Revitalize Your Brain With Plasmalogens Transcript

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Introduction to Dr. Goodenowe

Welcome back. I'm excited to introduce you to Dr. Goodenowe. His research into the biochemical mechanisms of disease started in the 1990s. His curiosity about the biochemistry of life is as insatiable today as it was over 30 years ago.

In the past 30 plus years, Dr. Goodenowe has invented and developed advanced bioinformatic technologies, designed and manufactured novel novel supplements and identified biochemical Prodromes of numerous diseases, including Alzheimer's disease and other neurodegenerative diseases.

You can see why I've invited him here today. I am so excited to introduce you to Dr. Dayan Goodenowe. Thank you, Dr. Sandison. Very happy to be here. So let's dove into it first.

Personally, what made you interested in neurodegeneration? Well, from the very beginning, just as a young boy, I was very interested in how the brain works.

And so as one thing leads to another, you know, you can think of biology as the interaction of groups of organisms with each other. And then you go through school and you think of physics and you think of the subatomic particles.

But it was really chemistry that really grabbed my attention because we kind of live in a chemistry world, right? The lowest common denominator is the atom and the classical laws of thermodynamics that, you know, matter can't be created or destroyed.

It really is the transformation of these molecules. And we either have chemical transformation that we do in chemistry, like burning fuel in your home, or you have biochemical transformations which the human body does.

And as you go on, as you go deeper and deeper into this thing, the most fascinating mystery of all is really how the brain works and how the brain somehow creates a quantum mechanical power that it can do choice making in consciousness, which is really a quantum mechanical thing and understanding how that works.

And so, you know, as I went through school, my Ph. D. is actually in psychiatric medicine looking at the biochemical mechanisms of disease. And, you know, you just grab a piece of the yarn, right?

And you keep on pulling until the whole sweater comes apart, so to speak.

Why Neurodegeneration and Brain Chemistry Matter

And one thing leads to another. And so as you as you each question raises a list of other questions. And since my background is in synthetic chemistry and in technology, so not only do I have the opportunity to investigate these questions, but actually build technology for it and that's really where this whole Alzheimer's and plasmalogens came in.

It really came from advanced mass spectrometry. And it's still the technology we use in the programs can test today. And then as you learn more, you start changing your preconceived or your or your plot options, right?

You know, we're taught certain things and we assume those assumptions are correct, are based upon, you know, centuries of research. And some of them obviously are, but some of them aren't.

And a lot of science falls into this association is causation problem. Right. And associations are really valuable because we use them for diagnostics.

Right. And a huge part of medicine is about diagnosing a disease. And then it's only kind of an afterthought. We think about the causation. So anyway, so that's how for me the neuroscience.

And then as we looked into molecules and this is where the plasmalogens story came in because they were identified using this high field mass spectrometry to be associated with reduced cognition and the incidence of dementia and all cause mortality.

And previous researchers, you know, I've seen some of this research work because the plasmalogens story and we'll get into it in a minute was you know, we were invented back in the we're not invented.

They were discovered back in the 1920s, I think. And then we've known since the 70's that children with deficiency using plasmalogens from from genetic inborn errors of metabolism are, you know, very damaging.

They typically die within a few years of birth. So we know how important this. Kind of plasmalogens what is that? What are we talking about? Right. So as we start talking in the middle of the story. Exactly.

So what are plasmalogens? Right. And and why have you never heard of these damn things? Right. Right. Well, this is where you almost like as you like.

I'm a neuroscientist, right. And I my piece is actually in looking at the biochemical mechanism of psychiatric disease. And I had never heard of plasmalogens, never been thought of a plasmalogens, was never in any of my textbooks, was never ending my lectures.

And then so I use this mass spectrometry technology to find examples plasmalogens. So then of course, you know, you start doing the research as to what the hell are these things.

