Introduction and sponsor messages

Welcome back folks. I'm Natalie Nim, your host. Powerful question. What happens when environmental toxicity stops being theoretical and becomes personal?

Well, you're going to find out. In this episode, I sit down with David Roberts and John Gildea to explore how a cancer diagnosis sparked the creation of a stabilized form of sulforaphane.

And what they've since uncovered about inflammation, detoxification pathways and modern toxin exposure. If you care about longevity and protecting your biology in the toxic world, this conversation connects some really powerful dots.

There's a special offer for the next week for longevity loyal listeners. So if this resonates for you and you want to get your hands on this product, you can get an exclusive 25% off at mara-labs.com with code NAT at checkout.

Now the exclusive offer ends on April 7th. After that, the discount will revert to the standard 15% offset. All is not lost if you don't listen in the first week.

But if you make it in on the first week, you will get that very special discount. Now, this episode is brought to you by Vitaly, which is the GHK Copper Skin Care, Troscriptions with Trozee, and Liver Complex from Level Up Health, all with incredible special offers just for you, the listeners.

All you've got to do to get to them is check out the show notes below for details. And now, enjoy the episode. John Gildea, David Roberts, welcome to the Show.

It is a pleasure to have you here today. Thanks for having us. Great to be here. Yeah. Appreciate the invitation. At last. So I'm going to actually direct questions to each one of you initially.

That way people can kind of get their heads around who they're talking to. Let's talk about just David, and this is we're going alphabetical, no particular order.

Before MaraLabs existed, what personal health or life experience first made you realize that Environmental toxicity wasn't theoretical. It's actually personal.

And, you know, we talked about this the other day. We ran into, not knowingly, ran in to each other many, many. You've been at this for a while. Yeah.

Even before, all the things we talk about now were so top of mind. I mean, I first got exposed to environmental toxins in my public health degree at Johns Hopkins in environmental public They're just everywhere.

And a lot of them are really harmful, and we don't even know how harmful they are. Around the time I was graduating, there was a study out of France on women having flame retardant in their breast milk.

That flame return came from just sitting on couches and furniture from absorption into the skin. So that was pretty… And that was 2004, pretty eye-opening, like, okay, this is a real thing.

Then fast forward to 2012 when my late wife got breast cancer. And then that is also another, how can we help her mitigate the toxin she's trying to detox from?

in our home. So, cleaning supplies, soaps, everything. We just went down detergents, went the gamut to figure out, okay, how can we minimize what we're getting exposed to?

And it's so interesting what you're saying, right? Because this is low-hanging fruit.

David's environmental toxicity wake-up call

This is not, and there's a French expression, c'est pas la mer à boire, which is it not about drinking the ocean, this not an overwhelming task to clean up your household products, to go after even your beauty products and to respect that the skin is your largest organ of absorption.

If you just did that, I think that if everybody just that it would make a massive difference in the load that they accumulate over time, and in a given time.

And it can sometimes make the difference, not always, when that load gets overwhelming to the body. Maybe it could slow it down, maybe could set it off.

So that is just right there, is so powerful. Guys, you may have heard it before, but I got to tell you, You cannot underestimate the value of cleaning up your home environment.

And it's just not that hard. It takes a little bit of thinking, but it is not hard at all. So you've shared the story about inflammation impacting your ability to play guitar.

As a musician, can you walk us through what happened physiologically and emotionally in that window, like how that kind of affected you? It was during when my wife was sick.

I wasn't really taking great care of myself. And basically, it would be within two minutes, I had to stop because my knuckles and my hands were in so much pain.

it stabilizes, as John said, blood sulforaphane. And about six months after she died, we had it, had broccoli on the shelves and were for sale and we were all taking it.

I noticed within, I mean, literally a first dose or two capsules that my hands were not in pain anymore. So physiologically, basically what we showed We did actually a study because we were also like, this seems to be working fast, we did an internal study on friends and family and showed basically that two capsules of 10 milligrams of sulforaphane drops IL-6, which is pro-inflammatory market drops at 28% in 24 hours.

So that's enough so you can feel the difference. That's very powerful. And so, I mean, we're jumping way ahead because I haven't even gotten to John's introductory questions, but just while we were here, and we'll probably come back here later, in your case, what would you have attributed the inflammation to?

Was it arthritis? was it just general systemic inflammation? I very ill, obviously chronic stress was your constant companion at this point. And I just want to say, I'm sorry for the loss of your wife.

I haven't said that yet. But because, you know, there'll be different reasons why people have inflammation and you're mentioning a very specific marker for information.

Um, and maybe we'll get to this later, but in your case right now, cause we're probably talk later about the different ways that it works. Do you think, was it arthritis or was a just general systemic inflammation from your life circumstances at the time?

I don't know for sure, but my guess is that it was systemic inflammation from stress. You would know, right? If you had arthritis, you'd be like, oh yeah, no, I've had an arthritis.

Okay, cool. Now, John, over to you, sir. From a clinical and research lens, when did you first recognize that detox pathways, not just nutrients, were going to define the future of chronic disease prevention?

So I probably go a little bit further back. We had friends that had a severely autistic kid in 1992. And just because I

was the person who knew part of the physiology that they started asking us a lot of questions, me and my wife.

My wife's a biology teacher and a lotta times she comes along with me to interpret. And so this person sorta went down a pathway that I understood from going to a outta the autism conferences and for me, That was really surprising.

This new view of science was that the parents who were frustrated with the science not asking the right questions were driving gathering capital to fund researchers.

And so the researchers were directly getting the information back directly to the parents that were asking it. And I feel like they were at least 15 years ahead of everybody at the time.

You couldn't even look up a lot of the stuff that was being revealed back then. So this little kid got a lot of benefit from just going down simplistically looking at what were the things that made symptoms worse and which ones made them better, what sort of dietary strategies were in there.

That was also when I was introduced to gut dysbiosis. So, this person had a really dysbiotic gut and then when this lady who just wrote a book about healing her child, because they at that time said there wasn't a way to heal your child from autism, he was mainstreamed.

And they asked me to write the introduction to the book.

John's early work on detox and autism

So that was when I tried to formalize what I thought was going on here is that when a person is super motivated to find out their own physiology, they start digging really hard and I love the way that this particular group did it was at these conferences they asked for parents to answer questions about supplements that they had tried and through that, they ranked all the supplements and strategies that the did and so you could logically from their own end of one research figure out what was going to work and it's just so interesting what were the main things that they did and then secondary, but that was my introduction to it.

