Introduction to Elizabeth Parrish and BioViva

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We felt, hopefully, that this would be enough. Hey, everybody, we'll get right back to the podcast, but I do have some very, very exciting news I want to share with you, and that is that my new book, Brain Defenders, that we've been talking about on the Podcast is now available for purchase.

It is going to be published in August of 2026, But it is available now. If you want And that is oddly enough, brainedefenders.com. This is really empowering information as relates to charting your own brain's destiny.

Now let's get right back to the podcast. We've got quite a program for you today on the empowering neurologist. We're going to step boldly into a frontier where biotechnology and longevity actually intersect.

Our guest is Elizabeth Parrish. She is the CEO of a company called Bio Viva. And this is a company that is pioneering gene therapy strategies that are aimed at nothing less than redefining what we call human aging.

She is known around the world as the first genetically or GMO, genetically modified human, genetic modified organism, she made global headlines when she chose to undergo experimental gene therapy herself, an act of what we call scientific conviction that has clearly sparked a lot of debate, curiosity, and certainly extraordinary interest.

Her work has been featured in MIT Technology Review, The Guardian, Nature Biotechnology, Vice, the Washington Post, and a variety of international media outlets exploring both the ethics and the promise of genomic medicine, which is certainly what we'll be talking about today.

We're gonna explore this, as I said earlier, this bold idea that aging itself is a disease, one that may be treatable, through targeted genetic interventions.

We're going to discuss the genes that she actually received and has been receiving or began receiving in 2015. How they're made, how they are administered, what are the vectors that are used to deliver these genes, and why, if we redefine aging, this could open the door to really a new era, not necessarily human lifespan, but at least human health span and we will define these terms.

So this is a very forward looking groundbreaking and a conversation I think you're just not going to want to miss. Elizabeth Parrish, welcome to the podcast.

Thanks for having me. I'm happy to be here. We had a great Zoom call the other day, I guess you can still call that, and I felt like I had to cut it short because I didn't want to encroach on what we're gonna talk about today, but as we talked about in the setup, we've got a lot to talk.

But I think our viewers would like to go back to the beginning as would I. When all of a sudden you had this epiphany that there needed to be a better way of doing things, what happened to you years ago and when was that?

Yeah, so that was in 2013. So in 2011, I started an advocacy program for the use of stem cells, trying to get the government to consider using basically putting money back into stem, cells and that the public needed to understand that embryonic stem.

Cells were not fetus. and that the predominant use of stem cells was not embryonic stem cell. That was all very research-based, but autologous stem-cells, your own stemcell, had a lot of medicinal benefits that shouldn't be shut down or considered unethical.

From Stem Cell Advocacy to Aging Research

So I had couple years in regenerative medicine space and learned enough to know that there was fantastic research in universities and that doctors, medical doctors were taking this technology offshore and using it in patients to some benefits, but it wasn't really well documented.

In 2013, so two years later, my son was diagnosed with type 1 diabetes and I was thrown into a world that we really don't know exists in the general public, which is the world of childhood illness.

the hospitals, these children's hospitals are full of kids who need help and, you know, everything from type one diabetes, which is considered a manageable disease, but it's a minute by minute, manageable, disease all the way to cancers and childhood surgeries.

And I guess this really shocked me. It not only disrupted our lives as a family, but it made me realize that this technology that I had been learning about for years now was not translating to the patients who needed it most.

So that really is the beginning of the trajectory of my story. I go looking for cures for kids, I end up going to a conference about aging because the professor of genetics of Harvard is going be there and I want to know what can be done for And I wake up to the world of the idea that we could treat both sides of aging, the young and the old, vastly with the same gene therapies in many cases, and help them all live a longer and healthier life.

Let me stop you there for a second, because what you just said was really quite interesting. You said you were looking at treatment of ageing. And I think most people might be surprised to hear that, that we understand treatment of disease states, but here you're talking about actually treating something that quite recently would consider a normal part of the continuum.

That we all expect that this is what is supposed to happen to us. We're gonna age, we're going to decay, become less able to carry out our activities of daily living.

And so you were talking even now, years ago, Uh, about this concept of treating aging, I guess, as a disease. Yeah, once I went to this conference and I sat through many of the talks, not everyone at the conference, probably very few people at a conference would agree with me.

I immediately thought this is a. Because the pathology of aging is the same in everyone, there are shorter fuses. Some people are more likely to get dementia over cancer, and some people more are likely have organs fell before other people of their same peer group.

But if you cured any one of those disease, you will just die of another. And so the pathology of aging is succinctly there. And this takes us back to 1960 when we finally encouraged the U. S.

government to consider cancer a disease. As you know, it wasn't considered a diseases. It was considered the consequence of ageing. But before we like inflame and infuriate everyone watching this, there is a difference between chronological age and biological age.

Biological aging is the disease, that is, the biological process of your cells getting older, cellular degeneration over time, and that causing disease.

Chronological ageing, we want to embrace. We want you to get very old in years, trips around the sun. So, you know, separating those two to make people feel, I guess, less diseased is important.

Well, in my world of neurology, Alzheimer's was considered, and in many regards still is considered what happens to you. People would say Alzheimer is old timers, basically.

And statistically, if you live to be age 85, you've got about a 40% chance in America of being actually diagnosed with that pathological state. The most powerful risk factor for which is your chronological age.

