



The Victory Summit® Virtual Event: YOPD
YOPD: Biology, Treatments, and Living Well Today

Recorded on June 26, 2021

Davis Phinney Foundation

Amy Montemarano, JD (Davis Phinney Foundation Ambassador, YOPD Council Member):
Next up we have Dr. Rodolfo Savica who is a Professor of Neurology of Practicing Neurologists and Movement Disorder and Behavior Specialist at the Mayo Clinic in Rochester, Minnesota.

He's also a PhD. He has a particular interest in early onset Parkinson's, and initiatives regarding the clinical practice and research of early onset, and he is the chair of the International Movement Disorder Society Task Force on early onset Parkinson's, and he leads a lab with the goals of finding a cure and improving the quality of life for people with Parkinson's, Welcome Dr. Savica, thank you so much for being here.

Rodolfo Savica, MD, PhD, (Faculty, Rochester Mayo Clinic):

It is my pleasure. So, I would like to first of all thank everyone at the Davis Phinney Foundation. Thanks all the people, speakers, thanks Melani, thanks, everyone, that I've had the opportunity to listen to and talk to. And that Foundation that gave me the opportunity to talk to you today.

And I wanted to say right away, there is in its own nation is doing an excellent job to feel as we heard earlier on, Mr. Phinney talking about that. There was a purpose fill the gaps that are there for young onset Parkinson's location that working and trying to put people together. That is a very important mission and we're going to talk more about it.

I decided not to prepare slides, not put slides up for a major purpose, because I think that our audience, everybody have heard 50 million times about Parkinson's disease, what he develops, how it goes, what are the proteins, and the problem that we have in these, in this situation is that we are talking about that condition, but he's not very early on one is know what we're facing here today.

We're using knowledge of a condition that affect people in their 70s. And we are translating it in a condition is affecting people in their 40s/50s. The important thing that I want to cover, a few messages I want to mention is that the biology underlying the condition, the young onset Parkinson's disease is absolutely, totally different than what we see in late onset Parkinson's disease. So then why we call it Parkinson's disease when it's a different condition?

Well, you don't have better term, I would say, not so far. And in the past, there was this tendency to put together to clump together this condition, as one. We know now that we are not talking about one single disease.

We're talking about a number. A number of diseases that are different, our biology that are ending, ending we got the degeneration, with the death of some of the neurons, of the brain, but their final outcome is caused by a number of different problems, not by the same problems. So that's why talking about that first a new clean, talking about all the positive everybody's in the brain in somebody in his 50s and 40s.

I don't think it's going to be a good idea because it doesn't represent the real truth here from a biological standpoint, what we know now is that for a reason that I get quite unclear.

There is a group of cells, the cells of the basal ganglia, the famous cells of the basal ganglia, So the nuclei of the caudate, the putamen, nuclei that are indeed involving Parkinson's disease, for a reason that we don't know. In the younger individuals can be affected earlier on by a number of other biochemical pathway that are causing the damage that is equally similar to what we see in late onset, but it's different.

What is different about it? So late onset, we do have an accumulation, a lifelong accumulation of alpha synuclein, something that starts like in our 20s. We know symptoms are minimal symptoms, and with time, it grows, it grows, it affects that the bowel. It affects the olfactory bulb so causing people to have hyposmia, it affects our tendency to smell, to have insomnia or having agitated dreams. And then, after 20, 30 years, 10 years of mild symptoms, then people are having tremor, people are having the rigidity people are falling, therefore the diagnosis of Parkinson's disease starts.

There will be totally fair, and right, if we are talking about Parkinson's disease in general. But we all know we have 136 participants, and I'm pretty positive, pretty positive that what we are, indeed, experiencing, as a group is not what I just told you, I don't think that many people experienced for many years, constipation, I don't think that people are experienced for many years dream enactment behavior disorder before the disease start. I don't think that people have hyposmia for a long time. What does it mean? It means that the biology is different. It means that right away, younger onset Parkinson's disease we have an immediate damage, that is faster, is somehow more precise, less slowing progressing. That affects specifically the nuclei. Specifically, those cells, any just one question I want to tell you, I see you, I don't see me talking, I don't know if the audience are seeing me talking or you.

Amy Montemarano:

Dr. Savica, I just want to jump in for a second. So, there are some questions being asked but I will take them from the chat.

Rodolfo Savica:

I want to make sure, I don't know if they see you or me when I talk.