Hey folks, I just wanted to throw a little message in here. For those of you who are sending me all these incredible questions on social media or through YouTube or even on Spotify, wherever you're listening to the podcast, i wish i could get back to each one of And because I cannot, I created, about a couple of years ago now, a membership community where I get to hang out with people just like you.

I do live weekly Q&As, almost weekly anyway. We also do have podcast guests come in to answer everybody's questions and do presentations, you get to interact live with those podcast guests.

We will sometimes do challenges with some of the partners. Like in 2026, we have a mitochondrial enhancement challenge coming up where we're going to be testing mitochondria function.

we are going be to testing people's deuterium levels. If you haven't heard about deutereum, We've got podcasts coming down the pipes on that. And then people will be invited to follow a personalized protocol and then retest their mitochondria on the other side.

We offer that in the membership community and we do it at incredible discounts that you just can't find anywhere else. So if you're interested in hanging out with me and other like-minded people, which actually happen to include some pretty awesome practitioners and even some previous podcast guests who've come and joined the community, then I invite you to check it out on my website natnidam.com slash the dash longevity dash community or just go to natnim.

com and look for the longevity community tab at the top of the page. Click on that and see if this is right for you. I would love to see you there. and we could make 2026 our best year ever together.

So once again, that's natnidham.com. Just look for the Longevity Community tab at the top of the page. Now let's get back to the show. And I think what's important to take away from this is autism is Autism Spectrum Disorder.

And so there's going to be different drivers and different pathways that are relevant to different kids, which I it's an important thing to say because you're not saying that this one thing is going change every autistic child's life.

But I'd think that at this stage of the game we all should know that impaired detoxification pathways is a piece of the puzzle for every one of these children in some way, shape, or form, and will express itself differently depending on their genetics.

And I'm sure a million other factors that I couldn't even begin to touch. But the other thing that you said that, I think, is so important, then we've seen this a lot in rare diseases where the parents of a child with a rare disease who are unfettered by the guardrails of conventional science and what is known and accepted and should and shouldn't work, just become these uber-citizen scientists and the best ones will end up finding open-minded scientists to work with them to bring the whole thing together.

And we'll find solutions that were previously unheard of. Which is very inspiring right and and I think we see that a lot in the rare disease world where and sometimes they've found things that have changed a kiss like there's a company called you can that has this product called super starch because it breaks down very very slowly and the the source of that product is parents trying to solve a problem for a side for A child who had a genetic condition that could kill them and this product was their solution.

And then they were able to spin it out into something to help athletes and whatnot. But I think that's, you know, as an inspiration to start the podcast episode for parents listening to this who, or people with people in their lives who have challenges, if conventional medicine doesn't have the answer for you, just know that sometimes the answers can come out of the strangest places and not, not always the scientists, Yeah, that's a good point.

Back to you, John, you know, there's this idea that we talk about the body is broken or failing, but you talk About the idea That sometimes it's not that it failing.

It's just overwhelmed. So was there like early data or particular or maybe it was this child who said to You know what, it s not That he's broken, It s just that the systems are overwhelmed and they just need more support.

Yeah, this particular kid, just to stay on the topic, was talking and functioning normal and lost that ability and then was seemingly just stuck in his own body.

And because we knew that he had function to start with, that's a very specific case of autism. It's known to be that where you lose the ability to talk.

So it had to have been something where maybe there was a whole bunch of things that weren't working optimally, but there were some event that started cascade of getting into this, we often call it a metabolic sink.

You have four or five things are wrong and you have to move them all simultaneously with enough force to get them out of the sink, it came down to systematically figuring out what things were triggers.

And it took probably a year, but it was a couple of foods and it Was laundry detergent of all things. And they even did the reverse is went off the laundry, detergents.

put it back on, lost his verbal capacity. And so that was the trigger. From there, once he's verbal and able to talk to you about what's going on he'll be able say, you know, my stomach hurts, I'm bloating or whatever.

I just love that idea of… So powerful. bunch of things moving in the same direction. And we go back to that analogy all the time. Sometimes when you're floundering and you know you are flounding, first thing is just to know, where is land?

Where is safety? And let's just move that way at first. We don't know whether we're going to completely reverse this kid, but let us move in a right direction, like there is industrialized ultra processed food.

And then there's stuff that you can get from your local farmer where you talk to them and say, how do you raise your food? Are you composting? Those are the extremes.

So do the things you really know are going to help all humans, not just him. Just keep going down the line.

How Mara Labs and sulforaphane came together

Start with the basics. And of course, I mean, not to, we have to move on here, but obviously, just to respect any parent with an autistic child, it's not always as simple as giving them the healthy food, right?

Like these, these kids are not the easy kids to I, mean any child that doesn't want to eat healthy foods is a problem. But an Autistic child who has decided that the only food he'll eat or he or she will eat a gummy bear is a particular set of circumstances.

So we say this with the utmost respect for how challenging these situations can be. But definitely, I think the foundational message is so incredibly powerful.

I just interviewed Dr. Jeffrey Bland, today and yesterday, and it comes down to food. It comes to the inputs. How close to nature are the things that you're putting into your body so that it acts as a source of information?

It's not just about calories and energy. Ultimately, that's what you discovered with the sulforaphane. Let's go back to when you guys first connected.

What was that shared thesis about modern toxicity that made the collaboration, ultimately, irresistible. Yeah, I mean, we met the first week that Mara was diagnosed with cancer.

We had a mutual friend who's like, you got to connect with John and his wife, Heather. And so we got together, hit it off. I think I emailed him and called him emailed and or called him every day for the next two years.

I think I may have given a couple Sundays off, but just question after question just sees a wealth of knowledge about cancer. And, you know, I have a science background, he was 10 years ahead in knowing what he knows and knowing, knowing when he knew.

And so basically, we ended up, I asked him, he actually grew more as cancer cells in our lab. And so he basically determined that sulforaphane was number three in directly killing her type of cancer.

We went out to source it in supplements and realized really quickly that all the broccoli supplements on the market are the precursor molecule glutoraphanin.

Even though most of them say they have sulphoraphene, they do not. There are only two ours and one other from France have sulforaphane that's stabilized in the capsule.

And that the reason that they have glucoraphan in their precursor is that stable. Sulforophane is not stable if you're trying to harness it. It degrades within hours to days.

So I think what we did was we grew lots and lots of broccoli sprouts and juiced them every day. And because sometimes they go bad, they get mold. Mold.

Yeah. If you travel, you can't really travel with them. And so one day, I think, John and I were at lunch. I was like, it would be really nice to have a stable form of sulforaphane.

