Introduction and Summit Welcome

Welcome to the Parkinson Solutions Summit. I'm your host, Dr. Kenneth Sharlin And today I have the great pleasure of interviewing Dr. David Perlmutter.

He is board certified neurologist and six time New York Times bestselling author. And I want to just personally say being excited to interview Dr. Perlmutter is an understatement.

He has influenced many physicians who have decided to take the turn from conventional medicine into functional and regenerative medicine. He's certainly influenced me and a time in my life where I thought if things don't change, I'm just going to have to find a new career.

And I was so blessed to hear an interview that he gave at the time he had published his book Green Brain, and it completely changed my life. So, Dr. Perlmutter, thank you so much for what you've done for me and for so many other people suffering from neurological conditions.

So welcome to the Parkinson. And Dr. Sharlin Thank you so much for having me. I'm honored to have the opportunity to participate in this summit. I think it's really very important.

As a fellow neurologist, Dr. Perlmutter, curious about your own journey and how is it that incorporating things like food and movement and stress, resilience and sleep and thinking about ketosis and, you know, uric acid, the microbiome.

How did all those things, you know, those are things that, you know, we don't really study in medical school rather than maybe.

From Symptom Treatment to Prevention

Gout, you know, or neurology school for that matter. Right. Right. You know, I was out in practice for a number of years and finally had the revelation that I was treating symptoms of disease.

I wasn't treating disease. It wasn't treating Parkinson's, Alzheimer's, epilepsy, headaches, you name it. I was simply treating the manifestations. And I had found that to be less than fully rewarding.

I didn't want to spend the rest of my life just focusing on the smoke, but ignoring that fire. And fortunately for me, around that time, which probably would be about 25 or so years ago, we began to see evidence in the literature that people were indeed exploring the role that lifestyle choices played in predisposing people to neurological problems of one sort or another.

You know, even the notion that lifestyle factors could affect one's risk of something like Parkinson's or Alzheimer's back then was beginning to be spoken about, but was really pretty much off the table in terms of mainstream neurology.

And, you know, unfortunately, through your eyes and my eyes, it's still pretty much off the table. We don't really, even in the journal, see very much activity.

And interestingly, what I have found is even when these studies are published that deal with the impact of exercise on reducing risk of neurodegenerative diseases, my mention colleagues don't see them.

They're invisible to them. Articles are published living in it that I realized that my destiny had more to do with treating the fire and not the smoke, trying to figure out why the heck people were getting sick in the first place.

And that very much opened the door to the notion of disease prevention. And that's been my focus then for the past quarter century, is the idea that these compelling, challenging, devastating neurological conditions, neurodegenerative conditions, may significantly be preventable, I think is challenge is mainstream, that's for sure.

And that's good. I'm I'm fine with that. Always have been. But it really offers up an opportunity to be, in my case, more fulfilled in terms of what I do on a day to day basis.

Because, you know, it's a it's a pretty narrow group of us who are like yourself, involved in getting the message out that each person is the arbiter of his or her brain's destiny.

Yes, we tend to want to live our lives. You know, we're convinced that we should live our lives however we want and that there's going to be a pharmaceutical fix for whatever maladies befall us.

Right. Reality is, as it relates to neurological conditions, neurodegenerative conditions like Alzheimer's and Parkinson's, there's very little out there that actually treats the problem.

Yes. As it relates to Parkinson's, we can manage symptoms pretty well, but the disease continues relentlessly and inexorably worsening with time. And I think it's really important to get our arms around the notion that what you and I are going to talk about today is the actual way that we understand Parkinson's in terms of its genesis, what's causing this problem.

This can help people resist the opportunity to get the disease and even help them in terms of slowing the progression of the disease. Once it has manifest in that we're targeting the cause, not just focusing on the symptoms, the manifestation.

That's been very exciting for me. Yeah, 100%. And if I can slightly paraphrase some of the things you said, as you point out, your focus has been especially on prevention.

Gut Health, Microbiome, and Leaky Gut

And as you know, most of the things that we talk about in terms of prevention in the language of functional medicine can be mediators that ultimately change the trajectory of the person who has the disease now.