Yeah, but then we can take it deeper into biological age, and that's why people are diagnosed with suspected Alzheimer's at different ages. And so that would incur cancer.

kidney disease, COPD, dementia, basically all-cause mortality seems to run at a different biological rate in different people, and that's why you could have 20 people in the room that are all, let's say, 76, 20% of them might die in next year, 2% might go on to be 110. It's a biologically monitored process that has to do with lifestyle, and then it also has do to with genetics.

Right, so you pondered this. You confronted yourself with this reality, you ponder it, now what's the next step? So the next step of our company in 2013, well, it didn't actually exist then.

It took two years to develop the company. I had to figure out what was wrong then with this whole system. If there were gene therapies that could potentially reverse aspects of biological aging and those same gene, therapies could treat childhood diseases.

And let's just pull out the first two that we started the with telomerase reverse transcriptase. is a hallmark of getting old, so the consequence of not having it, let's say.

So our telomeres, the caps at the ends of our chromosomes, shorten with every cellular division, and 24 species, including humans, lifespans are tied to that shortening.

Now, it's not a perfectly measured biological aging clock because your telomeres can stay short at the end of life for many decades, but the diseases of aging that you are diagnosed with come when they're at about 5,000 base pairs.

Your cells then go senescent, so they no longer divide, the telomerase don't get shorter, they are a measure of disease. That gene therapy itself can also treat progeria, which is accelerated aging in children.

So they have a Lamna A gene defect. It creates more progeren in the cell and it degrades the telomeres at the ends of the chromosomes. These kids die of same diseases that we die in our 80s in their teens and early 20s.

We know telomerase are a big problem with biological aging. Another gene therapy that looked like it was super beneficial already in children with muscular dystrophy, but on the other hand could be recatered to an aging population in people who have something that's called sarcopenia or muscle loss with age.

Well, we wanted to take that, expedite the use of it, and create the first combinatorial therapy to treat aging. This took a two-year process of a lot of research trying to find funding for companies who were already looking to do this and them just giving up the technology for us to it ourselves.

So in 2015, I launch a company and the premise of the company is that we are going to treat humans. We are not going treat mice anymore. There is enough evidence in these gene therapies that it actually benefits their life span and health span, and I become the first test subject.

Okay, so here we, what year is 2015? And you have this company and you are personally convinced that there's enough data, animal data that you're going to go ahead and have these genes inserted into your cells basically using a viral vector.

We'll talk about that in just a little bit. And the two that chose again were folistatin and Tert. Yep.

Treating Aging as a Disease

Okay, so again, the first is designed to inhibit myostatin and therefore take the breaks off of muscle growth. And the second is to amplify telomerase enzyme to maintain or perhaps even lengthen your telomeres.

Correct. Okay. So what happened? So what happened is, well, we started the company in January of 2015. I needed to raise money to do this. A lot of investors walked away.

They hung up the phone. If they were in person, they just stopped talking and kind of slowly moved out of the conversation. And I finally found someone who believed in me and thought that this was absolutely the right thing to We have the therapy made and I traveled out of the country to take it So we let me just take a breath here for a moment because you know, we've got a lot to talk about but You saw this these issues in animal research showing positivity and you form a company you end up manufacturing these genes, you go offshore and you have the very first person on the planet injected, uh, to have these jeans injected into your body.

Um, what were you thinking at that, at point? I'm not saying what are you saying? What were actually thinking when, when you went to the clinic and the day is, here's the date where you're going to become the first GMO human.

Well, I think that like probably how people depict that in their mind is like, you know, we're putting on our capes and we were thinking we are going to save the world.

But actually, there's a lot going through everyone's mind because telomerase reverse transcriptase had not been used in a human, especially as a full body experiment.

So we didn't know for sure uh, that I would live. We didn't know for sure. I'm not, at that point, I believe that had taken the most gene therapy out of anyone on the planet because we had studied all of the clinical trials and that was a very large dose.

Now, since then, they have exceeded that and shown that if you exceed certain amounts, it's toxic. So luckily we didn' do that. but we had already done a lot of estimations.

There are genes that, like fullostatin, that you could put here and here in your arms, and they actually share the protein throughout your blood system, so that the proteins goes into your body, it blocks myostatins and has a myriad of benefits of lowering inflammatory markers and other things.

Telomerase reverse transcriptase is not shared outside the cell, so we needed to hit as many of the cells as we possibly could, and we couldn't afford to do an extremely large amount of gene therapy, but I did do more than had ever been done before.

And we felt, hopefully, that this would be enough. Before you underwent the treatment, you must have had a battery of before labs done. We did, yeah. And I'm sure there were countless, but you were looking, I would suspect, at things like metabolic markers, inflammatory markers measurements of IGF-1 insulin-like growth factor.

So you had to really, and a telomere length I guess as well, obviously you're looking at the delta there. So you have this whole baseline of laboratory studies.

You go to the lab in some country. I don't know if you're able to tell us what country it could probably tell the country, but anyway. So, um, you go there and the needles in your, wherever the needle went, uh, is it intravenous?

Well, the therapies I've taken them intraveinous sense. Uh, But at the time, because, Uh telomerase reverse transcriptase, The one that lengthens the caps at ends of the chromosomes had never been used in a human.