Kind of commiserating, not necessarily asking him to do it. But two years later, he was, like he said, You know, And

I was like, wow, yeah. So that was sort of the genesis of our getting to know each other.

Me and Mara ended up raising the seed money for getting that to market. John and I were working with a different supplement company at the time. And so we weren't necessarily thinking about getting it to the market, but then after she kind of pulled the trigger on that and raising the seed money, she took a turn for the worse and ended up dying.

And that was sort of the genesis of that. It was the impetus of like, okay, there was a moment of clarity and we're going to do this and kind of dropped everything and just made it happen and got it to market six months later.

That's a great story. So in certain circles, certainly in your circles and in widening circles sulforaphane is talked about in almost mythic terms. Certainly in the longevity circles.

Right. And because longevity people love to do that. They're like, Oh my God, I found this thing. It's the thing, right. So let's break it down biologically and what makes it so powerful.

I mean, you've touched on a lot of it already, but maybe just summarize a few of the points. We've talked about cancer, we've talk about autism, now what about in that population of people who run around thinking they're going to be able to cheat death for a really long time?

Yeah. I mean, I will do this sort of the higher level and then John can dig a little. But basically, inflammation. So basically we talked about pro-inflammatory markers.

It's a wonderful anti- inflammatory. BDNFs, so brain derived nootropic factors at the center of brain health, it preserves existing neurons, helps grow new neurons.

And so if you look in the literature on brain issues, BDNF is sort of the center of brain health. So that's super, super important for longevity. And then detoxification.

It works in all three phases of detox, but it's the best phase two detoxifier of any natural compound. I mean, longevity-wise or anti-aging- wise, it stimulates autophagy.

The showing cells that need be recycled, they show them the door, and then also mitophagy, so same with mitochondria. So those are two or kind of the, I guess, five different things.

But I mean, John can get into the weeds about, there are at least five other anti-aging mechanisms for syphilis pain. So you've just mentioned at least three of the hallmarks of aging right there, right?

We've got inflammation. We got autophagy and mitophage. The breakdown in those three systems right away is what's part of ageing process and accelerates aging.

BDNF, who wants to age without a working brain? And if you don't detox, you're just going to be a… garbage can, which isn't going to be able to do anything.

So that's a really nice explanation. John, what would you like to add to that? Because my next question has to with the NERF 2 pathway. If you want to leave this alone the way it is and move on to, you can or you could enhance a little bit of what David said.

Maybe the place to go is where my brain is right now, as opposed to the general ones. But I think a lot of people don't realize that sulforaphane does anything more than activate Nrf2.

So that pathway… seems to be tied to so many things. And one of the studies I really like is a pair of studies that have to do with skin. Just because it's a whole tissue, when they take an old mouse and take the old skin and graft it into a young mouse, that skin gets young.

Sulforaphane's anti-inflammatory and longevity mechanisms

And when you take the young skin and you graft it into an old mouse, that skin gets old. And in both cases, the signaling network maps back to NRF2. It's a master regulator of very many of the functions that are associated with aging.

So it is that way because it's it a hub that touches all of those longevity handles. To buttress that, I always have to say More is not better as well.

So you do a genetic overexpression of nRF2, the mouse lives half as long. If you take sulforaphane to activate nrf2 the lifespan increases dramatically in all the model systems.

And a very fresh paper right now is C. elegans where they just look at the entire transcriptome and it matches the young So it's making the whole transcript of the animal look younger.

sort of a good introduction. But the other thing that I think a lot of people don't realize is the mechanism that sulforaphane activates NRF2 works on a number of other molecules that are much less known.

So in the NF-kappa-B pathway, there's a direct conjugation of one of the regulatory units is is why it inhibits NF kappa B. It's it's covalent modification of regulatory molecule.

But why does that matter? What does NF Kappa B do? NF kappa b is, if you talk about NF2 and the major things that it does, it's kind of a choir director.

It turns on all the genes that are called antioxidant response elements, so it sits down in the promoter of all of the antioxidants. So it makes all enzymes that enzymatically inactivate reactive oxygen species.

And then for detox, all the genes that have detox-related genes, phase two, three, and also phase one by slowing it down, sits down and turns on all these genes in a kind of orchestra-style way.

So one of the lesser known functions is that by the same mechanism, a covalently conjugated molecule that inactivates NF-kappa-B is very powerful. And the thing that people don't realize in that system is, so curcumin is a fantastic molecule for NF kappa B.

That's what it's known for, inflammation, NF Kappa b. But curicumin has a tough time getting into the body and also reaching to every organ. So forfein has no problem with that.

And so because those two things work by different mechanisms together, they're even better. Kirkman also activates nRF2, but it's by a different mechanism and therefore it is synergistic.

In both cases when you take nrf2 and kirkman they are working on multiple pathways, helping each other on those two very, very important pathways. So NRF2 and NF-kB are central to so many things that are related to aging.

Yeah. This is the magic on the longevity and space. Sorry, David, were you going to say add something to that? I was just going say the NF-CAFB, I like to it's the holy grail of anti-inflammation.

So it is what the pharmaceutical companies, the pathway they're going after when they create an antiinflammatory. Beautiful. All right, so in your own testing for either of you, maybe this is more of a John question because it's a testing question, but you've both been testing stuff for a long time.

In your testing, what's the most surprising cellular benefit you have seen that most people either just don't know about other than NF-kB? Is there something else that's happening under the surface that people wouldn't even think about it, and there's such a powerful impact here?

I think we can both answer that. John, why don't you go first? Yeah. So I mean, just because I'm always thinking

about cancer and so people that come in front of me often, many very aggressive cancers have STAT3 as a component overexpression of STat3.

as part of the progression model, and that's inflammatory cascade. So, foraphane, that is another target that it does direct covalent conjugation to. It directly binds to STAT3 and inactivates it.

If that one of hubs that are driving your cancer, you know, it's a supplement you can take that can affect that particular arm of the disease. And we're not saying that cures disease, but it's a natural compound that is known to affect a pathway that doesn't have a pharmaceutical to it.

So no competition there. Well, and that makes sense, right? Because any cancer is going to be complex and any, you know, if you think about a surface with many peg legs, And this is our cancer, as many of those pegs legs as we can kick out, hopefully destabilizes it.

And if You happen to Be doing conventional treatment, Hopefully maybe that Makes it more weakens the cancer, maybe makes it more effective. I mean, obviously we have to tell people they should always check with their team, but this sounds like a very powerful addition or to a treatment plan that might be off the radar for people.