Amy Montemarano:

I hope that they see you, they see you, and I don't want to interrupt you,

Rodolfo Savica:

Oh good. I see a little version of me, that's why I'm asking. But that is an important thing. A much more focused and much more precise damage to where the basal ganglia. Coming, as I mentioned from different sources. What are the different sources? I think that this is a moment to understand that we are dealing with multiple disease, even young onset Parkinson's disease.

I'm pretty sure that what we just saw what we just heard may apply to some but not to all what we just saw what we just heard is different in all our 136 participants, and everyone, as they need, and we had the necessity biologically, and in terms of treatment to understand the differences and to customize the treatment, make sure that we are dealing with a unique pathology. I want to mention and then we're going to talk more about some of the questions and totally happy with that but there's couple of things.

Nowadays, every patient with young onset Parkinson's disease that comes to my door has been heavily tested to look for what they call mimickers. So, conditions that are called Parkinson's disease or Parkinsonism because this is what we call it. Why, because the symptoms are tremor, rigidity, stiffness, falls, but they are inherently different, because they have a different biology, I give an example.

A very common condition is a disease, Gaucher disease is that pediatric, so pediatric condition that seems to be affecting more Ashkenazi, Jewish people, causing a lot of GI problems, but we know that if a patient is carrying one gene not two, one defective copy, so if you have two copies of defective gene, then you have Gaucher disease, but if you have only one copy it may be another another gene that is not yet clear, a variant, what we call a variant of uncertain significance people may have Parkinson's disease, people may look like Parkinson's disease. People may have a metabolic condition characterized by an increase level, of one group of proteins that are belonging to the class of lysosomes causing the damage, similar to Parkinson's disease. Why this is important. Well give an example, there's some ongoing trial, about potential medications that are available in Europe already over the counter that can potentially block these accumulation

of these proteins. I am working heavily on that with some colleagues from Israel. But unfortunately, couple of things happen, other than coronavirus.

As you may know, just a couple of weeks ago in Israel, there was some sort of work and sort of attacks or this delayed many things with my colleagues in Israel but guess what I'm working with the pediatric people I not working with adult neurologists because they are not aware of that. I give an example if my patients coming back with this result, then I had to involve a pediatrician, one of my colleagues, which is an expert on this particular pediatric field.

Pompei disease, Pompei disease is an incredibly rare condition. But it's, again, caused by an accumulation of some proteins that are membrane proteins that are important for the neurons well-being. But yet to search for that why? Because if somebody has Pompei disease, if somebody has Gaucher disease, if somebody has some other condition that are rare mimickers, rare conditions affecting the kids that then can linger in the adults, they may not respond to the medication.

They may not absorb the medication, and therefore the delivery of carbidopa, levodopa, whether you do immediate release control release inhaler or whatever is completely impaired. And that is a problem.

In terms of customization, my patients are coming here and we have to study their biology, extremely different, each single individual. But then one thing that you do almost everyone. We end up looking at the genes. Not because genes are the genetic background, it is the answer to all our questions, because sometimes, many times, some genetic defect., genetic mutation can explain some paradoxical response of the treatment, and it's very important. I give an example. I was doing before giving slow increments of carbidopa/levodopa which is always what we do, whether it's a quarter, a half but there is some genetic mutation, which is a more benign Parkinson's and so something that doesn't progress as fast, very small doses of carbidopa/levodopa can cause a lot of problems, can cause dyskinesia, can cause dystonia, can cause a paradoxical response, way more than what we see in life, way more than what we see in our patients so the entire idea of understanding their biology, one individual is this.

I need to be knowing what I'm dealing with. And what I'm dealing with personally is different than what the other patient next door will work with. I'll give an example also is important, Ms. Quinn, was very good at talking about the young onset patient, I always say to my patients once you're a young onset patient, you're always a young onset patient. In other words, you would be able to be different than the older individuals, but obviously there's age, age comes along.

So if somebody started with a Parkinson's disease diagnosis in his 40s. Then his 70s or 80s, and still as the condition it is aging ongoing. But, for example I recently seen an individual that has been having that these mutation part two for about 20 years. The disease has not progressed but every time I give a smidge of carbidopa/levodopa there's massive reaction. So then we have a problem to work to do, but the entire goal is to really focus on customization of treatment, prognosis and medical management.

