GABA, Genetics, and Brain Inflammation with Bob Miller Transcript

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Introduction to GABA and chloride balance

Well, what's interesting, people tend to think of GABA as being this relaxing substance. And it is, unless some other conditions make it, not allowed to do that.

If different conditions exist, we're going to talk about how that happens, and the chloride flows out, it becomes excitatory. Interesting. So GABAA does the same thing.

It doesn't change. it opens the GABC receptor site. and it's the Chloride balance in the neuron that decides whether it will be relaxing, or excitatory.

Hi, welcome to the Maps webinar series, Healing Tomorrow's Future. We are thrilled to be bringing you this series packed with valuable information and education within our community.

My name is Honey Rinusella and I'm the Executive Director of MAPS, the Medical Academy of Pediatrics and Special Needs. Within these webinars, we're able to empower clinicians on the knowledge and tools to support patients facing a variety of health challenges.

For more information on MAPS or to register for a conference, please visit us at www.medmaps.org. Thanks for joining us on this journey towards a brighter, healthier future.

Well, hello there, Dr. Miller. You're one of my favorite lecturers. I really always learn so much from you. Thank you! I love your maps and how you draw things out so that we can use them as clinicians.

So, I'm grateful for that. Oh, good! Glad you like them. Yes! So I am excited to talk a little bit more about GABA, since that's what today's topic was, so explain what GABa does to the neuron.

Well, what's interesting, people tend to think of GABA as being this relaxing substance. And it is, unless some other conditions make it, not allowed to do that.

So a lot of people don't know what GABa does.

MAPS webinar welcome and speaker introductions

It has one job, and one only. There's what is called the GABAR receptor site. and it opens up the GABARreceptor site, that's all it does, it can't do anything else.

so when it that, two things can happen. Chloride can flow in to the neuron, which has a negative charge. Right. Or chloride can slow out. And that's it.

That's all it does. So what happens is, if conditions are right and the GABA opens up the gate and chloride flows in as it goes over the gradient, it's very relaxing.

If different conditions exist, and we're going to talk about how that happens, And the chloride flow out, It becomes excitatory. Interesting. So GABA does the same thing.

It doesn't change. it opens the GABO receptor site. And it's the chloride balance in the neuron that decides whether it is going to be relaxing or excitatory.

Interesting, okay. Yes. How then does flow of chloride, how is that determined? Well, it's rather complex. OK, so what happens is there's an enzyme called NKCC1 that takes extra chloride into the cell and KCC2 that take

chloride out.

So we have to have lower levels of chloride in the neuron most of the time so that when the receptor opens up it flows in and it relaxes. There's the million dollar question, how that got messed up.

Yeah. So that's what we're going to talk about here today, how that chloride balance gets disrupted. I'm thinking electromagnetic fields. Am I on the right track?

You're on right. What else? So let's first talk something else called microglia. Yes, please, let's. So microglia are glial cells that sit in the brain and the central nervous system.

How GABA becomes relaxing or excitatory

And they're your friends, unless they are not your friend. Understood. What they do, they on guard. Now, most of the time they describe this as M2 of micrograph.

It's chill, it's taking out toxins, its stimulating an enzyme called interleukin 10 that's relaxing. But when notice is a threat, it says, oh, somebody's here.

We got to take care of this. So if we've got a virus or bacteria, thank goodness it's there. Because what it does, It turns on five different enzymes that are inflammatory.

And those are TNF-A, interleukin-6, NF-kB, NLRP-3, which makes the inflammations, NOX NADPH oxidase that makes superoxide, and the INOS enzyme that make large amounts of nitric oxide.

So when that happens, the inflammation begins to kill the pathogen. Good thing as long as there's a pathogen there. But if it's being faked out that there is a threat when there isn't, that's when we have a prior.

So your microglia are just sitting there protecting you, but if they get the wrong signals, they go crazy. And then what they do, the NF-kB will actually imbalance that NKCC1 and the KCC2, pushing it more, Kcc2 that takes the chloride out, weakening it.

the NKCC-1 strengthening. So an endocampal B gets upregulated, that's what it does. Then through what are called inflammasomes, which I spoke about at the last conference in the fall, we make interleukin 18 and interlukan 1 beta, they also do the same thing.

They mess with your chloride balance inside the cell. So again, it's all part of the immune system's response, but now we have one little problem in this world we're living in today.