So I'm. Really glad you said that. You know, because. I think back of a criticism somebody levied towards me once and said that, yes, whatever you want to talk about in terms of Alzheimer's prevention, that's been validated. But to apply those to treating an Alzheimer's patient, it doesn't make any sense.

And I had to roll my eyes. Why wouldn't it make sense? You know, it's like, you know, there's there's merit to focusing on prevention. Desmond Tutu once famously said that, yes, it's important to pull people out of the river, but it's equally important to determine why they're falling in in the first place.

And that's where our emphasis is. That's what we want to explore, because we keep people from falling into the river of neurodegenerative conditions. You don't have to worry about pulling them out.

Yes. And yet we have about a million or so people suffering from Parkinson's and one of the fastest growing neurodegenerative diseases that we know. We're still not in this, you know, six times as many people with Alzheimer's, but we've still got a huge problem on our hands.

So it's good for the folks looking for solutions to know that there is absolutely much that they can do. Yeah. Yeah. And I would just like to take that offer, this opportunity to indicate that I think we've been dramatically underestimating the number of individuals with Parkinson's.

And I think that we now see literature indicating that we may have underestimated it by as much as 50%, 50%. So it's a much bigger issue than we've paid attention to.

Well, we then has been paid attention to in recent years. It's a lot bigger issue. And, you know, based upon some of the things that I think you and I are going to explore today, we can understand why that is.

That's right. And if I can also piggyback on an earlier point, because I think it's going to be critical for the viewers of this interview. I completely agree with you that, you know, we are definitely seeing in the peer reviewed literature articles about the microbiome and and fill in the blank, whether it's M. S., Parkinson's, Alzheimer's.

I, you know, articles about gut permeability, lipo, polysaccharides, all the things that we talk about in functional medicine. But where I think things have been missing within the conventional neurology space and most of the other areas of medicine is that that's great information.

But our colleagues really don't have the skill set or the toolbox to say, okay, there are alterations, the microbiome. That's great, we all agree. What do we do about it?

Right. Well, I. Think that's fair. I think that if somebody rejects the notion of this relationship because well, what do you do about it? I think where we are in the technology and understanding as it relates to leveraging this knowledge about the microbiome is at a place where we really don't necessarily know what we should be doing.

I mean, it varies if we're going to jump right in. One very small study recently published demonstrated actually improvement in motor function in in Parkinson's patients who had a fecal microbial transplant.

In other words, having fecal material instilled into their colons from a non Parkinson's patient, they actually had improvement. I mean, what is that Do?

It shows us that mechanistically there is something going on with respect to the gut organisms, perhaps well beyond the bacteria that influences neurochemistry, influences brain function that had a positive outcome in these individuals.

So but, you know, to to broaden that to a general group of individuals with Parkinson's, I think, to be fair, would be premature. But just the fact that we become aware of the relationship is important because what does that do?

It lights you up and it lights me up to start thinking about, hmm, if the gut bacteria are in some way related to the brain and specifically Parkinson's, then maybe we should be looking at some of the things that affect the gut bacteria and determine as their relationship to Parkinson's that we can see and be.

Does it make sense to do as much as we can to keep the microbiome as happy and functional as we possibly can, to make sure that we are looking at gut permeability via the various metrics that we have and realizing, as you mentioned, that we should be paying attention to LPs or other markers of gut leaking is because now we're seeing science that's relating these two things.

You know, where does that go in terms of therapy? How do we help? People don't know the answer to that just yet. But gosh, that's something that tickles your mind and something that you think about before you fall asleep at night because novel ideas will will sprout from that sort of understanding. Yes.

So the gut is really foundational. And that's something, of course, you hear over and over again, Parkinson's, Alzheimer's, health in general in disease.

And our folks who are looking for solutions, they do need to be thinking about gut health as being what a good stepping stone, if you will, to changing their own trajectory.

Parkinson's as a Whole-Body Disease

And I'm just wondering if you share the perspective, because you mentioned leaky gut, but I always say that while we can certainly measure markers of gut permeability, so inulin or whatever, I want my patients to think of leaky gut almost philosophy ethically that it's not something we treat like an infection.

You know, you get ten days of an antibiotic and you're good. You have to think that your gut is always susceptible to abnormal or increased permeability that drives this thing we call inflammation.