The last few years, I tried to dedicate myself to develop a different idea regarding young onset Parkinson's disease the idea is to have a complete holistic approach that goes from medication from prognosis for sexual dysfunction from social aspects for exercise and go on and we're going to talk more about some of these aspects because those are part of our life, and it's very relevant that we do that.

One thing I want to mention that is also quite important for a biology standpoint. One of the big original sins, let me pass a biblical term of young onset Parkinson's disease is to think that people that have young onset Parkinson's disease, have a strong family history of Parkinson disease. It's not the case. It is possible. I'm sure many of our people that are in the audience here, do not have anybody in the family with Parkinson disease, despite having young onset Parkinson's disease.

And this sin, this original sin, came to the fact that the first studies looking at young onset Parkinson disease were looking at young individuals that were part of large families. And this prompted to identify about 26, 27 genes that are the most common genes of Parkinson's disease, that are indeed relevant for the prognosis, and they can tell us more about it, but these 26 genes are showing only are really partial part of the truth.

They show only the group of patients that are positive are the ones that can have, maybe some sort of family history or maybe they don't know they have, this is an important point because the vast majority of patients do not have a strong family history.

So when we look at our genes we are looking again at a very small portion of the truth, and I keep saying that, and it's an important theme.

Amy Montemarano:

Dr. Savica:

Can I ask you a question, that sort of frames up what you're talking about.?

Rodolfo Savica:

Sure.

Amy Montemarano:

I know I've heard you say before it's difficult to define what young onset is versus what early onset is. Can you explain that a little bit more?

Rodolfo Savica:

That's right, yeah. Okay, okay. No, I'm joking about it. But he's an important point. One of the first purposes of the task force that I am honored to be the chair of is indeed to have a clear-cut definition of the age.

And this is a problem. This is a problem because age is different, that consideration of early, young or not, is something that depends a lot in terms of culture, as cultural differences, country differences, I always give the example and some of you have already heard about this example, in Australia 65 is the age considered young onset just because it's the age of retirement.

I don't know about that, you know, it's not necessarily the truth there. I think that currently young and early onset are considered synonyms. I say, early onset, when we are talking about something that is earlier than what we are expecting in the population.

The age of Parkinson's disease in the population, the median age is about 65 to 70 years old, more or less in terms of onset of the motor symptoms of Parkinson's disease. So, everything below before that can be considered earlier onset. Nowadays, the idea and the nomenclature seem to be moving more toward the idea of using the term young onset, which I like. I was not convinced about that, but with time I convinced myself it's the best term, young onset, and the idea is to use more or less 55, in terms of age of onset before 55, but then you say what about somebody 57, 60, where we going with that?

Well, they are still considered early onset to some extent, but we don't know. In terms of biology, are we dealing with somebody with an earlier biology or with a later biology? That is an important point because, as I told you before, this changes completely the way we treat the condition.

And that is a, that is an important thing. A very important thing, because that is a major fight. I'm serious about it, that's why I say, let's get ready to fight because that is something that constantly, and I'm losing years of my life trying to put together, try to put together with my colleagues and not only my colleagues, but also reach out to some patients, and patient groups, and everybody has different ideas, but why is it important to define? It's important to define for research, but it's important to define also for legislation to advocate, right? If, let's say, the Davis Phinney Foundation wants to go in front of the Congress to advocate more resources for young onset Parkinson disease.

The first thing that a congressman would ask is, okay what is the age we're talking about. And if you're vague about that that is not going to be good. So, that seems to be a silly dispute but it's an important issue because there are biological and also practical very practical implications. That's an important thing. I hope I answered the question but it's not very easy to answer this question.

I have some other questions here that are actually very, very nice. And I think I can cover some of them as I speak a little bit. For example, somebody saying, in Canada young onset is under the age of 50.

So, you see that is not stable across, it's not defined across the different countries, even in North America. If you ask 10 colleagues, as somebody said before, so called movement disorder experts, and somebody said, can you give me the definition of young onset, you get 10 different answers. Trust me, you get 10 different answers so it's something that we are required to adjust and we are trying also to educate.

I don't want to drift a little but it's important, education, teaching residents, teaching future movement disorder experts that we are not dealing with Parkinson's disease, our patients in the 40s and 50s, they're in top of their life. We are not dealing with patients, with all due respect in their 80s, or 90s, again, with all due respect, we need to deal with these patients as well. But there are some social, some situations, some issues, kids are young, you are still working. If we do not educate that there's a difference there and if I don't start doing that, we don't start to do that, there's a disaster. That's what I like about your Foundation because it's trying to do that and it's a very important thing.