I had the same question and there phospholipids and they're not trivial phospholipids they're actually a lot. You're talking 30% of your brain phospholipid volume are these plasmalogens and so phospholipids are these molecules that are like soaps.

We have a polar head group, we have a non-polar tail and your body uses these fossil this for many things the mean the one. With dissolve in oil and the other one would dissolve in water.

Perhaps one molecule with a polar head on each side, one that would dissolve in water, one that would dissolve in oil. So everybody can kind of imagine that, like if you put soap and water in the same container, you see how they separate.

And this is that bridge between those separated liquids. Correct. So if you take say you take oil and vinegar, right. And they have two layers and you shake it and then eventually it settles out.

Right. But if you put some egg yolk powder in there and shake it, all of a sudden it stays all together. And the phospholipids and egg yolk or what actually create this emulsification.

So when you combine fossil together, they create what's called a phospholipid bilayer, and that's actually a biological wall. So two of them combine and the fatty acid non-polar chains combine in the middle, pull polar head groups on the outside, and you create this impervious lipid wall and your body has 30 trillion cells like a massive apartment complex.

Every single one of these cells is defined by this membrane, and this membrane is made of phospholipids and plasmalogens make up a huge amount of it in the brain, 30% in your heart, 50% of the phospholipids in your heart are.

plasmalogens is an incredibly large number. And what's so interesting about these molecules is that they're so important to early childhood development.

So we talk about neurological perspective, these phospholipids and there's different types. There's the synaptic version. We talk about the Omega-3, we'll get into that and the Omega-9, which is the myelin.

So these membranes that you have in your body have different purposes and different functions. So in the for neurological perspective is that you have two main classes.

One is the myelin sheath. It's a protective coating on your axons that enables a signal to go from point A to point B, and you want that signal to pass through the axon and not leak out at all.

And so the plasmalogens there provide this protective coating. It's called myelin. And it's actually a key ingredient in human breast milk is for myelination of children, is that that the plasmalogens in and we're actually born with really kind of these raw bare wires so this myelination process, which is about 60% of the brain, is white matter, which is all myelination.

And then the other part is gray matter. And that's usually the cell bodies, but also the synaptic function where the wires end and they get bared, if you will.

And then they have to do they have to communicate from one to another.

What Plasmalogens Are and Why They Matter

And that communication process happens at what's called the synapse. And that synapse is where neurotransmitters get emitted from one neuron to the next neuron.

But of course, it's human biology, so they use biological materials to do this and biological materials that involve that the cellular fusion process are dependent upon plasmalogens.

Like if you have no plasmalogens in your synapse, neurotransmitters can get released. And so that's that's where plasmalogens are a critical component.

So from from the neurological perspective, it's really those two components. Then for cardiovascular health, it's you're involved in inverse cholesterol transport.

They're critical in lung function. So children that are born premature often will get bronchial dysplasia. They have a plasmalogen deficiency asthmatic.

So plasmalogen deficiencies. And so there's a few other core components of human physiology, all this stuff, all these stories with myocarditis. When you have heart inflammation that causes a severe depletion of plasmalogens.

So these these molecules are used all over the body in every single cell. Specifically, tell us again, how does this relate to dementia and how is this relationship between low plasma levels and dementia originally discovered?

Yeah, so that's what I that was my one of my claims to fame is my original patent portfolio was relating to this causation of plasmalogens depletion because plasmalogens are also your most important antioxidant, the body, and they fall into one of the three classes of molecules that are so important to human physiology that the human body does not depend upon any nutritional source.

Cholesterol, for example, 75 80% of all the cholesterol body you make yourself get very little from, you know, you could eat a completely cholesterol free diet and still have enough cholesterol in your body.

Same thing with possible choline is another molecule that your body fundamentally makes from scratch and the third the third one on that big huge list of these plasma allergens.

And so since you make a lot of them, but they're also they have this vinyl ether bond that makes them very sensitive to acid, which is what your body uses to use to fight inflammation.