So thank you. And I was just talking with a gentleman. yesterday, actually, U. S. Cancer, he's like, yeah, I'm taking sulforaphane. I am like you're not taking Sulforophane if you are not talking our product, because literally every…

Let's talk about that. Yeah. Every product except for ours and one other, they're are they not Sulphoraphene. And so they say it on the label, people, these supplement companies are marketing companies essentially, and they are getting more and more bold as far as not telling the truth of what they have.

And so there are on Amazon, there were probably six now that say they had sulforaphane on the label. There are dozens that they say that sul foraphene in the marketing, but if you turn it on a label, they said glucoraphanin.

Remind us why that distinction is important, please. Yeah, and glucoraphanin is also called sulforaphane, glucosinolate. And so that's even more confusing.

It's important because if you have a hit of broccoli, you start chewing it, break down the cell wall that has the enzyme or SNase that breaks down that glucuraphenin into sulphoraphine, then you swallow it and you get the benefit.

If you try to harness it it degrades. Sulphuraphene is unstable. And so glycografin is stable, and so that's where people basically put it in capsules because it's stable.

So John was able to figure out how to stabilize it. That's the key. We have a stable form of sulforaphane in the capsule. A lot of people in marketing companies that sell these broccoli supplements basically say, it will convert in your gut.

Well, maybe, but maybe not. And so you don't know. Most of the times it doesn't convert. If it converts, it converses at less than 10%, which isn't enough to make a cellular difference.

It doesnt impact your biology. What we do is we offer five milligrams per capsule. You take one or two and we We've shown it time and time again, and you can feel it.

It turns on these pathways that John just talked about, you feel a difference. A lot of people, early on, I was doing our customer service, a guy called, he's like, we have an unconditional money get back guarantee.

He was like this isn't working for me. I want my money back. Sure, no problem. Let me just ask you a question. Have you had the dreams recently?" And he pauses and he's like, no way.

That's this. And I'm like yeah, yeah. The BDNF. Yes. Yeah. So it does work. If you're not taking our product, you

are not so forthcoming. It'd be better if you just flushed your money down the toilet, period.

Well, okay, so before we… I don't want to go too deep into the product yet because I want talk about all the reasons why this is important, but I'm going to ask you this at the end because, I do… I want go through the hierarchy of foods, right?

I wanna talk of broccoli sprouts and then broccoli, eating broccoli and at end of the day it comes down to, can you get a therapeutic dose in a reasonable…

in a reasonable way, right? We all know that food is magic. And you know, it's funny to me how sometimes quite by accident podcasts get grouped. This week is the Food is Medicine group, how nature offers us so many incredible solutions if we take the time to pay attention and figure out how to harness it and bring it back to the body when we're out of balance in in a therapeutic dose.

So this, this is, it's, just always fascinating to me. Anyway, let's change tax a little bit.

NRF2, NF-kB, and other cellular targets

Let's talk about microplastics because micro plastics are getting a lot of airtime as they should be, because are, you know, I remember years and years ago, somebody saying to Me, oh yeah, these recyclable plastic bags, don't kid yourself, they're not disappearing.

They're just breaking down into itty bitty little bity balls that eventually are going to be everywhere. And at the time I was like, Oh, no, like can't.

And now I'm like, holy crap, they were so right. I walk around the grocery store, if I see a net of, I was saying this in the Grocery Store today, you know, the put organic lemons in a netted bag of this stuff that is never, it's not even going to pretend to disintegrate, right?

But it still will. It's still going release little bitty bits of crap. onions in these nets, and I refuse to buy the nets. But then if you go buy a lemon, it's a buck 50, whereas I could have gotten a whole bag for six bucks over there.

So it makes me crazy. Microplastics are here. There's nothing we can do about it. They've infiltrated our physiology and we stress about. Can you walk us through what the science suggests about the exposure and what it is doing in terms of of cellular stress in the body.

What are you guys seeing in literature? What's happening? Yeah. I mean, I think it's good to just take a step back and just say, you kind of did a good job, but basically microplastics are microscopic sized plastic particles, even nano-sized, that get sloughed off from things like water bottles.

Water bottles are sort of the the most ubiquitous and yes but but i mean if you think about it for uh frozen dinners if everything packaging packaging I mean, even my frozen dinners, basically you get about a million microplastics if you microwave it and then eat it.

If you have leftovers that are in plastic and microwave, put it in glass. Put it on a plate. Yeah. And throw a microwave to begin with. But put in your oven.

Exactly. tea bags that are the pyramids that they're shiny. Those are nylon. Literature basically says 2 billion microplastics just from that one tea bag.

And so it's just like, forget that. You know, things like John and I were traveling recently, we were at a hotel and the coffee maker. Oh yeah, the carrying coffee-maker.

Yeah. Well, yeah. There's a bottle of water to put the water into the coffeemaker. And there's plastic coffee pod. And then there's a paper cup, which paper is great, right?

But the paper's lined with plastic. And so you're getting three different ways of… So anyway, you are getting a good size dose of microplastics just from one coffee.

But how many coffees do we drink? If you go to Starbucks, take a to-go mug. Don't get their paper plate. So once it's in your body, You know, it's a big issue because the plastics are not neutral.

They are like sponges. Because they're embedding in crazy places that is blowing people's minds. Yeah. I mean, I think the craziest is the, We haven't done our own research on this, but you read about you're getting a credit card size amount of microplastics in your brain.

And then as some people say, per month, some say per year. But that's a big place. John's like a kidney expert. He would dissect kidneys and could see micro plastics and kidneys.

I mean, I think the issue again is that these micro plastics are like sponges and basically can have toxins, metals, plasticizers that make the plastics bendable.

BPA is one and it's an estrogen mimic and so it is a big deal because And historically, we can't get rid of these microplastics, right? And so there's a buildup.

And you're getting mixed signals. Estrogen mimics hormones or signaling, estrogen is a hormone that signals, it tells girls when they should go into puberty.

And so these microplastics and these BPAs and estrogen mimics are part of the reason, it's not the whole reason but part, of why girls are going into puberty earlier, why women are having irregular menstruation more, and why boys are growing to puberty later.

It's a big deal. And I like to think of it as like, You have two TV shows on top of each other and you can kind of tell, you know, okay, I can see a character that's this show, but you have no idea what the story is.

And so because the signal is all garbled and that is the same if you all these microplastics and these BPAs and this estrogen mimics, confounding and messing up and having noise of the regular estrogen signaling pathway, then you get a lot of problems.

Yeah. No, it's a good… I mean, one of the things that I think I've heard as well is because it mimics estrogen, that it occupies the receptor that estrogen would use.