We did over, I believe, 150 injections, the doctor said, all over my body in cancer-prone areas, sun-exposed areas because I really needed to make sure that it didn't kill anyone down the road.

And so it was, um, it felt like I had come in contact with a porcupine. I remember at the end of it, each one of the injections was pretty cathartic considering that I felt like I was in a battle against childhood disease and that we might be able to create therapies that could save millions of people.

And it sort of took that pain away of seeing all of those sick kids and really, you know, like when we work on aging, as you now, we look at all-cause mortality every single day.

And it's pretty grim. So on one hand, there was that, but I remember the last few injections, me saying, yeah, that's enough. I think that those ones were put in my hamstrings.

That was for full astatin. And I'm not sure that we do hamstrings anymore. That one is a more painful shot, but I had already taken so many shots over my body.

I'd had my stomach hollowed out and under the fat in one area to put a pocket of the gene therapy as well. So I was pretty tired by the end of that. But it was, you know, before we went into it, of course, I texted my kids that I love them.

And it was kind of like, you have that moment where it's like everything that we've been building up for has been paperwork and wonderment and waiting for orders to come through and making sure that didn't get stopped.

There were only like three people who knew I was going to do it, not my family or anyone. And you get there and then it is like oh, and now, we don't know what will happen, but it It was important, and we knew it was and nothing was going to stop us.

The criticism in many, much of the media has been that it is more than cavalier, that is was really almost inappropriate. As you know, I'm not telling you something you don't know.

And yet I think you portray yourself as being a vanguard, as a pioneer, And did that help you with the fear? You must have had some fear at the time of the administration.

Yeah. I mean, we didn't know what would happen. We had studied these therapies for two years. And there was a definitive line between the gene telomerase reverse transcriptase causing anything hazardous and the longevity studies.

In the animal research? And the animal research, so we felt very confident that at least this was probably the most promising drug for human life and health extension on the planet.

And for a single gene, it still is. So, I mean, the media, you do something and you risk death and when you survive, your risk your reputation. And I've had no problem with the kerfuffle that it caused because we- Kerfuffle, okay.

Kerfluffle. I think it cause a little, I don't know how diminutive kerfluffles is, but I would venture to say that that's an entertainment, go ahead, sorry.

We disrupted a lot of- There you go. a lot of areas. We disrupted a lotta parallels in biotech. One thing, biothech used to raise a lots of money and used be able to infinitely do mouse studies and those money raises almost were like profit to those companies.

And we came out and said, if the drugs don't work, you shouldn't raise ascent on them. These drugs, science was built to improve human lives. And if it can't do that, then it has no future here.

Launching BioViva and Choosing TERT and Follistatin

We also started to unveil different people's research that they had done for decades to see if that was real or not. You know, so I mean, from how drugs get funded to how we move development forward, we're not going to talk about things anymore.

Thirty six million people will die this year of biological aging. And I am a vanguard. I'm a person who is here to make sure that patients get access.

And since that therapy that I took, we recreated the first licensing partners in the world that would open these gene therapies to medical tourism. We opened investment opportunities for companies who would translate more quickly to humans.

And I went back to school and got my MBA in regulation. and started helping countries actually open up safe and ethical regulatory pathways to get patients treated.

So I am tireless in this. I don't feel shamed by what we did. And I think the only sad thing about it was at the time I really didn't know how to manage the media, but the medias reaction was strong and that shows that we did something very good.

Yeah, either way and thats good, has any research come out more recently that makes you question for example the safety of telomerase? No, the research just is more definitively showing the benefits of this gene therapy.

And now that we have four or five licensing partners, we're about to have the sixth one in the US, which is fantastic. We are seeing just more and more data that is pointing to that these people are healthier.

They have better immune function. As we saw in COVID-19, immune senescence, meaning short telomeres in your immune system, was a lethal cause of death in that condition.

And so when they tested people's telomeres and their T lymphocytes after death, they all had exhausted immune system. So lengthening those telomerase makes it so your immune cells, your T-lymphocites can actually divide to attack an invader.

And when you don't have that, that is consequential. Idiopathic pulmonary fibrosis is starting to get signs that lengthen telomares might be the definitive pathway for these patients.

Neurocognitive we released a paper years ago that was the first dementia study with gene therapy in the entire world where we use Clotho and Lambrase reverse transcriptase to see improved cognition in those patients and this tiny amount of TERT actually made their immune system telomeres longer.

These patients were followed for years. It's a requirement of the company that we get at least five years data. We're seeing healthier, more radiant patients after using this type of technology.

Hey everybody, we're going to get right back to this podcast in just a moment, but I wanted to tell you something really exciting about 40 Hertz light exposure.

We have actually done a podcast on this. When you're exposed to 40 hertz stimulation, this is linked to what we call gamma increasing gamma activity in the brain, in other words, balance in brain which is really a fundamental for brain health and brain functionality.

In fact, an early clinical research using what's called 40-hertz simulation, researchers have reported signals that are consistent with a slower decline in cognitive function in some participants.

So the company that we are talking about, who is in fact, sponsoring our podcast today is Optosudix. Their EVY light is designed to bring a 40 Hertz light and sound into a simple at home routine that you can use while you're reading or watching television.

I use it actually while I'm working on the computer. Optsudx makes it easier to try. So you can get 90 days of risk-free trial with support from their care advocate, somebody who's going to work with you.