And so how we move through our lives, how we navigate that moving forward, really has to be with that leaky gut mindset all the time, essentially, I would agree.

But, you know, it becomes really sort of a a holistic perspective we take of the entire body, not just the gut, but everything. Yes. You know, originally Parkinson's was thought in in Chinese literature.

The Nanjing, the yellow emperor in the fourth century B. C. looked upon as a liver abnormality. And how intriguing it is that, you know, modern in modern times, we have recognized genetic issues that predispose people to problems with how their liver can detoxify certain things being related to Parkinson's disease, either through the mechanism of toxicity, which, you know, hopefully we'll have a chance to talk about.

So, yes, it is. It's a holistic perspective, that's for sure. We know that, you know, the gut is highly influential. We know when there's leakages of the gut.

Oftentimes there's permeability or weakness, if you will, of another membrane, the blood brain barrier, which does allow certain things to get into the brain and quench the brain that otherwise should not have had that ability.

And, you know, these are similar entities. So it's not good enough to take care of one problem and think, therefore, the whole organism is going to be healthy.

We've got to look at everything that every may, every member of the orchestra is playing a part here. And to consider Parkinson's as being a brain disease is beyond myopic and out of date.

We recognized, for example, that there's a super highway between the brain and the gut called the vagus nerve, and that there's a relationship between the functionality of that vagus nerve and Parkinson's risk, such that when the vagus nerve is compromised, risk for Parkinson's actually goes down.

Well, what does that tell us? Does is something being something being transported in this superhighway, that vagus nerve that may relate to risk for Parkinson's disease.

Is that alpha synuclein or abnormal normally folded protein? Who knows? But the point is that it just solidifies this notion that things are related, delivers related, genetics is related, detoxification pathways are related, and we need to gain a much broader understanding of our patients rather than saying, look, part of your brain is degenerating, the part that makes a chemical will give you back that chemical.

End of story. Yes. Well, for patients with Parkinson's, you know exactly what I'm talking about. You go into the neurologist and that medicine that levodopa or the what mimics levodopa in the brain is what you're getting to put it back, because it's got it's not there anymore as much as it needed to be.

And that is the really the extent of the therapy. Where is the question answered? Why is it gone in the first place? My gosh, that's an important question to ask.

Why did I lose my those cells in the past? Act of my substance in anger that makes the dog meat. How come I lost those cells? What's going on? Why don't we pay attention to that?

And again, that's looking at that fire, not just treating the smoke. Yeah. So as we all say, common things being common and we do, of course, try to take that more personalized approach in the clinic, the individualized timeline and so forth.

But from your perspective, or the three or four or five things, there are the most common triggers that you you think of that you've seen in your experience? Well, it's very difficult to say.

We live in a very toxic world and Parkinson's is getting worse with time. And I think it's a manifestation of basically toxicity. You know, in our world, this is incredibly toxic with things that are hidden from us, things that are in the groundwater, things that we're exposed to, the particles in the in the air that we breathe.

These PM 2.5 that are so ubiquitous, even mild head trauma, you know, in a repeated way is certainly a risk factor. Being male is an important risk factor.

Various medications that affect our digoxin location ability are looked upon as being risk factors. So, you know, these things that I've mentioned are all common and who lives a life that's free of exposure to the various things that we are now exposed to aggressively.

So I don't know that I can, you know, put my nickel down in terms of saying this is the most common cause that I see in my patients because, you know, there's not one variable.

People have their genetics as the background upon which environmental factors then play out that I just mentioned. Right, was, you know, many others and we can talk about.

But I think that it's quite clear that toxicity in one form or another will break them down is playing a central role. I mean, you know, Parkinson's is primarily pretty clearly a failure of mitochondrial function, failure of the energy producing parts of certain cells.

Toxicity, Mitochondria, and Disease Mechanisms

And why specifically in that part of the substantia nigra in the brain stem that makes dopamine and therefore we have all these downstream manifestations is certainly unclear. But I think that this is an area of the brain that's very highly metabolic and very highly dependent upon really tip top mitochondrial function.