And so now you have an inert, somewhat toxic molecule sure basically preventing the proper messaging of estrogen to the tissues right there and so that's where that and it does it in a bunch of different stuff so let's talk a little bit about and maybe i don't know john if you want to take this like When these plastics accumulate intracellularly, what are we seeing happen to mitochondrial function?

Well, we just talked about hormone signaling, frankly, and endocrine balance. I'd love you to speak on mitochondria function and a little bit about chronic immune activation, if that might be getting triggered in some way by these plastic particles.

Yeah, so probably the place to start would be why do they accumulate where they do in the body? So the places that you have contact with the outside world or primary, so your lungs, your digestive tract, and your skin.

And then the cells that tend to accumulate them are all cells have high rates of phagocytosis. So macropinocidosis just gobbles up the fluid that it's surrounded.

Those have the highest rates accumulation. Then the professional eaters that travel around and eat things which are macrophage and in fact macophage derivatives.

So highest is along the lungs, along that digestive tract, in your spleen, and then in you macorphage or any

macrophages like molecules. In your brain it's microglia and so they're basically the macrophage of your brain and so you can track all the dysfunction basically to all of those things so in every lymph node that's trying to communicate to you know for any kind of foreign body will Those cells that are supposed to be doing that, are filled with microplastics so that their lysosomes are full.

Changes the pH of the lysose, it's not degrading misfolded proteins. And so there's just general dysfunction. It would be like the same as your apartment if you just never flushed your toilet.

You wouldn't be enjoying life in the apartment that much. And there are things you can do to move that out or you're stimulating particular pathways that make your plumbing better, make more toilets, Pipes this is the way to go because it's flux.

It's you know, how fast what's the capacity of yourself? And so All of those things are sort of linked together. So you you asked me to answer the question of mitochondria so mitochondrial Actually will physically contact and interact with lysosomes.

They they are part of the signaling network for lyses ohms and they will be part of the system that decides where things go in the sorting machinery inside of a cell.

And so if you have lots of energy and you're producing lots ATP, you'll have all the systems up and going. If you block the toilet in that system and and your not making ATP as well, it's going to affect every ATP dependent function in also.

and you're going to get miss sorting.

Microplastics, endocrine disruption, and cellular stress

The one that I'm particularly interested in, because I am an exosome person, is that when you block the lysosomes that normally takes care of misfolded proteins, instead it gets shuttled over into the system that shuttles these proteins into exo-somes.

then you have them bottled up into an exosome and then your affecting all your neighbors. So even if you weren't an inflamed cell, you're close enough to a damaged cell from microplastics that you are getting transported genetic material from that cell transfecting you and changing your genes.

That's one of the ways that You spread your inflammation into a local tissue, so that's probably enough information there. But because it's an immune cell, then you have a very distracted immune system.

They're not primed to be looking for foreign objects at that point. analogy of Yosemite Sam, like this hair trigger sheriff that's just shooting in the air spinning in a circle.

He's not really effectively taking out the bad guy. he's randomly shooting people and that is your immune system. Okay, so immune systems, mitochondria, endocrine signaling, hormones.

Give us some hope. You've reported mobilizing. There is hope, there is. So let's talk about what the heck we can do about it. Because God knows we cannot leave people on this note.

It's a little dire. I want to just talk how the body gets rid of these things and where we need to measure them. Like there's this school of thought that says we should measure in blood.

Another school that said we could be measuring in urine. Why is measuring, and I think that in your world, measuring excretion, what we're getting rid of is even more meaningful than measuring just blood levels alone.

And I might be starting from the end here because people are going to want to know, well, how do I know? How do know and the three of us can sit here and say, because of all the reason we talked about 10 minutes ago.

But talk to me a little bit about how people measure this stuff or can it be measured? Yeah, I mean, do you want to take it, John, or do want me to…

Well, the bottom line is we don't know. So you find it in urine, you'll find in feces, and you will find that in sweat. And so those are the main routes that are going out.

There hasn't been a way to mobilize them previously, so they stay inside your cells. The highest counts are in feces right now, and I think the reason for that is that that's the biggest dose from your food.

And so if it doesn't get absorbed in your alimentary canal, it shows up in you fece. So that where the majority of them are going right. If I had to guess, I would say when they're mobilized.

I think it's going to be back into that system, the way that you get rid of things through feces. That is the that's gonna primarily go. If they are bigger than a 50 kilodalt protein, they don't usually go through the kidney that way.

So it would have to be exceedingly small for it to make it past the glomerulus. That's not going to the primary way, it'll lodge in the tissue for sure because there's a lot of surveillance of the kidneys with macrophage.

I was going say it must be damaging to kidneys in some way because they're so delicate that These things are like little BB gun bullets. Can I talk about the study?

Yes, I want you to. I was going to ask about it, because you've reported mobilization of microplastics. So what did you actually observe in those human cell studies?

Unless there's another study, you're welcome to talk about both. We didn't actually do the study. A gentleman from the Midwest, a researcher, we found out that he did this without our knowing, did a study using broccoli sulforaphane, our form of sulformaphene.

He did an N of 1 study and and basically showed he's a lysosome expert and showed that the sulforaphane degree so that they can be mobilized. Interesting.

And so he didn't show how they got excreted, but he did show from over a period of time. He actually had about a day after the taking of the dose of sulforaphane, he had the highest microplastics blood reading that anybody he has had from this, I think there's just one lab, Brian Johnson's lab that measures microplastics of blood.

And so he had the highest and then a day later it was all the way down to next to nothing. But to the point he did measure, he didn't look at the sizes of the micro-plastic and what John was saying is based Basically, most of the microplastics are larger than cells, so they're not going to be in cells and they are not gonna be peed out.

Most of them are gonna come out in feces, though the smaller ones can come in urine. That's the study we're working on over the next month with this gentleman to try to figure out how are they coming out?

Yeah. Well, you know, one thing we need to leave the audience with is poop rules. You got to be pooping every day, guys. Got to dump this stuff. So in a polluted environment after sulforaphane use, were there particular toxins that were shown to eliminated more efficiently or that couldn't maybe even otherwise be eliminated?

Yeah, I mean, I think probably what John's thinking too is that there's a Hopkins study out of China metropolises that, you know, if you've been to China, been Beijing, and you basically go outside 15 minutes, your black is not.

And so it's a huge, huge amount of smog. So they were basically looking at how it is caused typically from automobile pollution and industrial pollution.

They were looking people drinking a broccoli beverage with a certain amount of sulforaphane and they measured their urine and showed basically benzene, benzine particles from the pollutants coming out in the urine.