And if it's not creating value, then you're able to return it to Optocytics for a full refund. How can you lose? They're going pay the shipping both ways.

Quick note. Evvy is a wellness device and it's not intended to diagnose, treat or cure or prevent any disease. And this episode is, as I mentioned, sponsored by the Optosutics company.

So if you'd like to try it, go to OptoSutic.com and use the code PURLMUTTER26 and get $200 off your order. I think you're going to find this device to be really quite amazing.

Let's get right back to our podcast. Let you brought up clotho. So let's talk about alpha cloth. Oh, and you that is now part that's available for people to receive as well.

Many have called that really the prototypical longevity gene, I guess, taking away some of the spotlight from Tert. But that said, I mean, you know, this obviously getting a lot of press, people are talking about ways of naturally upregulating a cloth.

But here you are in your company, correct me if I'm wrong, but you've created the alpha-clotho gene as to be used, administered at these clinics that you describe.

And so what are you seeing in terms of people who are recipients of alpha clotho? Yeah, we just got the patent approved on that. So this is exciting. In 2020, We decided to work with a nonprofit to see if we could get patients who had dementia with suspected Alzheimer's access to a dual gene therapy, Clotho and Tert.

We were able to do it. Everything was paid for for these patients. They came down to Mexico, I believe in this case, in an investigator-initiated clinical study.

And they were administered these two gene therapies. Now, before they went, I stuck with my promise, and months before I went and took the gene therapy myself.

We've improved on it a lot since the first version of it. we have a better way of administering it, but it's directed internasal delivery with needles.

because AAV doesn't transfect well through the mucosa. So you're injecting it up through to the cribriform or where do you inject that? Yeah, well, the protocol is for the company, but I can ask if you can have access to see that.

OK, yeah, we can do that offline. But so now we're talking about a third for our viewers, a 3rd gene therapy called alpha-cloth, though, in addition, and these patients with cognitive impairment were diagnosed with Alzheimer's.

that they're getting telomere elongation or stabilization and this anti-aging gene that works via phosphate and vitamin D metabolism in the cell. And so what did you find?

well, their cognitive scores went up. And then we didn't happen. I mean, right. We started switching the tests out. we started with the full full steam test, I believe we worked to the mocha and we start to work with just some like, you know, hand drawn up different things that they shouldn't be able to do.

and their cogniz cognitive score went out and it lasted almost two years before we saw the decline again. One of the patients was able to go from assisted living back home.

Some patients knew their loved ones again. Patients started painting. One patient had no benefit, unfortunately. We don't know why. But again, it's dementia with suspected Alzheimer's.

So we're not sure what happened there.

The First Self-Administration of Gene Therapy

It's not 100% treatment and it wasn't a cure. But it improved these people's lives significantly and the families were all super happy to be part of that.

And it helped us look at the data and look how we can improve those therapies. Now we are working with Dr. Sheldon Jordan, who is a super famous doctor working in neurosciences.

and we're working on a new protocol to get more of the gene therapy to the specific areas of a brain, the hypothalamus in one case and the hippocampus and in the other case, in order to drench the lining of cells that are responsible for memory.

Well, I would say that just for our viewers, let's be clear. We don't have an FDA approved meaningful treatment for Alzheimer's disease. It does not exist.

The drugs that are currently used are drugs, that target beta amyloid. Do they effectively help reduce beta in the brain? Yes, they do. But that doesn't necessarily translate into any change in terms of cognitive decline.

Elizabeth, what you just described is an improvement in cognitive function. So, you know, it's breathtaking. Is this the answer? I don't know. It's a piece of the puzzle, I would say.

Absolutely. We couldn't, we shouldn't… It is not a definitive cure. Of course not. Upon autopsy, this paper came out like a year after we did this study, which was very validating.

Upon autopsy, patients who had all of the signs of Alzheimer's, the beta amyloid plaques and the shrinkage of brain, who didn't have cognitive issues, did not show the cognitive symptoms of disease, were all natural alpha-Clotho upregulators.

So, just working with… Interesting. You need to say that again. These people naturally had higher levels of this longevity gene called Clotha, And these people did not have cognitive decline.

That's right. Though they had the so-called hallmarks of Alzheimer's disease that had they have had cognitive declines would have ascertained the diagnosis.

Yeah. We would love to work with a company that's also doing the beta amyloid plaque removal and use the therapies with it. But right now, you know, we'll be working on exacting the gene therapy, getting more of the Gene therapy to the centers of brain that need it with the better delivery combination with this ultrasound guided therapy and exosome encapsulated AAV.

And I believe that, then we will see major score changes and then we will start working in combination with other companies who target other problems of Alzheimer's.

I mean the beta amyloid plaques are a side effect and it could be a site effect from a lot of different things that are going on. Well, it takes me to, uh, I need to move the conversation a little bit laterally because, you know, we now understand that the accumulation of beta amyloid is really an immune issue.

It's a shift in the brain's immune cells, the microglial cells from a being, in a phenotype or a, a description in which they reduce the formation of Beta amoyd and actually are able to phagocytize it or basically digest it away.

And, The activity of these microglial cells is really strongly influenced by their metabolism. And when I say metabolism, I'm talking about their mitochondrial function.