And we know that there's a very strong relationship between exposure to certain toxins that specifically damage the mitochondria and risk for Parkinson's disease.

And indeed, we can create Parkinson's in experimental animals by simply giving them some of these environmental toxins to which UDP goes, you know, clearly demonstrated with what's called the AMP experience and mean you.

May I go through that briefly so here's. Absolutely. A chemical that was synthesized. Interestingly, people may not know the story. We understand that it was synthesized by people wanting to get high.

It was like a Demerol kind of drug in California in the eighties. And and as soon as they injected this drug, within days they got Parkinson's or Parkinson's.

They were Parkinsonian, developed all the clinical features of Parkinson's and were actually treated with standard medication and had a response, meaning that, you know, you pretty well emulated what's going on.

And this turned out to be empty be metabolized to amp plus which is a mitochondrial toxin damages what's called complex one of the mitochondrial electron chain activity that makes up energy but the interesting part of the story is that actually happened for the first time in the late 1970s by a chemist, a chemistry student synthesizes chemical, and he was 23 years old and inhaled it.

Know, he injected it, actually. He developed Parkinson's immediate within days and ultimately died of that disease. Wow. So that said, the the upside of the story, if you can imagine there is an upside is it allowed researchers to then use that chemical to study what goes on with the chemical, how might it be damaging the brain?

And they studied it in rodents and in primates and in fact, were able to create Parkinson's immediately and demonstrated back in the eighties that this is a disease characterized by problems with how the mitochondria work in that particular part of the brain.

Okay, what are we doing now? We have taken a step back from saying, oh, your brain is low and don't we take this pill and I'll see you next month. So now beginning to understand the fire, not just the downstream manifestations that smoke the fire, what's really going on here is something is threatening the mitochondria in this one particular part of the brain.

And secondarily then the production of this chemical is compromised. Well, that really starts to set the stage for understanding what is the disease in reality.

Getting back to your question is, what do I think is causing? Because we know there's a lot of things in our environment that can similarly damage the mitochondria.

And importantly, wow, what can we do to help the mitochondria get back online? Even before the clinical manifestations have occurred? Yeah, I have to tell you, a study was published this morning.

I was drinking a cup of coffee knowing you and I would spend time together and this was in my inbox. A study from Australia indicating a new imaging technique is able to predict about 24 years ahead of time who is likely to get it versus who is not.

Wowsers, can you imagine? Now we know who to target so that we can begin to develop protocols to enhance not only the function of their mitochondria, but to protect them against damage as well.

That's. Oh, that is. I'll have to look for that article. That is fascinating. I guess I was I must admit, Dr. Perlmutter is leading you on just a little bit because you have touched on things that I wanted you to kind of say.

We've talked about the gut and the microbiome. We've talked about you've talked about toxicity. You've talked about the mitochondria and the empty model that came out.

And I was actually very blessed to be at Emory at the time that Melinda Long and Gary Alexander and Doug Wallace were all there working on primates at the Iraqis Research Center there.

So I very intimately familiar with that. And IT folks are interested there. Actually, the videos that were originally published in conjunction with The Lancet on YouTube, where you can see those cases and see the individuals coming in with Parkinsonism and then getting treated with leave a dope and having some, some with remarkable response to leave the dopa.

But what we're seeing, I think, is something that and I think you did use an analogy of sort of the symphony. And I would say, you know, there's different parts of the symphony, but in the end, it's the whole symphony right together.

It's never just the strings or just the percussion or just the horn section or what have you. And so we need to start seeing how our mitochondria work together with the gut, where it together with the microbiome are affected by toxins.

And then ultimately and there are other factors we could talk about like hormones and other communicators of the alluded to obviously dopamine we know other neurons transmitters systems are involved how these ultimately work in concert and then and then having that clinical framework to help people, you know, find solutions to what's now affecting them.

Well, you're right. And this is you know, I hate to belabor the point, but it's not typically the approach that we see in, you know, and as we're having this discussion in terms of what the mainstream is interested in.

So the main mainstream of neurology and I'm not trying to be derogatory, but it's really focused on helping people manage and that's it. That's honorable, giving the medications that allow them to be functional, help them with their tremor, whether it's medications or more aggressive therapy like deep brain stimulator or exploring things like stem cell therapy.

