVID experience a sense of loss of smell. But we do know at least that in people with Parkinson's a good percentage of them do have abnormal smell function. Another clinical sign we've seen are that people who exhibit a certain sleep disorder called REM behavior sleep disorder. And people who have this disorder move around a lot more when they're dreaming and sometimes could be very physically disruptive to their sleep and certainly their bed partner's sleep as well.

Brian Fiske:

So with these various clinical and genetic features, we can actually start to identify people who might be close to developing Parkinson's If we can find those people, and if we can get some safe treatments that can be used in those individuals, we might be able to actually delay the onset of Parkinson's altogether.

Maggie Kuhl:

So many people are using PPMI looking to PPMI for these answers that they seek to advance research and includes industry. Warren Hirst on our panel at the roundtable, spoke a bit about the impact of having these disease measures on drug development programs.

Warren Hirst:

PPMI in terms of the consortia there and there's many industry partners that are within that. And I think this speaks to the open nature of the work that the Fox Foundation are doing and how we realize that we can't just take this on our own.

Maggie Kuhl:

Mark, you alluded to this earlier, open science, this data sharing that PPMI has led the field in and ASAP is also really pushing for and advancing in its programs as well. So Warren Hirst spoke about this as well as Ekemini Riley.

Ekemini Riley:

Open science, open data, this is key to shortening the time to getting information out and actually integrating it into research.

Maggie Kuhl:

Mark, tell us more about open science, why it's so important.

Mark Frasier:

Yeah, well, science and research and drug development can be a very competitive industry, right? And there's incentives to be the first to publish something, or the first to discover some new finding, or the first to get a treatment approved. We see a lot of inefficiencies when sharing is not done and actually slowing of progress when people are being competitive and not sharing tools or information in real-time. And so what the Foundation and the ASAP initiative have emphasized is this more open science concept.

Mark Frasier:

And so that can look in a number of different ways, it can look in the way of PPMI where new data from that study are uploaded every week. Every week, this is an open study where every week there are new data shared, and it's shared through a research portal that is accessed by thousands of researchers around the world. It's not just the leadership of the study that can access the data, anyone around the world can access the data with legit to make credentials. This is all anonymized data, but we think it's important to share.

Mark Frasier:

Open science can look like fostering collaborations across global research teams. So the ASAP initiative in their collaborative research network is funding research across different laboratories that are sharing information in real-time through teleconferences, video conferences, and in-person meetings. They're funding not just within teams, but across teams as these laboratory teams generate useful research tools that they're using in their own laboratories, they're expecting and mandating these teams to share these tools in real-time with the other research groups. And we think by using this open concept and the

foundation has supported this model for a number of years, we really think that we can break down silos, accelerate progress, and get to new treatments and new discoveries faster. And so that's really what we mean when we talk about open science.

Maggie Kuhl:

You mentioned how PPMI data is available. PPMI data is downloaded on average every 40 seconds. So we are not just offering it, people are taking us up on it. And there is this real sense of community and collaboration, which I think as Brian said, leads to real optimism here. And just to round out our conversation going to have a [inaudible 01:06:58] from Ken Marek again.

Ken Marek:

I've been engaged in trying to find therapies for Parkinson's for about 30 years. And I think what is remarkable is that really in the past five to 10 years, the pace of discovery has really accelerated so much.

Maggie Kuhl:

So to close this out, I might ask both of you to reflect on that statement from Ken. What is behind that acceleration of the pace of discovery and what does that mean for the future? Brian, maybe you first.

Brian Fiske:

Yeah, no, I think it's a really powerful statement that Ken makes and for me, when I think of that, I just think of a lot of the work that I support through the foundation is more focused on the therapeutic pipelines. And I look at that list of different trials pretty regularly with the team, as we think about the different types of funding we can give out and just the number of irons in the fire, the number of shots on goal, whatever analogy you want to use. It just the whole pipeline is just so robust right now with ideas being developed and tested for Parkinson's. And so much of that really in the last five to 10 years seeing that progress has been just I think really exciting to see. And obviously, a lot of unknowns ahead as these trials report out the results. And as we talked about before, these are experiments like any other, but again, I'd rather have multiple experiments running in parallel than no experiments at all.

Mark Frasier:

Yeah. I mean, I think it's interesting to hear Dr. Marek say that and I'd like to think that there's two reasons behind that really that pace of discovery that he talked about. One scientific, one not scientific. And the not scientific reason I think is this open science concept through initiatives like PPMI and other consortia that the foundation and other funders have driven where it's really changed the culture of how researchers are collaborating and developing new discoveries and new treatments.

Mark Frasier:

So there's just really been a sea change in not just the culture, but it's really just an expectation that researchers will be collaborating in a way that they hadn't done prior to the last 10 years. I think the other scientific reason or technical reason is that there's been this sort of two waves colliding of really advanced molecular and genetic technologies that have become cheaper and more widespread to enable discoveries along with our computing power and our ability to collect and aggregate and analyze large amounts of data genetic data, and other data that have really accelerated the pace of progress. So, that's been really exciting to see.

Maggie Kuhl:

So it sounds like both what you can do and what you will do with that collaboratively. Well, great. Thank you both so much and thank you all for listening. There are so many ways to get involved. We touched on a lot of different in roads to change here. And Mark, we'll hear more from you on what's happened this year in our webinar on November 18. So join us live on that date, or if you're listening after, you can watch anytime on demand again at our website, michaeljfox.org. Many thanks for supporting what we do. And Brian and Mark, I don't know about you guys, but the fun way to start my morning, so I thought we might do more of these in the future. So if you too enjoyed listening, then stay tuned for more of these and see you next time.

Mark Frasier:

Thanks, Maggie.

Brian Fiske:

Thank you.

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