Introduction and the Mitochondria Connection

Oh, welcome, Dr. Perlmutter. I'm so glad to have you as part of the Summit. I really valued our friendship. And to everyone who's listening. David Perlmutter is a neurologist, one of the earliest functional medicine.

Neurologists really understood that this integrative functional medicine approach could lead to remarkable stability and regression of disability in people with, profound neurologic diseases.

That up to that point, we figured could not be stabilized or reversed. and so with that, David, I will let you expand on your intro. in case you feel like I've left anything out.

That's a great place to start, but I'm already the wheels are already turning. And, you know, you mentioned just kind of the broad field of neuroimaging under conditions.

And we are in an exciting time because we're now recognizing that upstream of what we used to think were the targets, the unique targets in Parkinson's and MS.

and Alzheimer's and other neurodegenerative conditions. They each have their own uniqueness that have been the targets. Certainly in the world of pharmaceutical development.

But we're in an exciting time that we're recognizing that upstream of all those seemingly disparate entities, in terms of pathogenesis, ultimately looks like it's going to be metabolic.

And and upstream of that, it turns out to be problems with respect to how the mitochondria work. So, you know, the notion that we can leverage this information to create programs that benefit the brain across the board, is really quite exciting because, you know, we live in a world of specialization and a specialized approach to MS and Alzheimer's, etc.

to think that we can unify our approaches to building a better brain to help it recover from whatever may be fall. It, I think is really very exciting.

So let's talk more about that mitochondria. as the big driver for neurodegeneration, and I see that as the big driver for disability accumulation for people with MS.

and neuroimmune conditions. what's your point on that? Well, you know, we all grew up in the world of of mitochondria being the energy factories of the cell and all.

Well and good. You know, we all had to memorize, you know, how the atps are produced and all of that. But, you know, it turns out that the mitochondria are really much more deeply involved in, so many processes within the brain, throughout the body.

But in this discussion within the brain that are really exceedingly relevant as it relates to neurodegenerative conditions. We know, for example, that, ultimately the the cells that are involved in maintaining the synapses, the connection between one cell and another, that that this is a focus, in Alzheimer's, that the synapses begin to degenerate.

And so there's loss of cell to cell communication. Similar pathways are involved in the maintenance of myelin, the protective covering sheath over neurons that tends to be digested away in MS.

And it turns out that mitochondria, through their regulation of the function of various cells, cells that are involved in the digestion of a male, and even those that are involved in the maintenance of myelin, that mitochondria are

involved in their functionality day to day, minute by minute, if you will.

How Mitochondrial Dysfunction Drives Neurodegeneration

even in Parkinson's, we recognize we've known for a long time that primarily in those cells, in a very specific part of the brain, the pars compact of the substantia nigra, that makes dopamine that for whatever reason, these cells degenerate as a consequence of mitochondrial dysfunction.

So, you know, as you're well aware, decades ago we began seeing research focused on how can we, in Parkinson's, improve mitochondrial function with an eye on helping people, with with Parkinson's.

And indeed, there were interventional trials with coenzyme Q10, etc.. So really pretty much across the board, we're looking now at mitochondria through a much, much different lens, well beyond cellular energetics.

But understanding that those cellular energetics within immune cells can become dysfunctional as a consequence of the mitochondria not working up to speed, and therefore it could change how the immune cells are able to work.

And these immune cells are involved in so much more than just what we classically understand as immunity. These immune cells are involved in the maintenance of neurons, the maintenance of myelin, the degradation of beta amyloid, for example, and all manner of saylor metabolism and homeostasis.

So it's this mitochondrial, centric, if you will, idea that we're now deeply involved in exploring as are you as you know, really would well explain the walls protocol, as you well know.

I mean, you know, one of the powerful ways to target my mitochondrial function is dietary and really exactly what you, over the years, have outlined for all of us.

So it's really finally become a central collecting point for us to galvanize our efforts that have wide ranging implications across the spectrum of neurodegenerative conditions.

okay. So if we're going to, better support, mitochondria, what is the approach that you would use in your clinical practice? in that you suggest to your followers?