Wow. Wow, let's talk about glyphosate and how it suppresses detox systems like NERF2. Is that a thing? And how do you reverse it? How do we change that?

I mean, again, like big fat problem, we talked about how important Nerv2 was at the beginning of the podcast. So if glyphasate suppress is detox like Nerf2, How do we reverse it?

In the lab, we did those experiments where we put glyphosate onto cells and measured nRF2, and it really tanks it. It makes it reduce by 50%, which is kind of crazy.

And then if you just take a dose that is what we know gets into the bloodstream, put on those same cells, It not only brings it back to normal, it's actually above normal.

So 30% above the starting material even in the presence of glyphosate, so super protective. Not to get into the weeds there, but we found that glyphosate also down-regulated gap junctions.

The way that you get these spot welds between cells where you're communicating small molecules, so it acts like a tissue. Glyphocytes made cells act more like individuals, which is pretty much the first step towards carcinogenesis.

And so that's what glyphosate has been the most connected to is that it's showing up in these cancer epidemiological studies. So we say glypsate's the master and toxicant because it is turning off the system that usually gets rid of toxins.

Glyphose plus mercury, much worse. Do you think the sulforaphane is displacing the glyphosate because you said the cell is better than it was before? Do think it's actually kicking it out also?

I mean, I don't know if you know this mechanistically, but is there a theory that perhaps one of the mechanisms of action is that it protects a cell, is it possible that somehow it is kicking the Glyphosphate off?

Yeah, so glyphosate looks like an amino acid. And so it is a toxin, it's the antibiotic. So it would be gotten rid of in general by phase two, phase three detoxification.

It does get rid it faster. I think some of the most interesting studies around glyphasate is that if you have tight junction problems in your gut, you end up with much more glyphosate in you urine and so it makes it past the enteroside layer and gets into your blood and then small molecule wise it goes out into, it's water soluble so goes into urine.

And that's sort of the beginning of how it got past people that were looking at ingestion. So they took radioactive glyphosate, gave it to healthy people, and more than 99% of it came out in feces.

But they weren't looking of people who had some disease where they have some tight junction. dysfunction, which is just about everybody now. And so I think probably the best way that sulforaphane decreases glyphosate toxicity is that it's known to tighten tight junctions, does it really well, shows up in increased tier values when you study that.

And so it's keeping it from being absorbed in the first place. And I think that's a great way to think of it as the First Round. That gets back at your question about immunity, where the problems with immunity is not fully digested proteins making it past the enterocytes so that you have some structure that your immune system starts making antibodies against and then The autoimmune version of that is that some of those look like things, repeat structures that are in your own body.

If it ends up looking like skin, you get psoriasis. So if it's lupus, it can kind of go everywhere. thyroid. That's the beginning of so many of the autoimmune dysfunction that's out there.

And so that a great way to think about being protective is tightening your tight junctions. We were talking about the health span longevity hallmarks.

Barrier is one of It does that in every epithelial cell line. If you can tighten tight junctions, every tissue is going to be more intact and it's going help you just in general in terms of health.

Measuring toxin excretion and mobilization

It's a great idea when it comes to just trying to buttress against this toxic world that's probably pretty hard to completely get out of. No, but it sounds like a really good start.

You know, you're getting at a bunch of very foundational issues here, which are not easy to get to, right? The microplastic problem is not an easy micro plastic, not a easy problem to solve.

The glyphosate problem, avoid, the cat's out of the bag. Okay, now how do we mitigate? So mitigation, like when it comes to microplastics and things like micro-plastic and glyphosate, that it becomes so insidious.

Or air pollution, anybody living in a major city, this is your reality. So at this point, what are you going to do about it? And this where this so powerful, it's the, how do we mitigate the reality that we live in?

And that is a big deal. It solves a lot of problems. So many of the papers have already been done to the point where you don't have to talk about our particular product because there's so many papers out there.

Look at mercury papers. There's some crazy papers relating to mice where they give enough mercury to where they're dragging their back feet and then they give them sulforaphane and they stop dragging the backfeet.

They did the same thing with to death, you know, give enough mercury that the mouse dies and 40% of them live if you give him sulphoraphene. That's an incredible dose.

You know that's how potent this molecule is for detox. Is it only mercury? Does it help with any other heavy metals or is it somehow specific to mercury.

It's all of them. Wow. Okay. I think maybe guys, we have to rethink our foundational stack here. All right. Let's move on to GLP-1s. GLPs are part of their claim to fame and part the reason why they can be so powerful and for the right person at the time in the way, just to be clear, is that they pull several different metabolic levers.

So you've developed a product that also addresses more than one metabolic. It's not just about satiety. Can you help the audience understand which are the metabolic levers that the GLP-Perfect targets simultaneously that kind of aligns with what a GLp-1 does so it can be a reasonable alternative to the right person?

So that's a me question. I mapped out when we were designing it to address a compilation of 26 metabolic handles that are associated with adiposity. And so I'm not sure which ones of those that if you just- We can get the major ones maybe.

Yeah. Okay. The big one for GLP-1 agonist, obviously, is that it's your one that's in your brain that regulates whether your stomach empties. When you do that really hard, it makes you basically not want to eat, which is kind of the mechanism that it works through.

Your stomach stays full. You feel satisfied with it. With no food, you're fine. And so that's one. So berberine induces GLP-1, but it doesn't turn it on and keep it constantly.

It doesn' actually stop your stomach from emptying. So that would be one of them. So if we just name a few of the ones that GLPerfect does is because muscle is the big problem with GLPs is that the weight that you lose up to 40% of it is muscle loss.

And then so we were particularly interested in trying to block that. EGCG affect myostatin, one, by inducing folistatin so that you get less. That's an inhibitor of myostatin.

And then E G C G transcriptionally turns off myo statin, so most people know about that molecule because of the cows and the dogs that have a knockout of it.

They look like Arnold Schwarzenegger. versions of cows and dogs. Well, it's not a knockout, but it is going to make keeping your muscle on much easier.

So if you are working out, you'll be able to put on muscle a bit easier, But we designed it so that you wouldn't lose muscle while you're losing the weight.

And then a couple other ones, the alpha-lipoic acid that's in the product actually makes you insulin work better. So you get glucose being able to go through a glute floor into your muscles.

That's another handle for not losing your weight through muscle. If I can maybe recap a little bit. Satiety basically helps with satiety without slowing down gastric emptying to the point where people are not going to be able poop out their microplastics, for example.

Sure. and maybe shut down some of the food noise, which I think is the other thing you were getting at with the Brain Center. It helps with insulin sensitivity.