Yes, we do. And that's called environmental toxins. OK. So one of my favorite jokes when I do consults with people, I tell them when was born in 1954, or I was borne on a different world.

Yes. We didn't have high fructose cornsear. That's right. we did not have cell fans, We did. Not have our animals growth hormones. When you have glyphosate, when you know COVID, you didn' have Lyme disease and mold was there.

But mold seems to be getting stronger. for some reason. There's all kinds of theories on that. One is EMF, but we don't know. But so when you get exposed to all these environmental toxins, and then you've got a genetic predisposition, proverbial perfect storm.

Perfect storm, yeah. So that's what throws it off.

Microglia, inflammation, and chloride disruption

Now, the genetic component to this. So why are some people being impacted more than others? You know, for

example, two people can live in a moldy house and one says, oh my goodness, this mold is making me so sick.

And somebody else says well, you must be imagining things because I don't feel anything. Exactly. So when that happens, then, there's genetic factors on the microglia.

There's an enzyme called Nrf2. And Nrrf 2 senses when there is inflammation and says, whoa, oh, if there was inflammation here, I need to start turning on my antioxidants.

Well, unfortunately, you can have what are called SNPs, single nucleotide polymorphisms, that you inherit from either mother or father or both. And the instructions to make the Nrf2 isn't quite as good as it should be.

Not that disastrous, but it may only be 70-80% if one parent gives a SNMP, 30-40% of both parents give a snMP. So if you've got a weak NRF2, it's not holding back microglia M1.

And Nrrf 2 also supports microgla M2. Now, if this isn't complicated enough, let's go a little bit farther. This is why I like your diagram. Yes, exactly.

Somebody that's listening to this, they may say this is like Chinese. That's why you need the diagram, fool to our best. So there's an enzyme called KEEP1.

And KEEP1 suppresses Nrf2 so that it doesn't get too carried away. Well, there is one snip that if you get it, the KeeP 1 is stronger. It's going to suppress Nrrf 2 even more than what it should.

If you get it from one parent, that's not so good. But the people who we see struggling the most, both parents give a snip on keep one. So therefore, the NERF 2 is asleep with the switch.

Wow. The inflammation comes along and Nerv 2 says, one inflammation. Right. And it doesn't hold it bad. Then there's another process called mTOR, mammalian tardive rapamycin, the growth of new cells.

If we didn't have that the sperm and the egg would never become the baby. The baby never becomes the adult. We need mTor for cell growth. So your cells constantly are dividing.

But then there is a process call autophagy where we take dead cells and recycle them back into the new ones. Well, guess what? We can have genetic SNPs, but the mTOR is overactive.

Now, on top of that, you know, we're giving our animals growth hormones so they get fatter faster. We're going to look back at that on Sunday and say, what were we thinking?

Back in the 1800s, girls got their menstrual cycles around 18 and 19. Then it was 12 and 13. Now I talk to pediatricians, it's eight and nine years old.

When I was a kid, 12-year-old girls were flat-chested little girls. Sometimes at 12, they look like 16- year-olds because we're stimulating amateur. So what are we doing?

We're now environmentally stimulating the enzyme that creates mTOR. And it's important to know that mTor doesn't cause cancer, but it is a cell replicator.

So if you would happen to get cancer it will replicate the cancer cells as well. So that's why some alternative, maybe mainstream, some treatments for cancer is mTOR inhibitors.

There's a drug called rapamycin that inhibits it. So now if we have mtor overactive, Nrf2 weak, environmental factors, here we go. Wow. And it's overstimulated.

Yep. The chloride balance gets off. Then you wonder why this child is sensitive to sounds, why they're sensitive, to touch, they can't focus, are irritable, can sleep, get agitated quickly.

Because the GABA is doing what it is supposed to do.

Genetics, NRF2, KEAP1, and mTOR

Open the chloride channel. When that chloride is off, it becomes excitatory. Now, what happens is when a baby is born, they're normally in that excititory state to have growth.

But over time, its supposed to move towards the KCC2. But when microglia is upset, stimulated by mycotoxins or other environmental factors, they never get there.

And you have, you know, either ADD, ADHD, autism, all of those things happens when your GABA is not relaxing. So that's why many people learn about GABO and somebody says, well, I'm going to take some GABo.

That's right. Then they get worse. Right. What is going on? GABAs supposed to be relaxing, but even when they take GABI, it does what it's supposed But with the GABA receptor site, that's what I do.