That leads me to another type of gene therapy, and we talked about it before, called PGC1-alpha. Yeah. PG C1 alpha is a gene-therapy that targets mitochondria, biogenesis, the growth of new mitochondria and fosters better energy metabolism utilization on the part of cells throughout the body, including the brains, immune cells, to

microglial cells.

As such, one would expect that amplifying PGC-1-alpha expression would allow these microglial cells to do good things as it relates to getting rid of beta amyloid.

And therefore, gene therapy would then be something we should consider targeting these microbiome cells, their mitochondria, with PG1 alpha. I think you're doing that work, aren't you?

Yeah, so PgC1 alpha was the next place we had gone to. PGC 1 alpha had been looked at and discovered around basically creating better function in Parkinson's patients.

We wanted to re-establish it as a potential therapy as the third arm to this dementia study. So when we looked at telomerase reverse transcriptase, we look at the research behind it, it actually seems to have a reparative effect on mitochondria in the same cell by reducing oxidative stress.

Now that there is a complete feedback loop between mitochondria and a lot longer telomeres. And so it had been shown in research, this feedback loops.

So we were excited about that. Also lengthening the telomerase created less tau tangles and neurons. and so we're excited that that alpha clofo was associated with less beta amyloid plaques.

So there is a cross between. But when we did the therapy, the complication of the protocol that's harder to describe is we were targeting different cells with different gene therapies.

The neurons and the microglia were targeted with a different AAV. gene therapy deliveries and the microglia were targeted with the alpha-clotho. Today we have a completely different association with a set of cells that we will be targeting even though we still target those cells and then PGC1 alpha So yes, it's associated with mitochondrial biogenesis, turning white fat to brown fat with this excessive amount of little mitochondria that darken the stain.

But we'll be talking about, we can't talk about yet, but we've got a new way to do this that is going to be a delivery in which creates a certain cell type to donate mitochondria to all the cells around it.

And we're gonna use gene therapy to do that, but we can't talk about it quite yet. Hey everyone, we're gonna get right back to this podcast, but first I wanted to share some information about olive oil.

You know that I love olive. So here's a good tip. The most delicious olive that you can get is olive, oil that comes fresh from the farm. This one, the olive is at its peak flavor and all the nutritional good things that we talk about, polyphenols, et cetera, are really at their peak.

When you buy your olive oil at the supermarket, you are getting an inferior product because they have been sitting on the shelf for months and they basically grow stale.

And that's why I've been getting my olive well direct from small award-winning family farms really around the world. Thanks to a fellow named TJ Robinson.

I have done Instagram live with him in the past. He's known as the olive-oil hunter. he's sort of the Indiana Jones of olive. He is able to source farm-fresh oils that are vibrant, they're healthful, grassy, incredibly delicious, and you can use them on whatever you want.

On salads, fish, vegetables, meat, even like I do, on scrambled eggs. So if you wanna taste the difference that freshness makes, TJ is gonna send you a full-size bottle, which normally will cost you $39. He's gonna sent it to you for free if them just a dollar that will cover shipping.

And that's how he then is able to introduce all of you to this fresh pressed olive oil club, a club that we are members of. So there's no commitment, just send him a $1. He'll send you a full bottle of olive.

You just want to go to getfresh389.com. That's get fresh 389 dot com and he'll make it happen for you. Let's get right back to our podcast. Yeah, so we do know that that's actually already happening within our brains, that cells that have neurons that had defective mitochondria receive through microtubules donor mitochondrial from healthier cells.

And that one of the LK variants that is associated with increased risk of Parkinson's, for example, is a failure to form those micotubule. So this is something that is already going on.

So I think I heard you say you're developing a cellular donor scheme whereby a cell can be introduced that can then donate healthy mitochondria to cells that need it.

Yeah, yeah. And these will not only be healthy mitochondria, but they'll be, you know, from your own cell line. In one case, we're also doing a donor cellline where we'll have seven lines of cells for donor mitochondrion.

But yeah, one of them would be donated from our own cells. Healthy, healthy, mitochondrial. Even if you don't have them today, We can make your cells make them.

Safety, Media Backlash, and Clinical Rationale

So just a comment for viewers, we've talked before about this PGC-1 alpha, this protein that then is helpful as it relates to your mitochondria and how we up-regulate PG-C1-alpha, of course, are things like exercise, getting up sleep, reducing stress, all the normal players, caloric restriction, et cetera.

But what we're talking about, Liz, is a way of keeping that switch on all of the time in a much more effective and dramatic way. So you mentioned the delivery system.

And I think traditionally what you and your company have done is called an adenovirus sort of delivery. What does that mean? I mean, some people hear adeno virus and you're injecting a virus that sounds kind of scary.

Tell us what it's all about. Well, there's two. So there is adenovirus, which is a large virus, and it is the one that kind of shut down gene therapy in 1990 due to an ill effect.

There's something smaller that eats on adeno virus. It's about this big in comparison and is called adenossociate virus and the thing is is that most people have seen it.

it has very low immunogenicity in humans. and it can deliver genes evading the immune system with immune suppressants, of course, and deliver small gene loads.

And so it became the darling of research, I believe, in the late 1990s and then started clinical trials in their early 2000s. This is the therapy that we use because outside of companies in the United States who have used two larger doses, it's actually a very safe gene therapy to use and the worst case scenario is something like a fever and then very little uptake of the gene and that's probably what happened in one of dementia patients.