Turns out that, pharmaceutical companies are now deeply investigating what can be done to target mitochondrial function. There's nothing really yet available that's safe and effective.

So we still can leverage lifestyle issues and certainly nutrition, nutritional issues and certain supplements that have been clinically demonstrated in humans to improve mitochondrial function.

We know, for example, that plain old physical exercise is a great way to increase mitochondrial function by targeting something called my top veggie. And what that is, is many of your viewers, I think, have heard of autophagy, where this is a process in the body.

Did I say process? I've been spending too much time in Canada, a process by which, the body is able to rid itself of damaged cellular components, things within the cell that are damaged.

We get rid of those components and replace those with healthy components. Specifically as it relates to the mitochondria, we call it Mitophagy, where phagy means eat.

We basically have a process by which we're able to get rid of Mitophagy by acting, activating certain pathways parkin pathways and and pink pathway. Those are those are some of the names.

But nonetheless how do we do that. How do we activate the digestion or cleaning out the cell of its damaged mitochondria? We do that, through our diets, through being in ketosis, through caloric restriction, through, as I mentioned, physical exercise, one, supplement that's been demonstrated more recently to, to enhance that process

of my top is called your olefin.

A so there are a variety of things available right now. And, you know, certainly physical exercise, caloric restriction, being in a key to, chaotic state are, well, available to anyone.

and this then targets mitochondria in such a way that we digest away the damaged mitochondria. And guess what? we instigate the production of new, very healthy, very functional mitochondria.

And we call that process mitochondrial biogenesis. And this is really what we want to be doing again, across the spectrum of neurodegenerative conditions.

And let me say importantly, that before we really cross the line and then think we need to leverage this approach to help us through a neurodegenerative condition, this is the foundational recommendation of keeping the brain healthy today.

You know, John Kennedy said that the time to fix a roof is when the sun is shining. So this is really something that these ideas, these techniques, should be embraced by anyone to keep your brain healthy so that you are at lower risk for one of these situations and really to enhance the functionality of your brain, day to day.

So, I take it from those comments and of course, you and I both agree is the time for, people with MS.

Lifestyle Strategies to Improve Mitochondrial Health

in any kind of, neurologic disease is to begin attending to your mitochondria as soon as you're diagnosed, as soon as ALS, ideally even before, when you're in the Prodrome state.

Well, I would say that this is something everyone should do, whether they're going to be in MS. patient or not, or an Alzheimer's patient or Parkinson's or ALS patient or not, these situations are fundamentally what we call mitochondria spaces, failures of the mitochondria.

So, certainly if a person is at the in the prodromal stage of multiple sclerosis, then by all means this is going to be a powerful lever to pull at that point.

But my call, you know, more recently at this stage in my life is really more in terms of prevention and keeping the brain healthy and helping, individuals develop lifestyle protocols to resist, these types of problems in the first place.

You know, I think that could take us to, the families who have someone, diagnosed with, MS. or clinical syndrome or any of the systemic autoimmune diseases with neurologic psychiatric symptoms, and they're struggling with, okay, I'm changing my diet, but I don't want to change the diet for my children.

what, what is your response to that kind of, response to your patient when you're talking about diet and lifestyle? I'm like, well, I can do it. It's, you know, I, oddly enough, you and I have had this conversation before, and, you know, it's tough.

I can recall one of our children, couldn't wait to go to birthday parties to eat the things that we wouldn't be eating at home. And, you know, I get it, but you can only do the very best you can to provide guidance.

but but certainly, you know what we are suggesting in terms of being now interventional with somebody who has, a prodrome for any of the issues or early stage for any of the issues we described.

it's certainly far more aggressive than some simple dietary changes. I mean, you know, going full on, ketosis or, intermittent fasting and certainly adding various types of supplements to the regimen is asking a lot.

And, you know, ultimately, ultimately, when do we begin this program? When should we start recommending that people eat a good diet? You know, and I think it's during infancy, for that matter.