And if the chloride is off, there we can. Now, the other thing that will stimulate this, which I didn't talk about today because we just didn' have time, but we're very interested in what's happening with arachidonic acid.

So what happens is arachidonic acid is your friend when it's in the cell membrane. Right. It gives structure. Yes. But interestingly, there's an enzyme called PLA2.

We've known about this for a long time. And it was probably just in last couple of months that we were like, uh-oh. PL A2, yes. I've been studying that one.

Yeah, so we're doing a lot of research on that right now. So PL-A-2 takes the aracidic acid, pulls it out of the Then an enzyme called COX-2 turns into what's called prostaglandins.

And then it can also make what are called leukotrienes. Leukotrien is what is behind asthma. But these prostoglandin, if they're excessive, they throw off the balance of chloride inside the cell.

Oh my. Oh wow. Yeah. So then what happens is that there's other things that will stimulate that PLA too, including intracellular calcium. There's an enzyme called NMDA that brings the, when it's stimulated by glutamate and glycine, and when its overstimulated, it will bring in extra calcium and that stimulates the sodium to go in to the neuron and throw things off.

And calcium will also stimulate PLA2. And Calcium will stimulate NLRP3, which we spoke about earlier, Which stimulates the interleukin 18. I mean, the feedback groups that we've just described here.

If anybody's following all this, God bless me if they are, because it's complicated. They can just say, no, wait a minute, that sounds like a feedback group.

That sounds another feedback. So your EMF and other things, homocysteine, glyphosate, stimulate an NMDA. then we can have problems with our dopamine. So we need dopamine, some people are low in dopamine but if you have like the COMT and DBH and problems your dopamine receptors, the dopamine stimulates the PLA2.

Now, what happens is we also need something called DHA. You know, like your megathruism. Dha slows down the arachidamic acid being released and the PLA too.

Well, as you can imagine, I'm going to tell you there's genetic mutations on enzymes called FADS1, Fads2, ELOVL2 that limit your DHa. So some of those people take fish oils and they feel worse because they're not converting them.

In that case, they need to take like an algae-based D Ha. Interesting. Like a krill oil, would that be an algae-based? I like to use, well krills still a fish, so I use Nordic Naturals meets an Algae DHA.

Interesting, okay. Then we have cortisol. Oh my goodness. So cortisol calms this all down. Well, guess what? Who controls that? Nerve two, keep one. It's so complex.

Yes. If you've got problems up there, and then to make it more complex, there's something called the sirtuins, particularly srt1 that controls a lot of this.

And it needs a supply of NAD.

Arachidonic acid, PLA2, and DHA balance

if we got some issues that we don't have enough N. A. D., we have a problem. Since we're making it complicated, why don't we go to the next step? Let's do it.

We're in the right place. The best practitioners ever are listening to this. So a lot of people are talking about the wonders of NAD. Yeah. Okay. And my goodness, it makes the NEDH that makes your electron transport chain.

Makes your NDPH, that recycles your glutathione. That's right. It's what the sirtuins need. You think the more of this, the better, right? However, There's an enzyme called CD38 that will take NAD and take 100 molecules of it and make intracellular calcium to stimulate more inflammation.

Guess what it's triggered by? Mycotoxins, EMF, Lyme disease. And then amplified, if you've got those genetic mutations, the TNF-A or NF-kappa-B are upregulated.

Wow. Then it stealing that NAD to make inflammation. That's right. So if somebody learns about Nad, it like, wow, cool stuff. Let me take some NAd. If that's occurring, they feel worse.

Yep. And they're like, no matter what I do, it doesn't work. So what we have to do in those situations is first see what's happening, because NAD also turns into NEDH, which is your electrons for the electron transport chain.

But there's an enzyme called NQ01, I put on my top 10 list of enzymes to look at. Okay. Because Nq01 takes NedH back to Nod+. And if you don't do that, those electrons build up at complex one of the electron transport chain, they can't process through and they make this free radical superoxide.

So rather than making ATP, it makes the free-radical superoxone. And damaging the mitochondrial membrane. Yes. Okay. Interesting. I didn't get a chance to talk about this today, but we've coined a new phrase, and that is cellular CPR.

Yeah, I like it. Construct, protect, restore the bilayer. So that's the direction we're going to be going next, that you're only as healthy as your bilayered.

Now, what happens when microglia and all these things get upregulated? Through NLRP3, we make gas germans that punch holes in the cell membrane. Through NOx and INOS, We make lipid peroxides that punches holes into cell membranes.