There are other delivery mechanisms, one, of course, using lentivirus. Tell us about the big difference in terms of integration between the adeno-type delivery system versus the lentovirus type.

The reason that people love adenossociated virus is it Vastly, 90 some percent of the cases creates what's called an epizome. So it means it doesn't integrate into the human chromosome.

And when it does, it's been found in places that have no oncogenic effect, meaning it Doesn't cause cancer. That's really fantastic. Lentivirus is a larger virus, but it is retrovirus and our genome unfortunately has a lot of docking sites for retroviruses and they're known to cause cancer.

So lentiviruses immediately wasn't something that we wanted to work with. We wanted just to just work something safe that didn't disrupt the natural human chromosome.

And so that's what takes us into the area of adeno-associated virus. Other clinics seem to be offering plasmid type delivery systems. Can you walk us through what that means?

That means a very, very short acting amount of just a genetic string. And so whether or not, you know, I don't really know the efficacy of those. They may be viable for short term expression, but they're not what the science is based on, nor are they what goes through clinical trials in the US for genetic disorders.

So in terms of continuing to do things in your body, how do you measure the gene therapy that you underwent initially and continue to get? How do measure that it's still doing something in you?

Yeah. So with telomeres reverse transcriptase, we year after year have looked at my telomer length and we look at that every year. And we just see the slow lengthening.

And then every time I take the therapy again, we see a bump in telomere length, which is really fun because that kind of shows us biologically how that's working.

With other gene therapies like alveaclotho and folistatin, we actually look at protein in the blood. So we have a lyso-kits run in third-parting analysis to see if the protein levels actually changed.

And so that's how we can see that it has. Then we continue to monitor patients over years to make sure that its up. And then we also use that for redosing.

So we're the first company in the world that's done AAV redosing successfully. We can show with our protocol that we can get increased protein expression.

And so in those patients, we like to watch it too. Now we were seeing that already with the telomeres, but we didn't start redossing AAv until five years after my first gene therapy because we had to do a lot of work to figure out how we could do that without having an immune response or rejection.

Mm-hmm. And I saw images, cross-sectional images. I believe they were of your thigh. Yeah. You know, some pretty significant gain in muscle mass. It's great that we gain muscle, but I think in the context of what's the opposite of that sarcopenia, one of the sarcomenias or the progressive loss of muscle is really powerfully associated with a lot of degenerative conditions, not the least of which is cognitive decline.

And, you know, we now embrace muscle tissue as being virtually an endocrine organ, creating great chemicals for the body and specifically for their brain.

So the more muscle mass we have, the repositor we, have the internal pharmacy, if you will. That's, I think, another argument in favor of the folistatin slash myostatin approach to gene therapy.

You know, David, what I love about your podcast, just being here right now is usually I have to go all the way back to basics and doing research on you.

I knew that I didn't. Oh, you know, and, this isn't like the first time, first rodeo that you've done. And you now what's actually happening for patients and it's just, it is such a delight.

It's very rare that I just get to answer answer short, answer questions. But you're absolutely correct. Follistatin, it's turned out since I did that in 2015, is actually involved in a myriad of different biological processes, including brain plasticity.

And that was not known. Reducing TGF beta, inflammatory markers, that is not know. It's believed to be protective against both breast and prostate cancer.

that when I did it was not known. So this single gene is very consequential for all people. And people would watch and they'd say, well, I can go work out and I eat right.

I could upregulate some of these genes, but we have to remember, why are we here? Those are the laws of diminishing returns. you're not going to get past 120 doing that.

The body starts making less of these proteins, we call it loss of gene function over time. And we can see that even in healthy subjects, We can that over times.

So that's why in case anyone's wondering why he would bother doing a gene therapy over, you should work out, You should eat well. But if you can increase a natural human gene function to help you support your activities, to support you diet and exercise.

Why not? You could see it either way. You can see either one of them being a supportive role in your life. Hey everybody, we're going to get right back to the podcast, but I have a very important message for you.

Y'know, why do we exercise? Why do pay attention to how much sleep we get and the quality of the sleep Why are we looking at our diets? Because ultimately, we are sending information to our genome.

We are affecting how our genes express themselves. I like to know about my genome, that's why I'm a real fan of the 3×4 genetics test. Why? So you can learn about your genetic predisposition.

You know, in my world, as it relates to the brain, there are some genes that in fact are associated with increased risk for neurodegeneration.

Clotho, Dementia, and Cognitive Improvement

A lot of people know their APOE status, for example. But I want to be super clear that this is not a genetic determinant. Maybe it's a generic predisposition, but what you're seeing here happening, these are our lifestyle choices that actually change our gene expression.

You should know about your genome. you should get the three by four genetics test. We'll talk about how to do that in just a moment. But there is a health program that they offer you as well.

When you get your gene sequence with three-by-four genetics, It's not just getting this information, but they're going to tell you what to do with that information.

So I think that's really very important. Head on over to www.3x4genetics.com. They have a special offer for you and all of you podcast watchers. Let's get right back to the program.

Getting back to the muscle mass, one of the myokines that's produced with greater muscle, produced in all muscle but more muscle more production of these myocines is irisin.

And we now understand why irisin is such a tonic because getting back what we were talking about before, it aids in the conversion of white fat to brown fat and that Fat is brown, as you pointed out, because it has more mitochondria.