And so they get if you have oxidative stress, plasmalogens get depleted in oxidative stress. So studies back in the nineties have seen plasma decreased in the brain of people still with Parkinson's or Alzheimer's.

And it was assumed that this was due to oxidative stress. So, okay, you know what? The Alzheimer's brain has oxidative stress, very well documented. Microglial activation, that kind of stuff.

And so this low plasma level really was assumed to be a symptom, not really a cause. And then when I looked at the temporal relationship of predictability of the future and the association in the blood, it became very clear that this was not happening after the disease.

It was happening before the disease. And then when you start digging into it, you start understanding why. And so the canary in the coal mine for dementia is the cholinergic system.

And we know that very, very accurately from the seventies. So things that improve the cold cell killing transmissions like you see the clean arteries, inhibitors, for example, will improve cognition.

And not for very long. But they still do like, if you like, the clinical trials back in the early nineties, we're pretty clear that it works, but it doesn't work very long.

It's not disease modifying, but likewise if you give someone scopolamine, like if you give a cooling antagonist, you can create dementia immediately. And that's a drug people use for for abusing people called the Devil's Breath.

So we know very clearly that from a cognition perspective, the cholinergic system is critically involved. It plays a role, but not it's not the only thing that can cause dementia.

Well, that's the that's the that's the core component. So the nicol's bizarre. And it's in its projections, the cholinergic projections throughout the brain and they act, you know, acetylcholine release from the presynaptic neuron activation of the muscarinic receptor and the postsynaptic neuron like even those are pretty robust and if you take a look at nucleus B-cell shrinkage for our terminal fields, if you look at functional connectivity of the brain and you look at the cholinergic endpoints, those things all correlate very, very strongly with cognitive function.

And so if you look at, you know, historical data on that perspective, so the question now becomes what are the different mechanisms that can lead you to the dysfunction of the cholinergic system?

And there's many ways of getting there, right? There's not just one way of getting there. And that's why we have these multiple causation, right? You can have an inflammatory, you can have vascular dementia, you can have Alzheimer's dementia.

So you have these different types of dementia. You can get a concussion and lose dementia. You can have liver surgery and have dementia. You can go under bread.

You can have you can have surgery for breast cancer and have post breast cancer dementia. So dementia is something that is clearly a behavioral psychological process.

It's a function now. It's a functional measure. It's how someone can actually perform in their work the world. It's no different than how fast I can run 100 meters.

Right? So cognition is a is a performance value, not a biochemical value. And the question is, is what are the biochemical contributors to that? And when you when you unravel that onion, you end up with this cholinergic system, which is why things that affect it, like the homocysteine levels.

So homocysteine levels are elevated and increase the risk because that's the methyltransferase system. So the weak link in this field is easy to call in nerve transmission and where the plasma which has come in is that they're involved in the circular release of neurotransmitters very clearly.

And the post mortem data is extremely robust, that you have a very strong linear correlation with the level of plasma genes in the brain, especially gray matter plasma and the omega three plus intelligence and cognitive function.

So we did very large studies with Rush University in Chicago. This has been reproduced in the ADI data sets. It's been reproduced over and over again.

So it's very, very robust information on this. So the question is why how how is this translating into the the behavioral observation that we see in the person in front of us?

And that really comes down to the fact that you see the calling neuron is very different than all the other neurons in the brain. So when you when you have dopamine or you have certain serotonin or noor adrenergic synaptic transmission, the presynaptic neuron releases a neurotransmitter.

So they don't mean, for example, for for movement like Parkinson's, when the don't mean performs its function on the post and haptic receptor, the neurotransmitter gets taken back up into the precinct on your own and gets recycled, ready for the next nerve transmission.

Well, acetylcholine doesn't do that. So you release acetylcholine. Acetylcholine acts on the positive, not the receptor. But then it gets metabolized down to choline and acetate and the choline gets taken back up.

And this is where it becomes a very different scenario.