That's right. So we're getting hammered from every angle. Yes. Wow. And then that inflammation from that stimulates PLA2. Yeah. Just feedback loop after feedback, loop occurs here.

Are you ready for one more feedback? I'm ready for you to tell me how to fix this, but I'll wait for one more first. Go ahead. So when we make something called peroxanitride, it stimulates mast cells, and that creates histamine.

That's right. And that histamin will mess with the uranine-anziotensin system, which makes something call angiotencin-2. Yes. Yep. The ACE-II enzyme turns that into angiotensine 1-7.

Guess what COVID did? It slightly damaged ACE 2. So now rather than going to the angiotensin 1-7 that's anti-inflammatory, it goes down to stimulate more mast cells and another feedback loop.

So what do you do? This is the moment we've all been waiting for because all of this is so complex. But when you take it all and you just say, okay, but how can we just course correct this?

What would it be? Sure. Well, firstly, you have to look at your genetics. And that's why we created functional genomic analysis. It's software that analyzes genetic data.

So when you work with us, you get a genetic test, he sent it to a company called Sampled. They pull out the data, they send it back to my company. So I'm kind of hands off.

I just have bought the chip and I don't have a lab that does the DNA testing. Sure.

NAD, CD38, and mitochondrial stress

And I take the test data and put it into the software. It's taken me 12 years and seven and a half million dollars to build the Wow. That analyzes the data.

So a lot of people get genetic tests and it's like, oh, I've got MTHFR and COMT. Right. Sweet. It's not even the tippity. I'm with you. Yeah. What we have to do is we to look at these pathways, see where the weakness is.

So I've been doing these shows for a long time. And one of the feedback I get from doctors is that this is really cool, Bob. This is the answer, but this was too much to learn and I'm not going to do it.

I need something easy. Right. So we're in the process at the time of this recording, we are in process of making an executive report. they will identify the snips that are mutated, that're evidence-based, because we only look at evidence based snps.

It'll be a piece of paper you can print out, so you don't have to take courses, you do not have study, and it's like, boom, there it is. We're going to identify, again, from that CPR, where the construction is a problem, whether damage is the problem or the repairs are.

So we're in the process now putting the logic, it's all there right now, but we are putting in logic to make it simpler. And that is it will tell you, you know what?

It looks as a lipid peroxidation as your big issue. Maybe you want to start here. Or maybe your TNFA is upregulated. Maybe your arachidatic acid is out of control.

Maybe you want to start here. So we're going to hold hands, so to speak, and give them guidance. And we are hoping to have that ready by maybe April, May of this year.

But I mean, it's all there. The maps are there so you can look visually at the map. That's lovely. You can even look at DNA. We're trying to simplify it.

And then you'll have solutions right next to it. If you want to address this issue, here's some nutrients that are evidence-based to handle that. Yes, exactly.

So if TNF-A is upregulated, we work with, and again, I don't have a supplement company. I just worked with a company called Compound and Nutrients. They're separate from me.

And I said, can you make this product? We're going to call it Plax that calms down T and F-a. so if it's T N F A, We have PlaX. if It's Interleukin-6, we have Cytocom- 6. If it's peroxonitri, we have peroxyblast.

If its NORP3, you have a product called Zome. So you identify where you need to go in. It's no longer a guessing game. Will it address DNA adducts and things more epigenetically, or are we going to go with the fact that your weaknesses, when addressed, will then prevent the epigenex from being as problematic?

Oh, absolutely. Now, on the other hand, if you've got mycotoxins, the primary way to get that out is through glucuronidation. That's right. And we do have a pathway on mold detox.

So this doesn't tell you whether you have mold. You have to do the regular mold test to define that. But you can see if your weakness is glucoronodation, that we need to go here.

You're predisposed to having a bigger problem. Right. So that's why two people could live in the same house. One person says, the mold is killing me. The other one says I don't feel a thing.

It's the glucuronidation pathway that might be weak that allows that to happen. And then we also look at things like oxalates. Then we look the whole NLRP3, is that operative?

So you can see where the problem is. And then we actually, one of our most proud things we've ever come up with now is a product called M2 support that's designed to support NERF2.

We can keep, we can MTOR if it's too strong and many other nutrients in there that support the balance of M1 and M 2. I think what we're going to find, I heard the analogy, for a forest fire to occur, you need a spark and then you the wind and the fuel.

and microglia, we believe, is the wind and the fear. So when you got that spark, which could be ammonia, it could a arachidamic acid, that's the spark.