Everything I've seen doesn't say that GLP-1 causes muscle loss. a muscle loss problem. It's a lifestyle when you're on the GLP-1 problem, but… If you basically…

You have to try very hard not to lose muscle. No, you have work at it. But it upregulates the expression of glut for a skeletal muscle, which means like the way you've designed yours, it's super smart.

You want to make it easier for the muscle to pick up the glucose so that you can exercise and build muscle but we don't have argue about that. I think that ultimately anything we can do to preserve muscle is going to be beneficial.

And if you've built that into this by affecting myostatin-folostatin balance, that is not something I've heard that a GLP-1 touches. So I think that's a really powerful lever to…

And I don't think we need to argue about it either. I just think a statistic out there is that people lose muscle when they're on a GP-11, whether it's lifestyle, reason for that.

Yeah, and there's papers.

Glyphosate, heavy metals, and detox support

It's also papers showing that that Most of the people gain them all gain the weight back, you know, it's as high as 70% I've seen in between 60 and 80 percent of people gained up gained the wait back But if you lost any muscle and that you're actually be behind from your way behind 100 I'm like a hundred percent with you for sure So it blocks de novo lipogenesis It blocks This is the GL Perfect.

Wow. Yeah. So the conversion of fatty acids and proteins into glucose, gluconeogenesis by your liver blocks that. Can you just go down the line? And I was surprised to find that because I wanted to know what percentage of the 26 handles did that.

And pain is one of them. The non-shivering thermogenesis it induces, that's through a norepinephrine dependent mechanism. So just keep that in mind in that you're going to burn more calories just without doing anything.

It just raises your basal metabolic rate. But it also tells you that you probably don't want to take this right before you go to bed. So this is a morning, first half of the day kind of supplement.

Actually, that's a good question. Do you take it before meals or is it the kind thing that take one today and it exerts its influence over the Yeah, I just say do it in the first half of the day.

If you want to do a direct inhibition in a shorter period of time, just parse out the berberine. So we have a berbalete product that is a bioavailable form of berbrane.

You can take that right before you go to bed. It actually helps you sleep. And it also helps right Before you eat. Love it. Do you wanna add anything, David?

Because now we're gonna go ahead to frontier research and what's next. We're looking ahead. Sure. Yeah. I mean, I just to jump in what John said, you know, that our berberine is in the GeoPerfect, but we have our own standalone berbere product.

And so that's great to take it. We say take two at night. It helps with if people eat late. Some people do, don't recommend it, sometimes you can't avoid it And so the issue there is when your melatonin starts being stimulated, it turns off the insulin production.

And if you eat late, you have high glucose in your blood all night. Disaster. So that's awful for a variety of reasons we're going to. Yeah. Taking berberine, that can then basically help metabolize the glucose.

It's not just sitting in our blood. all evening, but it's also great for your sleep. In fact, early on we looked at asking people who are taking our berberine product, hey, why are you taking it?

And they basically were saying about 75% of people were taking for sleep and we were selling it for ptosis. And so I looked on the literature and there's one of my favorite papers is how berbere now performs volume as a sleep aid.

You're kidding. Yeah, it increases. serotonin by 30% and dopamine by 25%. So anyway, it's a great product, especially if you're eating late and have sleep issues.

Well, berberine is that herb that just keeps on giving, right? It's so incredibly powerful. I actually want to ask you one question about it because there is a narrative out there that berberine can be anti-microbial and so can possibly maybe have a negative impact on the microbiome.

Have you seen that at all or do you think that's overplayed or maybe is it a dosing duration issue? John's nodding. Any thoughts on that? Yes. The general idea is that it's part of a natural…

A number of papers use berberein that they use berberine for SIBO. It's very, very effective, spending clinical studies to do that. And so it's effective in the small intestine when you want to kill off cells.

But if a very high dose, some people take as high as a gram goes down into your colon and sits there. It's antimicrobial in general and that's not a great idea.

And so I think the way to think about it here is if you have a smaller dose of a higher bioavailable berberine, it's going to be absorbed in your small intestine and a lot less is going going to make it to your colon.

And there is a paper out there showing that moderate doses of berberine actually increases acromancia. So it can be very beneficial for some people. That's one of those molecules, bacteria that people don't Talk about they just say it's good.

It's because it makes butyrate But actually it called acromansia mucifillia so it eats mucus so you know if you're not feeding some of the other bacteria and and things You don't want that You're all your microbiome to be that or it'll eat your mucous Membrane, and you now that's not good That make a mess of things for sure.

Yeah. And that is so interesting, right? So many people talk about increasing acromantia and the flips, the dark side very rarely gets mentioned. I think somebody needs to start a health company called the Sweet Spot Health because the fundamental reality of the human body is it's all about the sweet spot.

no matter what you're talking about, unless when you were talking in microplastics, but it's, can you hit that spot

that the body likes to be in, that is homeostasis, where it not too much, not to little.

Okay, let's look ahead. What frontier questions about sulforaphane and cellular health are you the most excited to explore in the next five years? Because I can see that you guys are not just gonna sit with your stable of offerings right now.

You've gotta be looking ahead at some other challenge, and particular to sulforephane in this case. So I'm particularly interested in synergies. It's one of those things that I just have always been interested I'm working on and so next supplements and all the ones that we have already, we've proven is synergistic with Nrf2.

So there's actually 12 ways to, you know, handles on how NRF2 is regulated. And so we want to regulate them gently. Even with the sulforaphane, and we wanna turn it on gently, but then you wanna support each of the ways that is downstream of releasing it from KEEP1.

translate into the nucleus, you have to get it to go to the downstream effects of Nrf2. I'm interested in the HDACs and things that turn on genes downstream.

And then nuclear export is a regulated process. Want to make a supplement that blocks the export of that. Additionally, I'm very interested in doing the same thing with quercetin and some of the known downstream things that are downstream of quersetin.

So it's a very strong synolytic. And so in the literature, all the querketin trials are with disatinib. I've worked a lot with the disatinib and It's a pretty promiscuous kinase inhibitor.

GLP-Perfect, berberine, and metabolic support

I don't like a lot of the ones that it's inhibiting. So I want to have a better one to punch for sinolysis. But that might be two of them that are down there.

A lot people worry about if you push so forophane too hard. There is a thing called reductive stress. It is possible. And so I'm going to make a supplement that buttresses the other side of that.

We already have it, but we haven't released it yet. It's coming. on its way, so you can assure yourself you're not going to overdo it with taking sulforaphane, even if you do what some crazy people do is take tons of it.

We're always having arguments with our people that are taking very large doses of that, and we'd rather not. You find out the limit of how much can you take.