One thing I want to tell you. Let's move because I'm concerned I'm gonna run out of time, I want to leave some time, I want to answer some questions.

Okay, let's say you see your physician, whether he's a so-called movement disorder expert or a neurologist or whoever, you receive a notice of Parkinson disease, what to do, what to do next.

Okay, make sure that all the mimickers, you need all the tests, all the scans, all the genes. Everything is pointing to a young onset Parkinson's disease, maybe you're 48. What do you need to do? Well, I need you to treat you. We need to make sure that you're good now. I really like what Ms. Quinn said, I wish I had started medication earlier. I wish I didn't defer anything for later. That's important. I always the analogy with diabetes.

If somebody has diabetes and their glucose is too high, we can try as much oral agents we want, but ultimately, we need insulin. Our insulin here is Levodopa. And there's a point that we need to use it, because otherwise people, as mentioned before, cannot exercise correctly. People can be slower, people can suffer. And we are not talking about movement, sometimes, levodopa is actually for mood.

Many times, it is used to increase the mood, increase the memory. As many patients say, I have brain fog. Why? Because the dopaminergic system is not yet simulated correctly. So first thing is to find a treatment that works for yourself. Whether it's a quarter of a tablet, whether it's three tablets, whether it's a different drug. It's okay. You have to partner closely with your physician, and we need to tell your physician that the patients know the body better than anybody else. I don't know your body, especially if you know about, you know, dancing or martial arts or you're very well aware of your body, or you're an athlete, you know your body better than anybody else so you can tell your physician, what is your requirement, your daily requirement of medication, that can vary according to the days, and according to their physical needs. What I mean with that.

I want my patients, all to exercise, all of them, but clearly if you have somebody in your 40s and 50s, I want them to exercise regularly. I want them to go, they want to do a boxing class? Good. Go to a regular boxing class.

I have some people doing grappling, wrestling, go to regular wrestling class, but then you say, you know, they're saying that the problem is that when I go to this class, I am feeling good, but not necessarily immediately. Sometimes, I feel that I need more Sinemet. Okay, before class, on that particular day you go to class, you take a little bit more.

And we customize, we tailor the treatment according to your individual needs. But then you say okay, but what about dyskinesia, I'm terribly worried about dyskinesia and you're right. There are some group of patients that have some specific mutations, particular genetic characteristics that make them more at risk of dyskinesia, compared to others. Our current studies shows that 52% of patients at eight years, with early onset Parkinson's disease, defined before the age of 65, they experience some dyskinesia.

Some of them is really modest, some of them are having severe dyskinesia, but this means that 48% are not developing that. 52 are, and this means that dyskinesia as shown before requires some adjustments, requires some clear ownership from the doctor and from the patient to adjust according to the needs, and according to what we can do. But people can tell you, I have many patients that prefer to be dyskinetic than be unable to move and this is a true story. It's something that we are required to find a balance according to the need. So this can be considered a very severe and bothersome symptom is not necessarily something that we cannot battle and we can work together to improve it in the vast majority of cases, that is an important thing and it's true. The more medication you need, the dyskinesia there's a risk of. Sometimes even small doses can cause that according to the individual needs of the patient. And maybe the characteristics of the patient.

One thing I want to mention, because the other part of my talk was living well, so treatment, as we know, we have one gold standard medication, which is carbidopa/levodopa, we have the agonists, we have other medications that are used at times, but the gold standard is the carbidopa/levodopa.

What about deep brain stimulation? Is deep brain stimulation the last beach, the last resort? Not at all. Deep brain stimulation is a procedure that can be done, needs to be done when it's needed.

Sometimes it can be very early in the disease, sometimes it can be later in the disease, but it's done when it's needed, it's not a last resort, and it's important to see, what can I achieve with deep brain stimulation. I have a little tremor, fantastic, that works for you. I have a little tremor, I am stiff and rigid, will this work for me? Maybe not as much, but I'm generalizing here, but it's important to consider deep brain stimulation as a possible tool that we have, but it's not the last resort.

And it's an important thing to consider, because that is what some of my colleagues wrongfully are thinking still. And it's coming again, to try to translate information from a different disease into young onset Parkinson's disease.