Plasmalogens, Dementia, and Cholinergic Dysfunction

This is way back when WORTMANN and a bunch of people were trying to find a way, how do we improve cholinergic systems? They tried, you know, massive doses of choline supplementation and a whole bunch of things, right?

People would smell like fish. It would be coming out of their pores. And it was very, very difficult because back then we had the history of L-dopa for Parkinson's, just absolutely miraculous drug.

Right. Like you can give people L-dopa and it's the you know, the awakenings and it's incredible. Right. And so they thought, well, if this works for dopamine and for Parkinson's, if we could find a precursor for coaling, then we should be able get the same observation in Alzheimer's or dementia as we get in Parkinson's.

The problem is, I don't mean of course, it's a very selective molecule. It's only involved in certain cell types. And so the the neurons that take dopamine up are very selective.

Choline is part of every single cell of the human body. It's one of the most it's one of the most core nutrients of all human physiology. And so the question is, you can't just if you just you can't just give people who are calling and expect it to hit the cholinergic neuron system.

And so that's where the problem came in. And then it wasn't until the late nineties, well, actually early 2000, I think it was where people discovered that there was a very specific optic protein called the choline high affinity transporter that brought choline up into acetylcholine neurons.

And that transporter is only on the vesicle. So what was happening in Alzheimer's disease and what we see the shrinkage like Alzheimer's causes or dementia causes neuronal shrinkage doesn't really cause apoptosis until very, very late stage in the disease.

But anyways, the point is, is that if you impair vesicular fusion, not only do you stop the transmission of acetylcholine, you stop the re uptake of calling into those neurons.

And so the plasminogen deficiency actually causes choline starvation of the neurons. And so it has it, it's actually

blocking choline uptake because the uptake mechanism is on the vesicles.

It's not sitting on the presynaptic button. And so this was, this changed. This is where the causation comes in. And this is why homocysteine and these are not in fossil choline, because if your brain has to make acetylcholine from scratch, okay, it makes it from a method called pulsatile ethanol.

And Methyltransferase is the metal transfer system. And when that gets turned on, homocysteine goes up. And so this is why these markers, we have really good markers and they're accurate and they and they're good to measure for a reason because they tell us things.

So anyway, so that's how the causation pathway really came into why these low plasma allergens were selectively or we saw a strong association with dementia right in their social with Parkinson's.

They're also socially with ALS or other neurological diseases. But very, very clearly of all of the neurological diseases, dementia is the strongest association because the acetylcholine neuron has a selective sensitivity to this, the cellular fusion process in the brain.

So that's a long story, but that's how. So that's where, you know, I think wrote the paper back in 2007 about the peripheral ethanol in plasma allergens as a causative factor.

And I went through all of these details and really extensive research in the past and kind of combined it all together. But then that leads us to the next problem of, well, that's great, you know, great answer, what are you going to do about it?

And I thought, well, what the hell? Like, why are these? Why like it's this plasma allergen. This problem has been plaguing us since the seventies. We got these kids with with our Viso, Miller, Connor, Dysplasia.

We got Zale Wilkerson, we've got Luca Dystrophies. We have, you know, neurodevelopmental disorders all associated these little plasma allergens. And there doesn't seem to be any way of getting them in.

And so that's kind of where I've put my medicinal chemistry hat on and synthetic organic chemistry. And still we started looking at how do we get precursors that can restore plasma?

And so now we can do that. Is really good for the environment. Graphic Well, and then you have the worry what's squealing and some other things that go in and it's all saturated so you can't target so you want so you have specific Plasmodium specific purposes.

And what I also discovered was the structure of the precursor was really important. Like you actually had to put the fatty acid that you want on in the center position.

So if I want to restore myelin, I have to put omega nine oleic acid at the center position, but want to increase neurotransmission or neuromuscular function or reverse cholesterol transport for cardiovascular disease.