And I also believe that, you know, what a mother, a mother, is consuming during pregnancy is extremely important. So I've often been asked, well, which the Alzheimer's prevention diet be started.

And we know that as it relates to Alzheimer's, for example, the hallmark changes in brain metabolism are discernible, in the in individuals 30s and 40s.

In other words, decades before people begin to notice cognitive dysfunction, it is only when the brain begins to fail and people begin to notice that they're unable to remember things, children's names, etc., or grandchildren's names, Wi-Fi code, you name it.

We call that subjective cognitive impairment because it doesn't yet test out, on neurosis testing as being cognitive impairment. But here you're beginning to notice that things aren't quite right.

That's when people begin to think about doing something. And I mentioned a quote from John Kennedy earlier the time to fix a roof is when the sun is shining.

When you are cognitively intact, long before you begin to fail, that's when things need to start being put in place. So as mentioned, we can see, changes in brain metabolism in a person's 30s and 40s that clearly represent the earliest evidence that Alzheimer's is, is going to rear its ugly head.

And these are metabolic changes. These are areas of the brain that begin to fail in terms of how they utilize glucose as a fuel. And that can even at that stage of the game, that can be remedied.

You can see that those areas are functional but not functioning. Put somebody on a ketogenic diet and re scan them using a different type of tracer. And you can see that these areas show function.

So you know, it's not like we're waiting for something to happen before we start instituting a plan. You know, the mission here is to really let people know that what you're doing for yourself, in your third and fourth decades and probably even earlier, is exceedingly relevant because you're going to be in your 60s and 70s before you know it.

And, you know, the, the most powerful risk factor for Alzheimer's is, you know, people say, oh, I just asked this at a conference I was at, just last week, and I heard, well, diabetes, obesity, you name it.

Now it's age, the one, you know, important risk factor over which you really don't have much control or any control calling it like it is. And the reality is, in going from age 55 to age 85, your risk of developing Alzheimer's increases 700 fold.

So you can affect that number dramatically, and you begin to, pull the levers to reduce that risk in your 30s and 40s. And my hope is that, you know, soon people are going to begin getting that message earlier in life.

You know, as as we're having this conversation about Parkinson's and Alzheimer's, you know, I'm thinking about the Ms. patients, you know, many of whom are diagnosed in their 20s and 40s, and they too, are probably having alterations in, in brain function and metabolism that I'm speculating is occurring, a decade earlier.

So in their teens probably that and then when I look at how, poor our diets are, how many of our young people have this explosion of anxiety and depression, social anxiety, learning disabilities?

and, I'm thinking that all of this is a program for a neuro immune diagnosis, and Ms.. in the, following decade. And I would completely agree with you, I think that, you know, now that we, I have humbled ourselves to recognize the incredibly powerful role that gut and mouse organisms are playing in terms of the control and balance of immune response, the control of our gene expression, the regulation of gene expression that bacteria are regulating the expression of our life code, our DNA, and really how, the things going on, the metabolic products produced by, the in the gut, by our bacteria are actually ultimately able to regulate neurotransmitters and even regulate mitochondrial function, throughout the body.

And that includes within the brain. So the, you know, the entree to our gut bacteria are the foods that we eat. And people think, well, I'm going to eat these foods because I like them.

And you don't need to really you do need to realize that, you know, we say when a woman is pregnant, that she's now got to be careful what she eats because she's eating for two.

Well, you know, every person walking this, face of the planet is eating for billions right now. So they're these organisms in your gut are going to eat what you eat.

Why Prevention Starts Early for Brain Health

And when you think about that, you begin to recognize that this is a powerful entree to either better or or dysfunctional health and primary, as you well described, it deals with immune regulation, directly through various metabolites made by these bacteria and slightly indirectly through the role that our gut bacteria play in managing to keep the gut lining intact such that various components within the gut, don't make their way into the systemic circulation and challenge our immune systems.

again. So everyone who's listening, this is why David and I are so passionate about what we are eating. This is, it's just such a powerful tool that we have that will either feed the correct bacteria.

so we have the correct metabolites made, or we feed the wrong bacteria in yeast and have the wrong metabolite to make. That's right. And and, you know, it's all about balance.

and, you know, over the years, I have to admit, I've, gotten sort of away from the notion of good and bad bacteria that, you know, some bacteria under certain circumstances do good things or in under other, contexts.