So that's why exercise is mitochondrial therapy, not just through activation of PGC-1 alpha, but through a resin as a myokine converting white fat, we call it the bronzing of fat.

and basically increasing mitochondria and therefore energy utilization, keeping blood sugar balance. That's good for the brain as well. So there's a lot to think about.

Since you were first, there have been obviously lots of others. What is your sense in terms of what people are experiencing? Well, when we work with companies, we license technology and then there has to be a level of safety and they have to follow the protocols and everything else, but we don't want to know who the patients are.

We're not interested in that. we do not want any direct correlation, We just want see the data. Occasionally these

patients walk up to me and it's amazing to hear directly.

So we see numbers, you know. They tell us their life experience and how it's changed their lives. So Alva Clotho has a myriad of uses and one of them is for chronic kidney disease and cardiovascular disease.

And we had a patient who was on the donor list, on a list to get a donation of a kidney and had to and immediately needed one and he took the gene therapy directly, image guided gene to the kidney he came off the list for two years and to see him in front of me and say, and he wasn't sad about it, you know, because like to me, that's a huge disappointment.

He came up and said, I'm the person who took the gene therapy for chronic kidney disease. I needed an immediate donation. And I was two-years off of the donor list.

But I am okay with that. But he was okay with it. It got him two years. And it's really inspirational to see people who have Parkinson's disease or Alzheimer's and being sent videos from them being confined to kind of pretend jogging down a hallway with no walker.

And yet it's not a cure. And so it is killing me, literally, that we have to move faster. So, you know, I get to see the beauty of the short-term response, and a year or two to someone means a lot, but we need to do better than that.

That's just not success. You know, it makes me think about many, many years ago when I was a much younger man. It was learned that a certain part of the brain was deficient in glutathione, an antioxidant, a detoxicant that does various things.

And it was seen to be related to Parkinson's. So I thought, simple enough, if Parkinson s brains have low glutATHION, let's give them glutATHION and see what happens.

you know, cause people were, it's relatively safe. So I began doing this to my Parkinson's patients and filming them and posted on something early on called YouTube, crazy.

It was very earliest experiences on YouTube. You pioneer. Pardon me? You pioneer. Yeah, I know. And so, you know, there's the video is this very sweet gentleman who is really, aggressively compromised by his Parkinson's gets his glutathione shot and then we video him I think 20 30 minutes later.

Now he's walking down the hallway and he is smiling and happy. His wife called me a week later and said, he has been up on the roof replacing shingles.

And I'm thinking, that's not exactly what I wanted to hear. Two things happened after that. First, people were kind of amazed. But some of the comments on YouTube were that he was obviously a paid actor.

Okay, you know what it's like. The second response was that this is kind of an off-label type of thing, and why are you doing it? And there was no downside.

So I know what it's like. I really have been there. And every once in a while, I go back and look at that video and think about, that's what we need to do.

We need push. It's nowhere near what you did. I know it is because the thing is you saw something. You saw a mechanism of action that could help. And so those patients need access to that.

What happens is, you push And first there's nothing, there is no echo and then the echo comes back and tries to knock you off your feet to stop you from doing it.

And it doesn't make sense because these are the type of things that we have to band together and figure out how do we cure these people. Because today 110,000 people will die of biological aging and not to mention the children and a myriad of accidents that might have been foreseen or stalled if we could think faster or, you know, had a

better, smoother running community out there.

And we are the vanguard of making sure that this happens and that we don't get knocked down by someone saying, You shouldn't do that. You should do for somebody who's dying, to somebody whose suffering.

It's their choice. And when you have a patient with, as you know, with dementia or anything, you're not just saying, hey, your not cognitively functioning well, how about a gene therapy?

Or how bout some glutathione? You actually have to get their family to sign off of it, on it and their medical doctor. So it's not, just you are not trying to go around, You're trying not to hurt anyone, Your trying save lives.

Yeah, I remember when that glutathione video finally was posted and all the people were raising their eyebrows. The comment was, well, you really need to do a double-blind placebo control trial and publish it in a peer-reviewed journal.

And I thought, it's working. Why do I need you to that? OK, we'll do it. We did do. It and we did it at University of South Florida and the movement disorder clinic, and it proved incredibly effective.

And yet, you know, people were aware we are at this time, here we're 2025, that we still just treating symptoms, we just treated rigidity and tremor, were not treating the underlying disease process.

And I think If we focus on what you're doing more than anything is treating the fire, not just the smoke.

PGC-1 Alpha, Mitochondria, and Future Delivery Methods

You're treating upstream issue here. The primordial issue, you know, the manifestations. We in America, in the Western cultures, just focus treating manifestations of disease.

You know, in, your son's case, it's treating the high blood sugar. It's not treating, the autoimmune loss of the beta cells of, of pancreas, It is treating.

The manifestation that's what insulin does. That's, what metformin does in the type two diabetic, but we're missing the boat on the underlying problem.

Let me, explore with you, where we go in future. So why we look at these gene therapies is because they're treating biological aging at the cellular level.

We're not, targeting genes that just have some sort of effect of masking a symptom. We're actually trying to make the cells younger. And so that's where we're gonna continue to go.

So there's 12 agreed upon hallmarks of aging. It's going to grow because it's a wish list, right? And it the things that are driving the processes of cellular degeneration or the inability for the body to restore itself and regenerate.