Is there something that shows up physically if somebody's taking too much that they can tell? So in the studies, it's all gastric distress. And so 350 grams is what the literature says.

Yeah, 350 milligrams. Water bottles. We have a number of people that even after talking to them, they're taking quite a bit more than that. So 350 is the ceiling, basically, of what a person should take in a day.

You're taking more sulforaphane is actually protective on kidneys. And so that's one of the issues with like, you can't take a certain amount of Advil because you're going to kill your proximal tubules.

So that is the opposite with sulphoraphene. The more you take, the better your próximo tubule will do. But not more than 350 because then you'll make other problems.

I mean, yeah, basically. Or at least not long term. Maybe you could do a pulse or something, but anyway. Yeah. And actually, in terms of something as far as studying, there's a lot of disinformation about sulforaphane.

Basically, one of the more popular influencers out there on sul foraphene says 60 milligrams is the way to go, and it's just not true. 60 milligrams is…

I mean, John talks about there's a ceiling, not just a feeling of what you can take, but if you're just taking… And part of the reasons we like to look into synergies is we call…

John calls it sulforaphane equivalents. So basically in the blood, there is a limit. And if you take 20 milligrams or 60 milligrams, it's the same amount you get in your blood.

And so that's why we have broccoli plus, we put in the cousin molecule from watercress called PITC, phenethyl isothiocyanate. And so that works in a true synergy.

So one plus one equals five. It's five times more effective in stimulating the inner two than just sulfuric vanillin. And, so, curcumin does the same.

to be truly synergistic with Sylphorhaine. And so looking more into that, as John was saying, but also dispelling the common misconceptions of what is glucoraphan.

Precursor, a lot of people say that's fine. How much sulforaphane do you have to take? What are other synergies? Our broccoli, we talk about sulphoraphene, but it has nine different isothiocyanates in it that all work that are from the seed.

So that are basically very similar to the Johns Hopkins studies in that they have not just sulfur vein, but everything that comes from the seed. And they all work together.

So looking more into how are they working together? Yeah. One thing I wanted to mention actually before I just have a couple of questions left, but this whole, I want it to get back to this like eating broccoli sprouts versus eating Broccoli.

Um, and this, you know, sprouts being so much closer to that. You're mentioning the seed. The seed holds all that potential. There's so in there. Once it sprouts and it gives up some of that potentially, the sprout is still a very concentrated source of nutrients and micronutrients.

By the time we get to the broccoli, the full food, which is powerful and great. A lot of that material has been basically used up to produce the food that we eat, I think.

So for people listening, if they're using broccoli or broccoli plaits, should they also be eating the sprouts? Are sprouts enough to move the needle ever?

I mean, you'd have to eat buckets of them, Yeah, I mean, basically, two of our capsules is the equivalent of five pounds of mature broccoli, which is equivalent to about two and a half ounces of sprouts.

So I think the big thing to keep in mind is not all broccoli seeds are created equal. We've done extensive testing, you know, back in 2020 we tested a batch, we were trying to looking for organic seeds and we bought seven different kinds and six of them could not produce cellophane.

Interesting. Yeah, and then we did it again six months later and instead of of the nine could produce clorophyll. And so it's basically we sell the seeds.

We're not a seed company. we don't make money off of it really. But basically people want to grow seeds to produce sprouts. I guarantee you to get 500 milligrams.

Wow. That's amazing. Super awesome. Okay. Are there other areas of aging, cognitive resilience, immune memory, epigenetics, where broccoli might play a role, but the research is still too early?

And you can be brief here. I mean, are there any of those areas that you're like, whoa, there's some real potential here, we're not quite there yet. We can't make the claims.

I think the epigenetics component of it are often through HDACs, histone deacetylases. Yeah. Famous one, a CERT one. So I the way to think about those connections would be through things that our technology doesn't actually help.

Butyrate, all the different ways that you can make butyate. you know, by taking your prebiotics, gosfos, all those things, you're making more butyrate.

That's the way that I would try and get at those molecules. And then we sell a really bioavailable version of Vizveratrol and so that it's longer lasting in your blood.

And so, that's a cert activator. So, I think there are really good ways to get at the epigenome in ways that don't really have to study that much. I prefer to go at it instead of making a more potent version of sulforaphane is just to rest on the fact that it has been shown to be an HDAC inhibitor.

It does move the needle in the right direction. There's a big paper coming out soon in C. elegans that is basically saying that. So the ways to get at those pathways, for me at least, is that you have to hit all of the known hubs of signaling.

So there's basically nine major hubs that everybody knows is what's regulating longevity.

Future research, dosing, and closing remarks

And so you can just follow it right in the products that we make, that every one of them has a number one handle that it works on. And that when they're put together, you have a cohesive package that is moving every handle.

And so at some point in the future, we'd love to do some of those age, grim age. True age and all those things to prove it. Biological age? Yeah. We kind of hope other people are going to to it for us, but we may have to.

You never know. I could be arranged. David, anything to add? Yeah, I mean, two things that we haven't talked about. One is I would be bald if I weren't taking sulforaphane.

Interesting. It basically downregulates the DHT, the bad form of testosterone that can lead to And then we've touched on microbiome in different areas, but one of my favorite anti-aging studies is a mouse study looking at basically older mice taking sulforaphane, having the same younger microbiome of younger mice.

And so there's just a lot of different ways it helps. That's one. Powerful. That's very powerful. All right, let's close it up here. You guys, this was great.

Thank you so much for your time. I want to mention now, because this is time sensitive, that you've made a very generous offer to the audience that for you guys you get for the next week.

So this podcast will drop at the end of March until April 7th. If you go to mara-labs.com forward slash nat and use code nat at checkout, you will save 25% off your purchase.

And then after the seven days, hope is not lost. It just reverts back to 15%. But if you jump on this right away and you stuck around till the end, well, I might mention it at the beginning in the intro.

But nevertheless, 25% off for the next seven days. So David, John, thank you so much for joining me today. I know it's a Friday before the long weekend, so I'm sure you guys have plans.

Um, I really appreciate you taking the time and for doing what you do and bringing this to the world. It's really powerful and, you know, heartfelt mission.

Thank you. Yeah. Thanks for having us. This was a great conversation. Appreciate it. Oh, why don't you tell people where they can learn more. Sorry. So obviously the website mara-labs.com.

Are there any other channels that people should know about? Yeah. We are on social media at the Mara Labs on Instagram and Facebook. You can follow us there.

And then we have We have a blog that we post on every week with recipes and science articles on Moira-Labs.com. Beautiful. Thank you.