I'm in I mean, people have to be able to function. But let's be clear. If we're able to treat the disease earlier and not the symptoms that disease so that people don't progress and require as much symptomatic treatment, I think there's much to be said in terms of why that's valuable.

So all of these systems, these functional systems, you're mentioning energy and gut health assimilation. I'm translating a little bit into the framework we use through AFM.

Of course, they all converge on some basic processes that I think are very central to what you write about and speak about. For example, oxidative stress and chronic inflammation action, which then in turn sort of fuels that fire, does it not?

Ketogenic Diet and Mitochondrial Support

It does. You know, unfortunately, when when mitochondria become dysfunctional, then the local environment is more polluted, if you will, with higher levels of these damaging chemicals called free radicals.

So more oxidative stress occurs in and around these mitochondria that are dysfunctional, that have now been traumatized by whether it's DDT or wrote down or Paraquat or AMPTP that we just talked about, or trichloroethylene or any number, manganese, you name it all of these toxins that are related.

So once that happens, mitochondria then function less well and increase production of free radicals that damages that local environment, which causes more damage.

And so it creates, we call it feedforward process. And we really want to do what we can to limit that damage and in an ideal world, enhance mitochondrial function.

Again, to the extent that perhaps consider growing new mitochondria does not sound way out. Well, it's not we call it mitochondrial biogenesis, and we know how to do that.

That's something that happens when you fast. For example, it's the reason that rapamycin, for example, which is a drug that you and I looked at years ago in terms of being an immune suppressant used in transplant patients to keep them from rejecting their transplanted kidney or whatever it may be.

But now a day is looked upon as a medicine that enhances the growth of mitochondria, enhances the body and brains as if they were different clearance of defective mitochondria, and specifically can enhance the function of that one part of mitochondrial function called complex one that is not working well in the Parkinson's brain.

Again, rapamycin, it's become you know, the talk of the town is related to longevity. We do know that it is effective in a particular mitochondrial disease called laze syndrome and in play early country syndrome.

It's a specific genetic defect that codes for less mitochondrial function in complex one works there so there is some preliminary animal work now being done with rapamycin to enhance and protect mitochondrial function.

I think that's going to go a long way. Great. That doesn't mean that people have to come off of their levodopa treatment and suffer. No, these things can be done together.

But that's a far more comprehensive approach in dealing with Parkinson's, treating both the smoke and the fire, treating not just the symptoms, but actually treating the underlying disease.

And I think that's the approach that that people deserve. Which is that's a wonderful lead in to something you've been a huge proponent of that supports both mitochondrial health and function overall.

And that is things like a ketogenic diet. You know how we use food as medicine and in particular maximize the ability of our mitochondria to utilize energy through burning fat for fuel?

Can you kind of update us on that? Yeah. So, you know, one would think then that going on a ketogenic diet and and enhancing mitochondrial function which it does do and enhances mitochondrial biogenesis and autophagy would be a good idea.

And in fact, an interventional trial was carried out by Dr. Matthew Phillips in New Zealand and he demonstrated some pretty compelling improvements in what is called the UPD, RC, the unified Parkinson's disease rating scale, kind of a scale used by researchers who are going to do something interventional, determine if, you know, it helps Parkinson's patients in terms of their functionality.

There are three different components of the scale and he demonstrated that by putting people on a ketogenic diet. So that's mitochondrial therapy. Interesting story, how I found out about his research.

I'll tell you the story very briefly. I was on a boat in British Columbia. The guy in the boat next to me had some troubles with his water pump system, fresh water pump system.

And I love doing those kinds of fixes. So I offered to help him and we're in his engine room. I'm looking at his water pump, whatever it was. And we're talking, of course, as you know, about our children.

And so my my son is in New Zealand. He's a neurologist and he's researching something called the ketogenic area. Ketogenic something diet is in respect to Parkinson's disease.

And he looked at me like, you know, not that you, the boat mechanic, will know much about that. And I said, Oh, really? That's that's kind of interesting.

I kind of know a little bit about that. It's kind of like why I said, I really think that's interesting. I'd love to talk to him and I said, if you look over if you look over the boat you see right there on the windowsill is a bottle of MCT oil.