They are they are threatening. I mean, we have a lot of clostridium bacteria in the gut actually doing some good things as it relates to the mucin lining, of the gut.

so, it's really about context. And now we're at a, a stage that we are recognizing that to a significant degree, our gut bacteria are modulated in their functionality, moment to moment by viruses that live within the gut, that outnumber the bacteria 10 to 1.

You know, we talk about the huge number of bacteria outnumbering our own cells by whatever that factor is. But it turns out that bacteriophages, which are what these viruses are called, that are intimately involved in regulating bacterial function moment to moment, are incredibly numerous.

And you know how exciting it is to see new research showing improvement in memory in a rodent and a mouse, by increasing the presence of a particular virus, in its gut.

So, you know, what is next gen going to be? Next gen is going to be not just thinking about taking a bacterial probiotic, but with the addition of a bacteriophage or complement of bacteriophages, to modulate the expression not just of those bacteria in the product, but also the resident bacteria that are already that are already there.

You know, this is really a great conversation. We were just talking, in our research lab because we have a microbiologist, who's also an immunologist, and we've been analyzing our stool collections that we did in the Waves trial, comparing the swank in the walls diet.

and you're exactly right. It's not the individual bacteria. It is the species in community. It was. And we could identify different communities that would predict, those people who were more likely to respond and those people who were less likely to respond.

And, I think this was in response to what the community and what the viruses were. And, of course, what people were eating. Sure. And oh, hang on. Yeah, we had an early start, researchers over, you know, decades ago.

And there was the identification of various species that live in the gut. And, you know, now we are looking at subtle genetic differences within, various species.

And, even looking then at their, their genetic complements and beyond that, what their metabolites are. And that becomes a, an incredibly vast amount of data.

And then to be able to correlate that information, with physiology and pathophysiology is very daunting. But you know, the exciting is we have the ability now to crunch those numbers and, and try to begin to see patterns emerging.

You know, we're not going to be real specific. you know, at, species specific, recommendations for people because we don't yet know, know the context within the individual.

But when we have that and we can compare that with that individual's genome, for example, I think it's going to be revolutionary in terms of what we're able to do.

I mean, basically we're talking about factories within your body right now that are making powerful chemicals that are changing gene expression throughout your body.

My goodness, that is, you know, it's just uncanny to think about how where that might lead. Okay. Thank you so much for joining us today. I hope you found our conversation insightful, engaging.

And if you're a summit purchaser, stay right here. Because we're about to dive even deeper into this wonderful conversation. If you're not, click on the button below or on the side and get access to a world of information.

Get ready to reclaim your health. If you're watching this, thank you for being a valuable member of our community. David. Let's continue, this conversation about the bacteriophage, the viruses, and their impact on our health.

Sure. So, you know, interestingly, bacteriophages have been used in industry for decades, in terms of factories and farming as a way of getting rid of potentially pathogenic bacteria, for an awful long time.

they are what we call generally recognized as safe. and in fact, now our, we're starting to see them appear, in retail, in addition to probiotics. So I think that any kind of manipulation tool we can get our arms around in terms of changing bacterial expression and functionality, I think it's going to be very helpful.

You know, for many years we we mentioned how for a probiotic to be effective, it must be alive. And, gosh, we would have been deeply involved in that research.

And it turns out that just the covering over the bacteria has effects in terms of changing, the functionality of other resistant bacteria within the gut.

for better or worse. So, you know, there's a lot of change happening, and I think it's very exciting. I mean, you know, face it, a couple of decades ago just told we would never have had this discussion. The, bacteria were called germs.

And that's sort of a negative connotation. How incredible it is that, you know, within the mouth that there are bacteria that are resistant, Neisseria act in a mighty species, for example, that live on the top of the tongue, that are fundamentally important.