Then I need to put Omega three DHEA and that will actually be delivered into your cells and allow your cells to make the final product. And that's where the very pure individual precursors are required to deliver very specific outcomes, which is why we have dramatic results.

Yeah. So tell us about the results. What have you seen and what kind of trials have you done on this? So if someone were to take this, what could they expect?

Well, we had some pretty crazy results. So we have a clinic in Canada now. It's up and running and it's a restorative clinic and we use higher doses and things.

But in the general population we have the. So for example, when we look at advanced MRI, so I have more I have multiple sclerosis patients where you can measure the change in their neurovascular coupling.

They find their their functional conductivity and people that have not had lost a vision for 25 years, they're actually getting vision in their eyes for the very first time in 25 years.

I've ALS patients that are now walking, actually walking. I have dementia patients that are that were that were that were under conservatorship that are taking their businesses back.

Yeah. So these are. Dementia patients and Alzheimer's specifically. So when it comes to dementia and Alzheimer's. So Alzheimer's is a disease of it's a pathological diagnosis, right.

So you have two types. So Alzheimer's disease is a challenge because we have Alzheimer's dementia and we have Alzheimer's disease. And they both have the same acronym of E. D.

and they're very, very different things. So if I take a postmortem brain and I measure the pathology of the brain, I can diagnose Alzheimer's disease.

Whether or not that person actually had functional dementia is is separate. So Alzheimer's disease is determined by the accumulation of amyloid plaques in the internal neuronal space and neurofibrillary tangles in typically the dendritic inside the neurons, they were dendrites are attaching to the cell bodies.

And so they actually occur in different places of the brain at different times. So people think that they're together, but they're not. They're actually very different.

The neurofibrillary tangles are entirely driven by the methyltransferase deficiencies. So when you have homocysteine and those and so you have these tauopathy, they're very highly dependent upon this elevated homocysteine or DNA.

Also almost an S-H is highly driven. The all of the nerves are tangle type pathologies and these affect axonal function. And then you have the amyloid plaques which are fundamentally driven by cholesterol transport of the membranes.

And so the APR e genotypes, for example, people that are able e for positive one or two ALS, they'll have higher levels of amyloid. And the amyloid it's the elevation of amyloid in April for carrier that contributes to the risk of dementia.

So if you're an April E for carrier but you do not have elevated amyloid, your genotype has no association with cognition or dementia. So it's the so it will be for puts you at higher risk of amyloid, which in amyloid itself, which is a biomarker of of fossil of of of cholesterol transport.

And so in plasma elegans play the yin and the yang to the people eat. So APOE is your HDL particle of the human brain. So that's Alzheimer's disease. So in Alzheimer's, the Plasminogen precursors, because we've shown this in we published all this work.

If you increase DG plasma allergens, we can reduce amyloid formation and postmortem brain studies. People that have high DG plasma issues in the brain have low amyloid in their brain.

So we have very clearly from the laboratory and in humans the association with DHEA, plasma allergens and amyloid function, which is the mechanism, is actually very well drawn out.

Restoring Plasmalogens with Targeted Precursors

So the so people think of amyloid and they think of the beta secretase pathway and beta secretase is actually relatively small contrary to of the amyloid precursor protein.

So we're getting the weeds on this thing, but the mechanisms of this whole is extremely detailed. Alpha Secretase is the predominant enzyme that breaks down the amyloid precursor protein, and alpha Secretase is highly

dependent upon plasma levels.

So we can turn alpha secretase up and down just by changing plasma levels in the membrane. So people that have high levels of plasma in their brain have high activities of alpha Secretase and the pre and alpha secretase when it's high means AP is processed through that process, it also creates this secretory APC, AP Alpha and that's actually hugely neurogenic.

It's, it's what we use for neurogenesis. So people look this Alzheimer's disease, right? So the all this amyloid is really they try to develop animal models that don't make any amyloid.