So I kind of know if I ketogenic diet. So he called his son and connected us and he in fact was at that time finalizing. His name is Matthew Phillips, finalizing this study on interventional trial, ketogenic diet and Parkinson's.

And I actually ultimately interviewed him on my podcast and and got to know me then published a similar study on Alzheimer's and demonstrated not just stabilization but actually improvement in cognitive function, as you might expect by putting people on a ketogenic diet.

Uric Acid, Fructose, and Parkinson's Risk

And again, these approaches are dietary, nutritional and are not done to the exclusion of using pharmacotherapy. By all means. If you have a patient who seems to do well on levodopa therapy or some sort of agonist therapy or amantadine, whatever it may be, by all means those can be continued, but at the same time, let's target the underlying problem.

Let's tie in which we know been established. Getting back to our understanding of this MPP story that it's a mitochondrial issue, we know that man and magazine, which are used to get rid of pests on our vegetables, are mitochondrial toxins.

That's how they work. It's why these over the counter pesticides are also used in experiments for laboratory animals to create Parkinson's, and yet you can buy them online to treat your vegetable garden.

Now, something really. Really disturbing, right? Right. You know, I really want to ask you about uric acid, because I know this is the topic of your newest book and folks need to go out and buy that book, drop acid.

But uric acid is actually not only just fascinating in general, but has kind of a paradoxical role in Parkinson's. And I've been I've been so looking forward to this interview, and I'm been wondering how you can guide me, really, as a neurologist and us through understanding why is it that while we look at this as a marker of inflammation and we generally want lower uric acid, why is it that higher uric acid levels in Parkinson's appear to have some protective

effect?

Doesn't make sense. It actually is that difficult when I explain this to, I think get your arms around it. Years ago with that observation, I was actually treating Parkinson's patients, and this was long before our new understanding of your gosh, this might have been 15 years ago by giving them I know seeing and because I was seen actually giving and actually will increase uric acid and and we thought that because of that observation that it seems that, you know, Parkinson's patients had a low uric acid that we can raise of your gas that might be protective because we know that uric acid can be an antioxidant, but also a pro oxidant.

So it functions in both ways. And where it acts as an antioxidant is in the serum and where it acts as a pro oxidant, a damaging, free, radical enhancer, if you will, it in the cell where the mitochondria live.

So why would a Parkinson's patient have a lower uric acid? Well, think about it. What are your Parkinson's patients look like? They're generally pretty thin, aren't they?

And they generally have pretty low body mass. Unclear why. I mean, I can speculate, but that would tend to correlate with the you're a lower uric acid so you know, to answer the question, a study was actually carried out published in the Journal of the American Medical Association in 2021, where they did exactly what I had done 18 years before.

They they had this observation. They said, Well, why don't we raise the uric acid level in our patients and follow them on the unified Parkinson's disease rating scale?

And lo and behold, it didn't help them actually worsened their situation, as you would expect. Yes, you might have increased antioxidant coverage in in the blood, but you actually worsened free, radical mediated stress within the cell where the mitochondria live.

So it is as expected, uric acid is a mitochondrial toxin and at high levels would be expected then to worsen Parkinson's disease. So it would have been great because it's sure very simple to raise uric acid.

It's exactly the wrong direction. You want to lower it and, you know, uric acid does when it's elevated is not only threatens mitochondrial function, but is involved in increasing body weight in increasing blood sugar, insulin resistance, reducing blood flow.

So there are a lot of down downsides of having an elevated uric acid. And I was very, very much looking forward to that study being published. And it was it would have been a tough thing to explain.

But finally they said no giving people, I'm saying, to raise a uric acid bad idea as it relates to Parkinson's for the reasons I just explained. And then you talked about some of those drivers of uric acid.

And one thing in particular is that the viewers of this interview can immediately take action on, and that is to eliminate fructose high fructose corn sirup or fructose from their diet.

It's ubiquitous. That's right. I mean, you know, we live in a, I would say, world, but at least a country in which at least 60, close to 70% of packaged foods have added sweetener.

And these days that's generally high fructose corn sirup or a derivative thereof y because it's really, really sweet and it's really, really cheap. So, I mean, it comes from where do you think it comes from?