Ultimately in regulating human metabolism. Now, you might wonder, well, how does that work? Well, turns out that these bacteria are involved in converting nitrate that we consume in our foods.

Gut Microbiome, Diet, and Immune Regulation

Foods rich in nitrate are converted to nitrite by these bacteria, and that nitrate in the human body is the material

from which we make something called nitric oxide.

Nitric oxide is actually a gas that regulates, how our bodies are able to use insulin and how blood is able to flow to our organs. So what I've just connected, then, are the bacteria living in the mouth to blood glucose regulation and blood supply to the brain, for example, think about that through this connection through something called nitric oxide.

So, you know, I led into this conversation with the understanding of the good bacteria living within us versus characterizing them as germs. And what pinged me mentally to do that are these commercials on television that are trying to convince us that we need to eliminate 99.9% of the germs in our mouth by using this or that mouthwash.

And interestingly, we're seeing peer reviewed research showing that people using mouthwash two or more times daily may have as much as a 48% increased risk of becoming diabetic, and as much as a 100% increased risk of developing hypertension in comparison to those who do not.

Now that seems way out there, doesn't it? I mean, you know, this this is the same doctor promoted that brought you, gluten in the brain and then this whole notion of uric acid.

I don't make this stuff up. And when you understand the mechanisms by which, you know, these, issues are connected, it begins to make sense that the notion of killing off bacteria in your mouth day in and day out has consequence.

And it's all through the this pathway involved in something called nitric oxide. You know, I think it, just reinforces my expectation that we have this very, collaborative relationship with our bacteria in our mouth, in our gut, probably on our skin, in our, in the tears, in her eyes.

and when we disrupt that relationship, bad things happen. And you know, and our health declines. I want I want to circle back to fermented foods for a moment, because I know when I was reviewing the literature that people consuming sauerkraut and kimchi, the fermented cabbage families, have, healthier outcomes, right?

Less respiratory infections, less pneumonia, less ICU deaths, longer lives. And I'm beginning to think that this is tied in part to the bacteria, but also probably to the bacteriophages that are in those, fermented foods.

what are your thoughts? Well, I think there's a part three to this story, and I've not actually seen the study that compared cabbage consumption to fermented cabbage consumption.

So cabbage happens to be a pretty darn good prebiotic. So, aside from bacteria content, bacteriophage content, the fact that people are eating a lot of fiber rich foods and nurturing the microbiome that they already have may be a factor as well.

But, you know, when you go back to our considering what our hunter gatherers would have consumed, by and large, yes, we did some hunting, but pretty much it was gathering and the gathering actually included meat when meat would be found already dead, not hunted and rotting on the ground.

And same thing with fruits and vegetables rotting on the ground, rotting. in the case of things that contain fruits and vegetables, it's called fermentation.

And that fermentation process, created things that when we were hungry, we would have to eat. We wouldn't pass by, you know, a rotting piece of fruit on the ground thinking, well, that's just not up to my standards.

You'd eat it, and that's how you survive. So we've had this wonderful relationship, you know, as primates for a, well, 8 million years at least, whereby we would imbibe those foods that were fermented, we ultimately developed changes, in the way that we would handle the alcohol that the, that fermentation produced, by up regulating or turning on, over millions of years to, to make this happen.

Enzymes that would help degrade the alcohol so we wouldn't toxic for our brains and could remain fully functional. So we as humans actually handle alcohol pretty darn well because it's a legacy for us, that we have these of enzymes for alcohol degradation.

Similarly, you know, we've developed, enzyme changes that have allowed our uric acid level to increase over time. Why would that be? It's because having an elevated uric acid is a survival tool.

By allowing us to have more body fat, allowing us not to use fat for energy, compromising mitochondrial function, which sounds terrible, but it allows less energy utilization, which is a survival tool during times of food scarcity, increasing the production of glucose in the body through what we call gluconeogenesis from the liver.

And, actually raising our blood pressure to allow us to remain functional when we couldn't find water. But these days, you know, that pathway is being stimulated by our fructose consumption, the sugar found in fruit that we would find as hunter gatherers and elevating our uric acid.