Cell membrane damage and inflammatory feedback loops

But then the M1 is what pushes it down through five inflammatory enzymes. So we now believe that. Really fascinating. We believe, That's a key point here.

That that microglia that was there to defend us is now overactive due to all the environmental stuff. Someday we're going to look back and say, what did we do?

You know high freak toast corn syrup glyphosate, you know, everybody's got a smart house. I have teased already I've told people you I'm gonna make t-shirts that say be really smart live in a dumb house Exactly right.

Yeah, and if anybody come to my office My computer is connected by ethernet. Yep wired microphone wired mouse I even hook my cell phone up to the internet so that my cellphone isn't I didn't know you could do that.

You can do it. Yeah, you get a wire that you can connect your cell phone. So what I said… You could hardwire your cellphone too. Hardwire my cellphone to the internet.

Brilliant. See, hard wire everything. He turned the Wi-Fi off at night and maybe put a timer on it so it's easy. And these things combined with dealing with the genetic…

Right. Right, yeah. For example, for Wi Fi, we made a product called Calcicolm. Okay. Which is designed to slow down the NMDA receptor so that it is not overactive.

And then we have a product called Plax that's designed to calm down the PLA tunings. So if you were addressing EMF as the main culprit… Calcicom. That's the product.

What's it called again? Calcicom. Calming down, because it's a calcium. Running down. It's basically everything that holds back an NMDA receptor. I feel like I wish I had that with me here today, CALCICOM.

You know, with all the EMFs at a conference, I think that that is a universal need. Sure. Yeah. And I mean, great hotel here, but you know the headboard has all these wires.

They all do, right? You don't want to be within two feet of the… So you're putting your head right, Liz. Okay,

wonderful. Wonderful. People wonder why they can't sleep at their conference.

What's interesting, because I've been studying the PLA2 from Dr. Leo Gallin's work with spike protein and how it's causing a chronic elevation of PLa2.

So that sparked my interest when you brought that in. Because you don't hear too many people talking about PL2, right? I'm going to talk about that at conferences this fall.

I am looking forward to it. Then we're going be talking how the spike proteins combines with, you know, creates all the mast cell activation. That's right.

But it is through this pathway that I just define. Yeah. So the spike protein stimulates the microglia, then that stimulades the inflammation. That superoxide and peroxide nitride stimulate PLA2, and we're on one loop-de-loop.

And then if your insulin is up, you're aggravating PL A2 further. Yeah. I'm speaking about spike 14 on Saturday, so my head has been into that same pathway.

We'll hear that one, yes. And then, as I said, it interferes with your ACE2, which then just makes more inflammation, and around we go. And that's why I think we see these conditions of, you know, chronic COVID, long-haul COVID.

Practical interventions for EMF, mold, and toxins

Because once this thing starts spiraling, And then the senescent cells are, I think they're also impacting CD38, which you mentioned, is one of the reasons that it creates the neighboring cells and the autophagy just gets stopped and blocked by it.

So it's just really interesting. I want to look at your pathway and delve into it even further because it is all connected. Everything is connected? It's really, really fascinating.

Yes. Anything else we need to know going forward that you didn't share yet? Well, what you can do environmentally, as you said, stay away from EMF, no high fructose corn syrup, make sure your house doesn't have mold.

Can't tell you how many times I measure people's mold in their urine. They've got mold and they say, but there's no mold my house. Yeah. You're okay. That's the most common thing I hear.

I think it's because it stresses patients out the Most. Right. They see, oh my gosh, you know, how do you even begin? And I Think that it doesn't have to be so bad.

Sometimes it s just an event or a crawl space. It doesn t mean a whole tear down. And think people are afraid to look. Right, and then also maybe an air purifier in the bedroom.

Yeah, exactly. And just look around a little bit. We've seen children recover just by switching bedrooms. Right. Sometimes, you know, turning the vent off in their room and just sealing off one room.

Sure. Well, mycotoxins stimulate the NF kappa B that stimulates the PLA2. That's right. Back to the PLA2? Yeah. Oh. It's really refreshing to see somebody who's into the PIA 2 because most people are like, what?

I really love the geeky stuff. You just took the words out of my mouth. We speak the same language. You're a fellow geek here. I hope everyone listening speaks this language, yeah.