Like, for example, they try to block AP and you can't you can't you can't get a viable rat pup with no ATP. So APE is obligate for human survival, mainly in survival.

So anyways, so back to Alzheimer's and Plasminogen precursors fundamentally, I don't have a single situation. Actually, we don't have we don't have cognitive improvement with plasma.

I just don't want seriously, I don't have a single situation and people that I work with. And how long does someone need to take them at one dose? Now that's the where we learn.

So we first clinical our publication with Dr. Jordan's group in Santa monica. We didn't escalating dose, we started at one gram, basically 900 milligrams and then we which just pharmacokinetic study basically to say dose escalation see if you can reproduce in humans what we had done in animal studies in the past.

And so we went from from 900 milligrams to 800 milligrams to 3600 milligrams. So we just did dose escalation and we saw we had a dose dependent increase in plasma allergens, but we actually saw in 22 people a statistically significant improvement in cognition in four months.

Okay. And we now see with higher doses and the mobility improvements like the neuromuscular junction improvements were actually greater than the cognitive improvements.

And these are clinical dementia rating two subjects. We had 75% of the people with mild to moderate dementia improved an entire score on the clinical dementia rating in less than four months, and that's just in a dose escalation study.

So now when we deal with it, we we do a little more higher doses. So we have children that are on ten meals a day, you know, they're for the Luca Dystrophies and that we've they're no longer terminal.

For example, these children are growing and their brains are developing again. So weakness. And so for dementia patients, I just want to pivot. We're on dementia. Yeah.

So it says at the Alzheimer's Summit. And so I want people to really understand and and so they can get the practical application of this and get the benefits of it right.

So what would they need to be taking in for how long before they could expect to see a change? So if we go to higher doses, if you go up to about five, even 5 to 10 mils for dementia, where people that actually have an actual clinical representation of dementia and you go to a relatively high dose between five and ten miles per day, you will see usually improvements within a couple of weeks, actually, certainly.

And so normally I tell people, invest in one month of high dose therapy and then you can scale back from there. Okay, so then. You can recover, please, and then you can go into a maintenance mode.

Which is a reversal of how we started this. We thought we started saying, okay, let's, let's do dose escalation, right? Let's start small and go. So we now we kind of go up, you know, obviously we go carefully with people that make sure they don't have any, you know, because it is it can be quite stimulatory like for an 88.

Yeah. Tell us about like side effects. So we do we have to worry you. Know these might effects we have it really in people with bipolar or ADHD. Okay.

So those individuals we typically treat with the omega nine plus mountains for the myelination. So because you're is quite stimulatory like the omega three or people wake up that you start seeing them, they're engaged, they're moving.

And so if you have any, you know, if you have anxiety or you have any kind of ADHD tendencies, go a little slower because. It can be stimulating. It's very stimulating. Yeah.

And then that's kind of typically tell people, take it in the morning, early afternoon, and by lunchtime ish we switch from the new way to three to the omega nine for nighttime.

So make it nine, which is we use for brain inflammation, concussion, stroke, all autism type programs. But for dementia, seriously, it's we've learned now that if we ramp up quickly, you start seeing and you and you know, we can we see it on MRI quite robustly like that the neurovascular coupling improves the functional MRI like the board, like we use a blood oxygenation level dependent scanning to look at the connectivity of the nucleus, bizarre and other cortical regions that very reproducibly improves.

And so. I mean. Yeah, so it's, it really, it really does work. And then we use plasma allergens as a it's kind of like now that I got your attention, let's fix a few more things while we're at it, right?

Like, it's not the only thing in the world, it's a pretty big hammer. Like, it really is something that I have to say. Like as a scientist, I'm pretty shocked myself.

Like, you don't really you you don't program them to see these effects. Like you're really not programed to see people just wake up like we have people that haven't walked for four years in literally six weeks.

They're dancing with their parents, with their little I guess this this is really kind of crazy stuff like and and it's crazy when it happens once, right?