Bacteriophages, Oral Bacteria, and Metabolic Health

And interestingly, it all, comes together when I tell you that elevated uric acid compromises nitric oxide production. Elevated uric acid reduces how insulin works in our bodies and compromises blood supply to our organs.

And that is a much bigger story about uric acid than simply that, you know, when it's elevated, it's going to give you gout in your toe and it's going to hurt.

High uric acid is associated with significant increased risk of diabetes, of death from cardiovascular disease. death from stroke, even all com all cause mortality, meaning death from any, any kind of any source whatsoever.

So, uric acid is a problem. What do we do to lower or your gas? I think the obvious thing that I thought I heard in there was stop drinking fructose sweetened beverages.

so most of our sugar sweetened beverages, and, coconut sugar, that's high in fructose. Yeah. I mean, what else, is something that is called nectar, of agave.

I mean, how could something be threatening for you if it's made if they call it agave nectar? Well, the reality is that, fructose is metabolized into your body, into, uric acid.

That's what happens downstream of fructose consumption. More than 65% of foods that are packaged in gross on grocery store shelves have added sweetener in these days, that's mostly fructose, because it's easy to make, high fructose corn sirup.

And it's very, very cheap. And it's really very sweet. So, you know, when you look around at the rates of soaring rates of diabetes and prediabetes now affecting 85 million Americans, the incredibly increasing rates of overweight and obesity, and then the metabolic consequences that ensue, as a consequence of these issues, ultimately manifesting as mitochondrial dysfunction.

And you and I are right back to where we started. So, it, it links, which is I know where you were going. It links then diet, this high fructose diet that is so pervasive, not just in America but globally.

Now to the incredibly increasing rates of, obviously obesity and overweight. But the downstream consequences of that, which include neurodegenerative conditions.

Getting back to what you talked about, I mean, this is a surefire way to disrupt the gut bacteria, to increase the predisposition that an individual may have, for inflammation and for immune dysregulation and autoimmunity as a consequence of diet.

These are diets way too high in fructose and devoid of fiber to nurture the gut bacteria. So I think I heard, two big

actions that that people listen could take one.

Stop consuming sugar sweetened beverages. two, look at the, ingredient label of your food and look to see was there added sugar? in. Because most of those sugars are high fructose corn sirup.

and don't fall for some of the other sugars, such as, agave sirup, date sugars, coconut sugars. I believe those are all, fructose sugars as well. And agave being one of the most threatening.

I mean, I just get such a kick out of when somebody says, oh, it's not made from sugar. We used agave in our whatever cookies bar at the. You know, you go to trade shows and they have a bar.

No, it's sweetened with agave. As if that's something to be, to be proud of. Sorry, but it isn't. You are loading up on fructose. And that is about the worst thing, that that you can do.

I mean, and to make matters worse, your body will make fructose from glucose. Your body can manufacture, fructose as a survival mechanism during times of of food scarcity or or water scarcity as well.

When you become dehydrated, you activate within your body, a pathway called the poly all pathway. And there's an enzyme that gets turned on called Aldus reductase that converts blood sugar into fructose, that then becomes uric acid.

That then will, make your body retain fat. Now, if you're dehydrated, why would you want to retain fat? Because it turns out that body fat, when it's burned or used for energy production, is broken down into two things carbon dioxide and water.

So you create this internal water. When you're using fat as an energy source, that helps you if you're dehydrated and your sensor in your brain to determine if you're dehydrated.

Sense is the salt concentration, in your blood. When the sodium concentration in your blood goes up, your brain send signals thinking that you're dehydrated and you end up, getting, absorbing or keeping water in your body to offset this sense of dehydration.

And you activate this pathway to make fructose in your brain, and throughout your body, as a matter of fact. So, why why might you have an elevated sodium level?

Well, you know, parking yourself on the couch in front of the playoffs and sitting there with a bag of salted pretzels, is going to raise your sodium level.

Your brain is going to think that you're dehydrated and you're going to activate that pathway, creating even more fructose on top of what you may be, drinking in the drink that's going to, you know, be necessary to have, along with your pretzels or potato chips.