Another big thing about living well, so exercise is crucial. I always tell my patients, pleasure. Pleasure doesn't mean doing exotic things, it means carve time for yourself. Pleasure also is part of having a fulfilled life, which I like.

I heard even, Mr. Phinney say, please live your life at your best, and it's exactly what we need to be doing. This condition has a social stigma that we are still working on, and it is tough to remove, but it's a condition that once there, nobody would like to have it, but we have to face it, to have a full life. This means, please be vocal with your physician, be vocal with your friends. If the antidepressant that you're taking is causing you to have urinary retention, or is causing to have erectile dysfunction, tell them right away. Those are examples of practical things that you can see.

If you see, and if your physician doesn't want to change it, or blows you off, change physicians. I'm sorry to say that but it's the way to be doing it at times, if you don't find a good connection or somebody that is willing to work with you, customizing your treatment, that is not the right choice. Okay, that is not the right choice whatsoever.

One thing that I want to mention, I'm very big into supplements and diet. Supplements need to be cheap, need to be making sense. All my patients, as you may have heard, many times, are using folate, B6 and B12, all of them. And all of them are eating more flavonoids.

So the little chemicals that are present in the skin in purple and blue vegetables and fruits. What about all these very expensive supplements that you find online that are claiming this or death effect, are these detrimental? Not necessarily, are they detrimental for your pocket? Absolutely. They're cost a little money.

Do we have strong evidence that this would work? Not necessarily. If I don't have that strong guidance that something would work, I don't give to my patient, because I am not happy about giving something just to try. That is for sure. And supplements, there's so many. One thing I want to tell you right away. And please bear with me and then I'm going to answer some of these questions, is what about natural carbidopa/levodopa? I don't take Sinemet, I am anti-drug. It's a normal thing I hear. I am anti-drug and I guess anti-pharma, I don't want to use it, I don't want to take drugs, but I've found this particular thing called mucuna pruriens that can use instead of carbidopa/levodopa. Is it wrong? No, it is not wrong, but it's not right.

So, what is the problem? Mucuna pruriens is what we use to extract dopamine, to put it in the legal norm. But every plant, every single plant of mucuna pruriens has a different amount of dopamine.

Sometimes you get 50, sometimes you get 70, sometimes you get 25, so we don't have a clear dose that we can use. In addition, what is carbidopa? Carbidopa is used to kill the nausea, block nausea. Mucuna pruriens doesn't have that.

So there is an increased risk of having people really vomiting, and having major puking if they use that. It is used in developing countries. It is used sometimes in Africa, where there's not access to the drug, and I'm okay with that.

But I don't see a good reason why using it here when we can have a pure and most purified and as you hear before we need to customize little doses, sometimes a little change of the dose can make a world of a difference.

So, I usually suggest my patient not to go that route. Some people are doing it, I don't recommend that, I would say.

Now, if it's okay with you, maybe for the last 15 minutes we can, more or less, we can, we can answer some questions. Some of them are very interesting actually.

Melani Dizon:

Right. Okay, so I'm going to ask this question I thought was super fascinating, "Research suggests reducing the amount of alpha synuclein in the system is a possibility of slowing the progress of Parkinson's much as the forthcoming someday repurposed meds will do. Is there a possibility of needing to have a certain stage of Parkinson's to have them work?"

Rodolfo Savica:

Man, what a great question. I really appreciate this question, it's great. So, there's a couple of things to talk about here, number one, it's true. It is possible that reducing alpha synuclein can improve the condition in patients that have an excessive amount of alpha synuclein. But if, as I mentioned before, you have a condition and maybe when alpha synuclein is not the major driving force, even if you remove that it is not going to change too much, number one. Number two, the dogma, it is a dogma, that is the same as Alzheimer's disease, it accumulates above normal proteins, and cause the damage. I remove the normal protein and things should be working better. That is a dogma, and it's something that maybe it's not working all the time, and I always say this, imagine alpha synuclein as the trash bins, that you have in your house.

So, when you produce trash you put in the trash bin, and then every week, there's a trash company, usually every week here, maybe somewhere it's maybe less than a week but every, every week, they come, the big truck removing the trash.

And this is what our brain does all of our life. We have a scavenger mechanism that is removing alpha synuclein. What if at one point, you are having a drug that kills alpha synuclein, what is

going to be the trash. The trash will be all over your house. And maybe, the other condition where the truck company comes every three weeks with a smaller truck, so what happens. The junk that alpha synuclein is not able to contain is accumulating all over the house, causing more problems. It's an important thing to consider that, I don't think, and there's many ads that report that, that removing alpha synuclein would work in everybody.