Corn, obviously, and corn sirup. So this is yet another way that the government can support the growth of corn because it can be made into sugar that can go into all of our foods that can be responsible for so much, so many of our maladies from metabolic perspective, driving up the costs of health care and making people sick.

So it is fructose and you know, over the years in medical education when we were trying to figure out, well, what causes high uric acid, two things. Number one, it was always in the context, gout, rising, high uric acid causes gout.

And really all we learned about. And number two, it was to put people on a low purine diet purines are the breakdown products of DNA and RNA that are found in various cellular foods like meat and yeast and certains and various fish.

And, you know, even certain plants can be high in appearance, for that matter. And also when we would break down our own body tissues like muscle, which is the reason that Parkinson's patients have a lower uric acid level, is because their muscle mass is lower as there's a, you know, obvious observation.

And so that's what we were told. Put people on a low purine diet, reduced their meat, their wine, their cheese, etc. and that way they won't have as higher uric acid.

Well, the biggest player by far and way is fructose. And fructose is 50% of table sugar. Table sugar or sucrose is half fructose half glucose. But we get a lot of fructose at higher levels.

I mentioned earlier that through the use of high fructose corn syrup, which is in the sodas and the sources of the condiments and everything, that's just basically that's sweetened.

So, you know, the point is that that is elephant in the room in terms of the reason that Americans and really the Western world and, you know, pretty much globally now we're seeing progressive increases in uric acid.

High levels of uric acid are far more dangerous, aside from just increasing a person's risk for gout. As I mentioned, higher levels of uric acid create metabolic mayhem.

They rate it raises blood pressure, insulin resistance, blood sugar damages, mitochondrial function. And as such, you might expect that higher levels of uric acid would then be associated with bad things happening in the brain.

And it it's true. True. One study looked at 1600 individuals, measured their uric acid adults, followed them for 12 years and found a significant increase in risk for dementia in those with the highest uric acid, about 80% increased risk, about a 55% increased risk for developing actual Alzheimer's and about a 165% increased risk of developing what's called vascular dementia or mixed dementia, as you might expect when you dove deeper into what elevated uric acid does.

So it's been it's a real learning process, I think, for a lot of health care practitioners to realize there's a heck of a lot more going on with uric acid than simply causing someone to act out in their big toe.

Yeah, so, so important. You know, quick anecdote. I heard this story on NPR about how high fructose corn syrup actually came in to the market in the United States.

And you were saying that it was cheap. The reality, at least the way this story was explained, is that it wasn't cheap in the beginning, but it had to do with a very savvy CEO of the Archer-Daniels-Midland company who manipulated the economy of sugar in the United States by limiting the import of cheap sugar.

Closing Remarks and Resources

So that. That's right. Yes. Fascinating. And then had this product that he really saw from a from a business perspective as being well, if I can just level the playing field in the cost of sugar I have something that these food manufacturers will love to use in their products and indeed that came to be so I don't want to blame person for Parkinson's disease and all the elements of the world, but certainly that appears to be very pivotal.

Yeah, it is. And I think, you know, for your viewers who are wanting to learn more about Parkinson's, it's interesting it revisited that. But, you know, I think there's a lot more you and I can dove into that will be nuggets to take home.

So there you go. I hope so, Dr. Perlmutter. I really hope so. You have enlightened us all in these past 40 minutes, and I really appreciate your time.

I know the viewers do that. They have gosh, I almost hate to say if they haven't heard of you, but if they want to find more out, more about you, we just send them to drperlmutter.com, D R P E R L M U T T E R. COM My podcast is called The Empowering Neurologist, and we're actually as of late, we've had several programs on Parkinson's, especially as it relates to toxicity and exposure to toxins.

So The Empowering Neurologist is a podcast, but I think the clearing house good drperlmutter.com And get on his mailing list. He's got a terrific mailing list.

He sends out, you know, the scientific papers, a little synthesis of what they mean and if you want to learn more, and I certainly always do so Dr. Perlmutter, I am on your mailing list.

I encourage other people to be on that mailing list. Well, Dr. Sharlin, thank you so much and thank you for the opportunity today. Thank you.