Then it happens twice and then obviously it starts happening quite reproducibly. Now we've been able to find an omega nine. You know, getting fossil cooling in the brain was critical.

So we know blended with we have a fossil cooling blend that really helps that part of the brain. And then I'm a big fan of other core precursors, like an testing carnitine.

CoQ10 So when we start this process, the plasma engine gets people hopeful, okay, things are happening.

Clinical Results and Dosing for Dementia

And then we say, okay, well, let's not stop there. Let's, let's fix a few other things while we're at it, okay? And obviously, you know, you have your lifestyle and other health issues and try to remove things from your environment as much as possible.

But at least we can get them. They get enough of a signal that they say, okay, you know what, this is serious and we can go. And then you can you know, we have a blood test that can measure these things.

They show the change in plasma allergens. You can also look at other core components like your HDL levels, and you're getting your triglycerides in the proper space, getting your homocysteine methyltransferase system properly, reducing the C-reactive protein and the oxidative stress markers.

Like one of the things that we found in the clinical trial that we published with Dr. Jordan's group was that we had a dose dependent decrease in Mount Aldehydes.

Oxidative stress markers went down, catalase went up. And so we know biochemically we have a very logical, systematic, mathematical, if you will, approach to this.

And the only really difference is now personal individuals will have their own like it's food, like we're not talking and plus plasminogen because we're dealing with bulk like it's not a trace level.

You you you need actual full material and if you take them in the fasting state to get past the blood brain barrier. So we like the whole concept of how we move things on kind of microns through the blood supply.

So the supply, your muscles and your brain. So there's, there's, there's different systematic ways that can improve it. But for most people, I say just take it in the morning on an empty stomach, if you can, with a little bit of protein or oil or fat, you know, kind of a a semi keto breakfast, and then you're going to get your best results.

And in those situations. Amazing. Well, this is so informative and so, so hopeful and really straight forward. I just want to kind of recap that. So if someone is going to take this plasma and start trying them, you know, of course, if you hear you're listening to this, you've already heard a bunch of other talks about Dr.

Reticence approach. And so you're doing well is exactly what Dr. Goodenough is suggesting, right? You incorporate knowing or considering incorporating the program sciences supplementation into an overall comprehensive, lifestyle based intervention for optimizing cognition at whatever stage you're at.

And so and you have said you would take a big of 5 to 10 milliliters in the morning because it can be a bit stimulating. In fact, that's part of what we want.

We want to wake the brain back up. Right. And then you do that for about a month or so. Try that for about a month, see how it goes, and then move into more of a maintenance dose.

What would a maintenance dose look like? Oh, and also you would be taking on an empty stomach so that it can be absorbed and then shuttled to the right places to get them.

That's ideal. Like if you can't, you know, for GI, obviously you the you can tell you the science, right? But everyone has to it has to work in your life, right?

You have to be able to because nothing is worse for compliance will will eliminate almost any good effect. So, you know, people have to be able to actually put it in their lifestyle.

And so that's kind of where it is. And go slow, start with a mil, go to two or three things. Our patients experience diarrhea. I know with it some of the fat soluble things.

So certainly MCT oils and coconut oils we see as people try to add those, they can be running off to the bathroom and that can be really frustrating for caregivers and for people suffering with dementia.

It's just a whole nother level of complexity and and mess. So do you. We actually don't see that at all. We don't actually. It's it's interesting. We have a lot of work in the gut world and you know, and I use some peptides for gut and so we use also humanoids, but the plasma allergens really have improved gut function in terms of people also to colitis.

And so if anything there it, it's only constipated, but it's, it, it's certainly not diarrhea. Okay, good. So have to worry about that more the the activity and the alertness.

That's fantastic. Okay. And then if you were to go down to a maintenance mode, what is that typically look like really. For people in the early like so as you for all of us in our over 50 crowd, if you will, in that three mile range is ideal typically six capsules in the morning of neuro.