As usual, our effort, is to work on identifying who and what works for myself, and not for Melani, and not for Amy, and so forth. That is an important thing. It can work, totally, I agree with the fact that it depends on the stage, clearly, all the neuro protective trials that have been done so far have failed. Why? Because likely the condition was to advance, the condition was already present when there was what, a motor onset.

But if we talk about regular Parkinson's disease. We know, and I told you before the disease can start 10, 15 years prior to that. So, we want to work at the very beginning at the inception of the disease for neuroprotection, not when it's already happening for a few years, that is a problem.

And I agree. There's a number of drugs that are used for a different condition, some anti diabetes drugs, anti-pressure drugs, uric acid that are showing some biological potential, in treating Parkinson disease in mice. And sometimes epidemiological, you see some association, but depends a lot on the stage.

One thing that I want to say that is very important. I've seen a number of cases nowadays people that are reached out to me with positive immuno stain of skin, so they go and do the skin biopsy, searching for alpha synuclein, and if it's positive clearly, they're worried, even without symptoms.

And they say, okay, do I have a disease? And the answer is no. I mean you might have the positive alpha synuclein in your skin, but that doesn't mean that you have a deposit in your brain.

That is not a surrogate marker of the brain progression. And that is, answering this question, we do not have currently a surrogate marker that can tell us what is the stage of the disease.

And this an important thing because if you have nothing in vivo that can tell us, we cannot put the disease at the inception, we can just see and try drugs, as things are started. It's a very complex phenomenon, but it's very important I would say.

Melani Dizon:

Okay, this is a great question and I have a feeling that a lot of people are probably having it as well. It says, she says, and actually Pam Quinn says this, I have always taken my drugs in

relation to what my day looks like when I need to be ON. My doctor doesn't like this because I'm not taking my medicine at the exact same time each day. What is your opinion on that?

Rodolfo Savica:

Doesn't matter. It doesn't matter. So, the answer is it doesn't matter, you require more dopamine, and some days you need to take more medication that day. What is the problem, especially later onset Parkinson's disease but also in young onset Parkinson's, so why doctors are insisting on taking medication at the same time?

Number one, because of the food interaction, you know, we know that the medication, I heard before, was 30 minutes before meals. I always say one hour before meals just to play it safe, or two hours after meals, that's my recommendation, one hour before or two hours after. The problem is that at times some people are having the wearing off of the drug.

So therefore, you want to have a predictable kind of response, let's say, taking medication at 8, Sinemet at 8 and they know that every day by 11:30 my medication drops down, so I will take it again at 11:30 and avoid the drop. It's a valid point, that's why people want to take it every single time. But, as I mentioned before, let's say Mrs. Quinn. She has to teach classes that day, she has like four classes one after the other.

She needs more. And it's okay to take more, and it's okay to take the relationship of what you need as long as you don't present wearing OFF, as long as you may have a predictable response, guys. To me, the analogy with insulin is the same.

If you are having diabetes type one and you work out and you need more insulin. There's little to say, and this varies according to the day. I hope that one day we will have the ability to measure, live as things are needed. The requirement of dopamine, as it happens with diabetes, like with the pumps that allows you to fix the amount of insulin that you need, but we are not yet there.

We are not yet there.

Amy Montemarano:

I think I probably speak for several people on this call who are kind of jealous to hear about your individualized treatment, because a lot of us have neurologist who don't do that. And one of the questions in the chat is, how do we access this kind of individualized treatment in the US? Do you have any advice?

Rodolfo Savica:

Absolutely. I'm sorry, this is a problem, there is a problem, and I am in contact with some patients, it has to do with education, has to do with training, it has to do with thinking outside the box and sharing the frustration that we are in 2021, and we don't have a cure for this



disease, and it's insane to me, that we are still banging our head about dopamine and about alpha synuclein and we don't know even what is working for and then we know that is not the case and so forth.

How do we have access to that? Well, I am not I don't like to use that advertisement at all. But at the Mayo Clinic, we do have these opportunities, and we are accepting more patients currently. So, if you contact us, we will not more than happy to put you in contact with these people. We know there are two people, me, basically it's me. There are two centers, unfortunately, as far as I'm aware, so maybe there's more, but two in the globe. In the world that are dealing specifically with this approach us, Mayo Clinic, and Nijmegenin in the Netherlands.