Now, you know, before we start our interview, we talked briefly about the, stem cell article that you had seen with, progressive Ms.. do we call that article David.

I do the article is published in cell. and the lead author, was named Doctor Lucchino and MD Lucchino, rather, MD, PhD, at Cambridge.

Stem Cell Research in Progressive MS

And the study involved, 15 individuals, nine women, six men with progressive multiple sclerosis who, underwent a procedure whereby stem cells, taken from a miscarriage from miscarried, fetuses, at six weeks, were then amplified and injected into the ventricles of these individuals, through a, an MRI guided approach.

And they measured, various things. They actually follow these individuals, and measured them serially with MRI

scans with gadolinium and noted that in comparison to their previous history, they, after the injection, the stem cells had no new lesions that were what we call Gatlin gadolinium enhancing, meaning active lesions.

and in addition, they stem the tide of brain atrophy, which is another issue related to multiple sclerosis, the loss of brain substance, depending on the concentration of the cells, that were injected into their brains, higher levels of concentration were associated with even less development of brain atrophy.

Now, this was a, a phase one study, pretty much to demonstrate safety. there were a couple of markers, though, of efficacy that were looked at as I just described.

So it's, it's pretty exciting. And I think that, I actually had the opportunity yesterday to interview the lead researcher on, on that research. which is why I'm quite familiar with it.

And it's very, very exciting because I believe that, what we stumbled on, in our conversation. Stumbled. I'm sure it's front and center for his his, thinking about why it worked is that these stem cells ultimately help, metabolism and aid in mitochondrial function.

And the reason that's important gets back to our original discussion earlier, and that is that that is sort of a central player in a variety of neurodegenerative conditions, which means that there may be a role for stem cell transfer, transplantation like this, across the spectrum of neurodegenerative conditions, if it indeed targets things like, immune reactivity, immune cell function, and certainly metabolism, as is related to mitochondrial function.

So, you know, this is pretty much proof of concept, as it were. and, you know, my belief is that there will be a time when we can augment stem cells within the brain, without having to be so invasive.

I think that's coming. I think if you look at the work done by Dr. David Sinclair at Harvard on cellular reprograming, that there may be a way in the future, whether it's using the so-called Yamanaka factors or not, but there may be a way to manipulate, perhaps using a viral vector, genetics of certain cells in the body, perhaps in the brain, in such a way as to revert certain cells back to a more primitive, probably, the expression, if you will.

And in terms of their functionality and phenotype, as in such a way as they're not too primitive and don't develop into cells that are dysfunctional or perhaps even malignant, but can, as Dr. Sinclair demonstrated, go back along the same path of differentiation.

so you revert them not all the way back, but pretty far back into their lineage that you can create almost stem cells from, using this approach. So, you know, we're early in this game, that's for sure.

But I think over the next couple of decades, it's going to be possible using viral vectors that are injected intravenously. to, approach problems, like these neurodegenerative conditions.

Closing Remarks and Where to Find Dr. Perlmutter

The issue may be the blood brain barrier, but I think that there are creative ways now of temporarily disabling the blood brain barrier or even seeing that, by using ultrasound to allow certain drugs to get into the brain.

So it's a very exciting time, that's for sure. Well, you know, I could just go on and on, but, we should probably wrap this up. You know, David, this has been just so wonderful chatting with you.

could you tell the listeners where they could find you and, how to, get more information about everything that you do? Best thing to do is go to drperlmutter.com that's kind of like the clearinghouse for all the things I do in terms of outreach.

I do have a podcast called The Empowering Neurologist and, I really am grateful for that. I'm grateful that it's giving me the opportunity to get really important message out to anyone who will listen.

But from a personal perspective, I get to talk to incredibly, incredible individuals who are doing some amazing work. I think you've been on the podcast a couple of times, and I just so look forward to the opportunity to connect with people like, like you and others who are doing amazing work.

So thank you very much. Thank you. And this has been wonderful. and thank everyone for listening to this great interview.