And so when we look at that with gene therapy, we have so many gene candidates and there's so of those hallmarks that we can target so far. So we're looking for more genes.

We're are looking to put together more combinatorial gene therapies. Of course we to raise money to do all of this, but we are very, very careful. we don't let any investor in because we so agile we can do first in many areas that larger companies can't do.

So where we're headed is the combinatorial gene therapy, hopefully sooner than later, that keeps your cells in homeostasis, keeps them from degenerating faster than they're regenerating.

And so what that looks like for humans is a homeostasis of the body in which you are not degenerating. You're not getting weaker or frailer over time, but you're staying in a state of ultimate health.

And then around that will be human enhancement. So every one of these gene therapies, already we go under fire because they are half enhancement and they're half preventative medicine.

And that's what therapies of the future are going to look like. So if muscles are stronger, it's an enhancement, but it is also a preventative medicine against type two diabetes and metabolic disorders.

You already talked about if your mitochondrial function better, It's the same thing. It is an enhancements. Think faster, move faster. Do things better.

But it will protect you against cellular metabolic issues. Lengthening your telomeres, that is by definition creating immortality, even if it's just because cells that couldn't divide 10 extra divisions can.

That's called creating cellular immortally in a dish. We're creating that in the human body. And that's to break lifespan. This bottleneck limit, because if you're healthy, You shouldn't die of anything.

You should have this wall that you hit. I mean, mostly we talk about health span and not lifespan, but they go hand in hand. And so, you know, we want people to ultimately live as long as they like and enjoy their life.

There'll be some comment under here that will say, well, I can't stand this world. that's clinical depression. And we're very sorry for you. We're working on those areas, too.

I have my sights on a lot of companies that are working towards depression and as those, you know, and things that we want to stop all things, that make life not worth living.

That would also be mental state. and I do think that the world will be a better place when people are living longer and they have to treat each other better.

One last thing, how we, how you and I got connected. So several weeks ago, I'm on the elliptical machine and learning about gene therapy as one would expect a person would be doing on The Elliptic Machine, of course.

There was Peter Diamandis introducing you, and you gave a talk And I did what everyone would do, of course. You know, you get you see a talk to very interesting.

Can you reach out to personal? I'm going to connect to that person and chat. So for our viewers, Liz and I spoke, well, like a week or two ago, and. I've enjoyed every minute I am.

And so honoring of what you're doing and your way on one side of the bill curve, which is a great place to be. The mission isn't to be thinking outside of the box, the mission is to make the boxes bigger, more inclusive.

And I think that at the end of day, that's got to your goal, and it's going to a long time until this is standard of care. but at least let us begin to embrace the potential here and check the boxes of safety and make these things, you know, put them in a place that we can make them verifiable and do all the work that needs to be done so that they become acceptable.

But I am very, very thrilled with what you're doing and grateful that you spent time with us today on the program. Absolutely. I mean, it's just so wonderful to And as the years go by, you're going to see one more and one regulatory system open up to treat biological aging and use gene therapy.

And in 2024, we worked really hard and the Bahamas passed a law to do such things and they will be running clinical trials as you would expect. to get these drugs into the U. S.

FDA regulatory system.

The Future of Combinatorial Gene Therapy

And then I also have a best choice medicine plan for the human health and services department. We're trying to that past as well. I believe there's even gonna be a march in Washington, D. C.

about it in 2026. Wow, there is a lot happening. How can people stay on top of this evolving story? Where can they go to keep up to date? They should follow you.

I imagine you'll continue to have conversations with me as we have new information. And then, BioViva, the company has its own social media. Most of the things on there though are value added.

We, I specifically read papers almost every day and I make it so you are getting what's happening in science today. It's not necessarily about the company and 99.9% of the cases science moves too slow for that.

So I wanna tell you everything out there. I want you to get excited about that. We've got a new project called Trial Atlas that's going to be coming out.

That's an AI system that'll help people find access to clinical trials. Learn about the genes behind their diseases. And of course you can always reach out to any of the clinical partners if you're interested in what that looks like.

But we always suggest that you go through a proper US clinical trial if it's available. first and foremost before you go outside into medical tourism.

And that's about it. So just for what we're doing and we'll keep you updated. You can get on newsletters or whatever, but following you is one way to do it There you go.

And I will tell our viewers, if you look at the bottom of your screen right now, you'll see some great websites to go to follow and learn more about this.

Liz, thank you so much for your time today. This was really fascinating. That's for sure. Thank you for having me. Okay, my pleasure. Thank for everything you do.

I appreciate it. Well, that was an extraordinary conversation, wasn't it, with I would say a trailblazer reshaping what we believe is possible in medicine.

Targeting aging at the genetic level isn't science fiction anymore. And as Elizabeth Parrish has placed herself at that center of that whole evolving story, there's certainly a lot more that we will be learning in the years to come.

I think really in short term. Whether or not we agree with every approach, the questions that she is bringing to our attention and the courage that's she's shown, and I call it courage, these things signal a future where aging may truly be seen as a modifiable disease state.

So thank you for joining me here on The Empowered Neurologist. I'm Dr. David Perlmutter. We'll be back next time. Stay curious, stay empowered, and remember that the future of your health really is being written right now in terms of the choices that you are making.

So again, thanks for joing us.