And it's interesting because these two centers are really working together we are trying to organize our efforts in order to keep things, at bay. Don't get me wrong. There are centers all over the US with people that are absolutely expert and they are great in dealing with this condition. But the idea is to try to have a larger view of the patient not just adjusting the medication but going a little bit beyond that. That is, to me, a very important and relevant aspect. And to me, what we should do when I say we, I mean myself, but I'm one voice.

Despite, I have a big institution behind me, I am still one voice. We need to advocate and allocate. So, advocate for more resources to allocate for young onset Parkinson's disease. And this has to be advocated, you know with Michael J Fox foundation, even with Davis Phinney, what you guys are doing, but then with NIH, the government to promote and develop training opportunities to increase the number of centers that want to increase their knowledge, and want to work closely, that's important thing, closely with a patient about what to do, or what not to do.

Because, the most important thing somebody say, oh what drugs to avoid, yeah there are things that you don't want to do. There are things that you want to do, and things that you don't want to do, but we're in an era when there's a sea of information online.

And you can lose yourself, you can lose yourself in the sea. So we have to make sure that we have a very good system in place, and a good networking, I heard before, Mr. Phinney was mentioning that the pandemic has been tough for all of us and I agree. It's been tough on everybody.

But has allowed people to accelerate their virtual networking. And since we are talking about a rare disease. Zoom meetings Facebook pages can be a great source of opportunity to build up a network that can help support each other, as Mrs. Quinn said and I keep on citing her was, I would say right away, I would try to meet more people and have more friends. It's up to you if you want to say you have a disease or not, I am not going to tell you what to do or not, but for sure, if you have opportunity to connect that helps. If you know oh there's the center there that

can help you with that. That helps. But it's very frustrating to me, that 2021, we are still struggling to try to allocate resources and and try to see anything beyond our own nose. In terms of treating patients with young onset Parkinson's disease.

Amy Montemarano:

We have a number of questions in the chat that were asking about light therapy and your opinion on that.

Rodolfo Savica:

Well, that is coming from Australia for the most part, so that was actually I don't remember if it was, I do apologize, I don't remember if it was Australia or New Zealand.

There are some, the judge is still out there, the jury's still out there, it is not yet judged positive or negative, clearly it doesn't seem to be detrimental. So the most important thing is that is, is it harmful? No. Doesn't look like it. Is it helpful? That would be the second question, and the answer is that we are not sure, do I have things against that? Not necessarily, as long as it doesn't cause very much damage, that's my only problem, but the only problem is that the light therapy depends a lot on the penetration.

So, anything depends on penetration of the skull. There's a coefficient of penetration that depends on the thickness of each other's skulls. And unfortunately, there are some people that have a thicker head. Therefore, nothing penetrates, even a magnetic, you know the magnetic stimulation will not work because it doesn't penetrate the thickness of the skull.

So there is a coefficient of thickness that is very hard to to predict, just by doing that, to the point that once, I'm talking about two years ago I was asked to be part of the scientific community to review one of these possible light therapy, one of the things that I requested was an MRI. You need to have an MRI or a CT scan that does tell us how thick is your skull, because if your skull is too thick there's no reason to do that because he will not penetrate.

So there's some practical problem but if from a detrimental standpoint it's not detrimental doesn't cause me you or anybody any particular issues right there.

Melani Dizon:

Dr. Savica, I have a question about exercise, and this was something I remember you told me a long time ago I thought was super interesting and really want to share it with this community. Can you talk a little bit about how you say, how you've said in the past, for you know, YOPD, young onset, earlier onset, go to regular exercise classes if you can and then can you talk a little bit about, you know the difference like maybe you won't tell your older patients?

Rodolfo Savica:

Correct. This is an excellent point. Thanks. Thanks a lot Mel. So if somebody, again, I'll give a specific example, if somebody is 43, 44 45, 50, fit, always work out, I don't feel that sending them to boxing class for Parkinson's disease will be of any help.

Number one, the intensive training, it's not going to be the same. Number two, psychologically you will see somebody that is maybe in their 90s, that can be or you feel that that going to be your outcome, but it's not.