Six couples at night of glia is typically the best long term. So when I showed my own brain, like I was able to

reverse 15 years of brain aging with my own brain using high dose plasma allergens, and we can look at volumetric white matter microstructure.

And so there are there are ability to actually restore function. And then if we want to kind of keep pushing the envelope a little bit, we need to you know, it's just it's a matter of getting materials above a certain pharmacokinetic threshold, fundamentally.

And then, you know, we have about 2000 doctors in our network. So we have lots of experience. And our roundtables like the stories among the physicians and the different situations.

And I'm like, we it's not just like the dementia is a is a huge deal of course. And that's our you know, it's it's it's it's the most obvious improvement.

Even normal people like you people, people come back and say, I just remember numbers better and they remember trivia better and they knew. So it is an interesting thing.

People with long COVID that are getting just they can't get the brain the brain fog that people are experiencing. Those are things that so it's quite a big, diverse group.

So that's why dementia itself, you have what, Alzheimer's or vascular dementia or Lewy body dementia or even, you know, post-concussion dementia type things.

These all have different pathways, but in the end they still affect this synaptic system. And that's that that system can be affected by acute inflammation.

It can be affected by nutritional deprivation. It can be affected by failure of having right methyltransferase system like B12 deficiencies.

Maintenance, Side Effects, and Broader Applications

So there's, there's more than one way for that neurological system to become impaired. But restoring that system with the core components is actually we finally have tools that are logical, like we're not guessing.

It's not it's not a shotgun approach anymore and a very reproducible works. So you have written a book called Breaking Alzheimer's. And so anyone who's interested in learning more from Dr. Goodenowe, you go into this in much more detail in this book, it's a great read.

And so tell everyone where they can find out more about you. The book and Prodrome Sciences. So at prodrome.com is where you can find supplements, blood testing, the roundtable access and all that kind of stuff.

And then at drgoodenowe.com is where we have the perpetual health are clinics more of our our research programs advanced MRI technologies. So those things are more at the Dr. Goodenowe, I have a charity for rare disease in children.

So Luca just we treat all these children around the world for free of charge. And so that's all found on drgoodenowe.com. Oh, fantastic. Well, thank you so much, Dr. Goodenowe, for your time today.

But just the work, the incredible and very unique work that you're doing in the world and the charity, I didn't realize that. Thank you for doing that.

Looks really special and amazing. Yes, amazing. Children, it's like these. Look at this trapeze and Catherine's disease and Rett Syndrome and big rise.

Amelia Connor displays a punk tattoo. And so, you know it's there. Yeah. So it's it's very exciting and it's just amazing to see these children come back.

What a massive impact you can have on the next generation. We're getting there. And autism is a huge program for us. I guess it's related that. You know I think.

Not nothing out of the glen typically so. Okay interesting I think of autism and dementia as kind of flip sides of the same coin right when it's happening early in life and the other later in life.

But absolutely. Correct. That's exactly right. You spend yeah it's a just myelination. Autism is an inflammatory mediated this myelination event. And so and then later on in life we have the same thing.

We can have the the problem is that when they're a child, they haven't built the plasmalogen stores up their myelination that we really do massive myelination for the first six eight years of her life and that's why autism doesn't really occur once a child.

If a child doesn't get autism by age six, it's quite rare for them to get autism after age six because they've built up sufficient stores. But then again, fast forward to our sixties and seventies and so we start losing like we it's exactly.

You're 100% right, Dr. Sandison. It's a it's kind of a a bell curve, right? If this inflammation happens early in your life, in your brain, it prevents the myelination from occurring.

If it happens later in life, it causes an accelerated aging process fundamentally. And then, you know, cognition is one of those early signs of we're aging, if you will, from a very simple perspective.

Well, how exciting to have things that we can do to address it. So thank you again for your time, your expertize and being with us here today. Thank you very much, Dr. Sandison. I was happy to be here.