Some people may be like, I have no idea what they're just saying. If not, you buy the program, get the one-page printout, and now you also can speak that language?

Right. So if we're speaking to doctors, what we want to do is we go to functionalgenomicanalysis.com, we get a free trial. Our staff works with them. If somebody's watching this who's just had a kid they want help, I still do consulting in my office, Tree of Life Health, so tolhealth.com.

Order the kit, do a consult, we do it on Zoom. And again, people don't have to worry. We're not going to tell you we've got this weird genetic that's going cause fill-in-the-blank disease.

Right. Something that you can modify. One of my favorite jokes is I have no bad news. The only bad things I could ever have would be like, I don't see a thing to help you with.

Exactly. So it's not catastrophic if you don' turn your EPA into DHA. It's no catastrophic, if your NERF 2 is a little weak. You know, it is better to know it than to compensate.

Because ignorance is not glist. So knowing where the problem is and compensating. And I marvel at, you know, 20, 25 years ago, we had no clue. We just threw things at it and let's see if that works, see that that worked.

But the precision that we now have, and even though I do this all day, I'm like, this is really cool. It is. Really cool to see exactly where we need to intervene.

I think that children's health is way more complex than it used to be. It's harder to recover the kids than I was even three, four years ago. And I because of that, practitioners need more tools.

They need to dig deeper, and I this is an avenue to do that. Sure. Thank you. Yeah. Well, in the old days, you know, give a little V12, those things kind of worked.

And people got better with very simple things. Even just a methylated B vitamin back in the day. Now, the methylation is making it worse. You have to give adenosine.

So it's just way more complicated now because there's way, more like you said, all those things pouring into the system or you just can't throw a few things in and expect it to work anymore.

Yeah. So you can see 20 kids in a row, 20 different protocols. Exactly. So if somebody said, what do you do for autism? It's like, I don't have a protocol.

And if you have so many things that are going to come up with the protocol, it might work 15% of the time.

Testing, personalized protocols, and closing advice

What have you seen have the greatest impact of all the things you would use to target in general, that most patterns show more benefit? I would have to say balancing the NAD.

Yeah, so we have about 80% of our people do a finger prick test that measures the NAD, NEDH, and NDPH. And then… Is that through your company, the Finger Stick Tech?

No, US Biotech does that. Oh, I think they're here. They're there. US biotec does it. So we put a little plug in for them. So, U. S. Biotech does that.

There's a company called OmegaQuant that looks at the arachidonic acid. So the NAD and the Omegaquant together are quite helpful. And then you've got, you know, Mosaic, who does the glyphosate test and does…

Those guys owe me a free dinner, don't they, for this? You're going to have to get a few. Yeah. So they do the mycotoxin test. They'll also do O test to look for oxalates.

That's one of my favorite tests. I get so much information out of that. So if you've got oxalates, that'll drive this thing too. And there's so many things.

Again, what happens is people think, oh, I'm going to eat healthy now. I'll improve my diet. They start doing green drinks, smoothies with spinach, kale, and beets.

It's like, What's happening? I am worse. Yeah. I don't eat, I stop my junk, eat healthy and I'm worse because they're just making all those oxalates. I think the one thing I got out of this talk that was really helpful is the fact that you have to listen to your body and you to have listen your patients.

When someone says this thing made me worse and your like, how can that make you worse? You know, look at the science you brought today that we didn't know yesterday.

And like for instance sugar, i've seen people recover by taking pure sugar because of a secondary porphyria. And they'll be like, oh, wow, I do feel better when I take share, but I don't eat it because it gets bad for me.

Not that that's the treatment I would use, right. But it's just so interesting. If you pay attention to the body, it gives you clues. It does. Gives you a really good clue.

And that is why somebody takes fish oils and they feel worse. Yeah, well, we know the fish wells are good for you. Well, not if you got the FADS-1 and Fads-2.

Sometimes we see people that are homozygous, both mother and father, give a snip on every evidence based snip, on Fad's one and fads two. That's incredible.

And it's like you and algae-based DHA are friends for life. Fascinating. Yeah, and I often tell people we have good news and bad news. The good new is we can compensate.

the bad new's is, we could compensate And I think just to remember there's always hope. Yes, there is. There's ALWAYS hope! There is! Thank you. That the bottom line, that there always is hope, but you have to do a little bit of digging, a bit a biochemistry.

I'm gonna have to listen to this one over and over again until I get everything you said in. Thank you. Well, you're a lot of fun.