So, psychologically that is wrong to me. Third, if you been always exercising but even if you haven't been exercising. What do you need to be doing, you need to test with people your age. You have a condition that limits somehow your movement. But still, you have the same age as somebody 40, 50, so forth. So always, please go to regular class, let's say, I will disclose too, who cares, I am a martial artist, I trained martial arts I teach martial arts, I spend hours in the gym. And I tell many of my patients, okay, going to Brazilian Jiu Jitsu go to grappling, go to MMA, go to boxing. Tell the instructor, hey I am a person with Parkinson disease, so I may not be able to get at the level, maybe. But your goal is to go to that level you got to get that dopamine level higher, and to do that you need to go to regular classes.

You can go to a Parkinson's class but I say, if you work out all your life, if you're fit, if you run marathons, if you're a cyclist, you need to measure yourself with people at the same age that you at, even if you have Parkinson's disease, that is crucial. If somebody is in their 80s, it is a different story. I say then go to a Parkinson's one, because sometimes there may be other problems. Let's say in your 80s you may have problems with your knees, that will not allow you to work and move very fast. So something beyond Parkinson's that can contribute to Parkinson's.

What we are doing, we are really trying to develop, and I'm really trying to develop something specific for young onset, but, again, nothing gets to be specific, it has to be regular classes. It's phenomenal to do that because we need for people to go to a higher level of requirement. To give an example, I follow a patient for now 15 years, young onset patient, he was complaining sometimes ago because he was 20 seconds behind these his friends, as he was cycling in Utah. This guy was 53. I said, sorry, okay 20 seconds. But you know, if I was sending him to our regular to our Parkinson cyclists class, he wouldn't feel good about it.

I prefer for him to be a little bit frustrated about missing few seconds, 30 seconds is a lot in cycling, I know that, don't get me wrong, but I would prefer that and you challenging yourself to a regular activity than going to a different activity. Make sense?

Amy Montemarano:

Yeah, thank you. We have about three minutes left, I want to ask a question that also has come up in the chat and that is your thoughts on neuro plasticity.

Rodolfo Savica:

Oh yeah. Well, neuroplasticity is a big word, it's a big word. What is neuroplasticity? It is the ability of the brain to recover, to change, to change characteristics. We are constantly undergoing the neuro plastic changes throughout our life.

If, if you guys remember Clockwork Orange. If I have you doing the clockwork orange treatment for sure, for 10 years, for sure your brain will be wired differently because I'm forcing use and specific information. I'm, I'm exaggerating but I'm telling that everything that we do every day changes brain. Clearly when we were younger kids, we have a sprouting of millions of neurons and information as we get older, things are different, and not as good, not as fast.

The entire goal of working on neuroplasticity is to try to find activities or drugs, or treatment that can help rewiring, re-changing the configuration of receptors and neurons in the brain. So far, so far, the only activity that seemed to have a neuro plastic role that seems to be working, is unfortunately exercise.

So, what about reading a book? What about doing one of these tests online? What about going to a play well, now that we can go back to plays and theater? Is this helpful? Absolutely helpful. It is part of the other aspect that I work with, pleasure. Do something that is good for you, because this positive, this good feeling, these fun things help your brain to recover.

Later, we're going to talk about music. Some people love music, some others hate music. So, find your activity and try to work on that. But are there medication or things that we can do to improve neuroplasticity. Other than exercise in this moment, we don't have anything pointing that direction.

In the recent past, there were some trials, working for example, on stimulating the stem cells, not stem cells, stimulating the available stem cells that have not yet work very well, but, as usual, we are talking about late onset Parkinson's disease.

We're not talking about young onset, we cannot translate the knowledge of that time into our time. That is very important. And it's the major source of frustration for many of us I would say.

Amy Montemarano:

Thank you so much. That was so helpful and valuable information. And so much of it was concrete, you know, we hear a lot about the research we hear a lot about studies. We hear a lot about theories, but some, much, if not most of your advice was really concrete and that's really, really helpful because we have come away with a lot of action steps that we can take.

Rodolfo Savica:

Thanks for inviting me again.



Want to Watch a Recording of the Webinar?

You can access the recording [here](#).

Want More Practical Articles Like This?

Much more can be found in the Every Victory Counts® manual. It's packed with up-to-date information about everything Parkinson's, plus an expanded worksheets and resources section to help you put what you've learned into action.

[Request Your Manual Now](#)

Thank you to our Peak Partners, Amneal, Adamas, Sunovion, and Kyowa Kirin, for making it possible for us to print, distribute, and ship the Every Victory Counts manual.