Origin Of Disease Is Hiding In Your Cells! Doctor Reveals How To Fix It Now l Dr. Natalie l Ep #419 Transcript

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Introduction to mitochondria and the guest

Hi, I'm Natalie and I am your host and welcome back and prepared to have your mind blown, especially if you're really into the cutting edge world of, well, mitochondria really.

So look, let's face it. I have the incredible privilege of speaking with world-class experts across every corner of longevity. So blessed and grateful I get to do that and, but every so often a conversation comes along that truly expands the way I see how human health is going to benefit from some of these technologies.

And this conversation really is one of those. This is exactly what happened. Today's guest is, I literally couldn't wait to share this episode with you.

So the guest Dr. Natalie Yevgi Ohana, and she brings an extraordinary depth of knowledge on mitochondria, breaking down how these tiny powerhouses influence fertility, hormone production, immune function, the brain, even the aging process itself.

We also explored the emerging science of mitochondrial transfer. That's right. transferring new mitochondria, baby mitochondrion, into aging cells and what this could mean for the future of longevity medicine.

And again, it touches on every area from rare diseases to fertility to aging to neurodegenerative diseases. It's nuts. So you guys are, I just know you're going to love this.

Now, this episode is brought to you and we need to thank our sponsors by Oracle, by Wizard Sciences. So this is their new hemp oil ozonated supplement, which is phenomenal.

Magnesium breakthrough from our friends at Bioptimizers. You guys all know by now that Tart cherry is literally my favorite product in that line, but all the mag breakthrough products are fantastic.

and of course Vitaly for their GHK copper skincare line. So all of them have special offers just for you. All you got to do is check out the show notes below for details.

And until then, enjoy this episode. Let's dive deep into mitochondria. Welcome to the Show, Dr. Natalie Yiggy Ohana. It is such an honor to have you here and thank you for taking the time to speak with me today.

Thank you so much. Believe me, a lot of people are going to be excited about this, including me. So we're going the tiniest little structure in the human body, I think, or one of them.

It's not the tiniest because it's got tiny things inside of it, but the mitochondria, what we talk about, the powerhouse of the cell, which I, think it is such a hot topic and has been, in many ways, gets reduced to less than what it.

But this is your career. This is what you've built your When did you decide that mitochondria was going to be your topic? I think mitochondrion actually decided I'm going too.

I am completely controlled by these organelles and fascinated. And it took a while. an expert in in vitro fertilization, and I started to study the role of mitochondria in embryonic development.

And I was fascinated by the fact that actually mitochondrial only inherited by their mother. Even if we do an in-vitro fertilization where we inject the sperm with the mitochondria into the oocyte, eventually no leftovers of this mitochondrial are there.

It's only maternal and it is selecting against the paternal mitochondrion. So that's like, it's enormous and there is a huge responsibility for us women to transfer this by the chondria to the next generations.

I have four daughters, so they will transfer my mitochondria. That's incredible because, and I mean, obviously we know that the ovaries are so dense in mitochondrion, right?

Which makes sense on the fertility side. Very important because there are many supportive cells and tissues within the ovarie to make that one special oocyte every month to fertilize.

And also when we go through first the puberty and then the menopause stage, what is the role of the ovaries in the hormone production and the mitochondria aspects of it.

So when I started my PhD, I was working actually on reproductive organ systems. and mitochondria. And the fourth role of mitochondrion that I learned about is, of course, energy production.

We'll talk about it greatly in a minute.

Mitochondria, fertility, and hormone production

But the role is mitochondrian steroid hormone production, so I don't know if you know, but the first hormone that is produced is in the mitochondrial And I studied a protein that transfer cholesterol from the outside of the mitochondria into the mitocondria, where an enzyme converts this cholesterol to prognanolone.

Prognenolones is now the precursor of steroid hormone production. All sex hormones, I mean, all glucocorticoid and mineralocortic and the sex hormone, All of them start in a process in the Mitochondrial.

And with mitochondrial dysfunction, of course, you get disrupted hormone synthesis. So it's super critical. There is no life without all these hormones, and the production of hormones by the mitochondria.

And there is not use in living without sex hormones and what it does to our bodies. Well, to everything. You've just blown my mind and I'm sure a few others.

cholesterol is used in the mitochondria like all these people like this race to the bottom on cholesterol as we age which ultimately could be I mean I don't know for sure but and I'm not saying but which could ultimately be feeding into the pathways that lead to aging process and decline not to say we all have to run around with high cholesterol but an important role for cholesterol both in building our membranes of course and drafts that are important with cholesterol inside them for signaling within the cells and outside the cell, but also for this very important task of steroid hormone production.

You cannot do that without cholesterol. That's incredible. So was there an aha moment when you decided that, you know, when your like, okay, medicine is completely underestimating mitochondria I'm going to do something about it.

Yes. So there are two moments. One was I was doing my PhD on this life creation with mitochondria and the fact that they produce energy and steroid hormones and there is no life without them.

And then I did my postdoctoral fellowship on triggering cell death through the mitochondria. So if a cell has a damaging response, mitochondrion immediately they behave like these smart bombs and they release content that starts and cell suicide mechanism that is irreversible.

And then that was the first aha moment. I said life and death controlled by the Mitochondria And that's where I should focus my next life on. That was the first aha moment, really realizing how important they are.

And then the second one came after I finished on my academic training, and I decided that there is such a huge unmet need in the whole field of mitochondrial dysfunction that the problem was that no one could really say

whether someone has a mitochondria dysfunction or not, because there were no methods to measure that.

And then again, we are all going to suffer a mitochondrial dysfunction as we age. So age is a kind of mitochondrion dysfunction. I said, no one treats it today.

There are no therapies to fix mitochondria dysfunction, that's where I'm going spend my life on. and I sat at home for like nine months just thinking and i read all these books and all and went back to the early days of how mitochondria were identified and there was the Lynn Margolis.

Lynn was a great scientist, she was woman. In the 1960s she tried to publish her endosymbiotic theory meaning Okay. So tell us about it. Give it to get, tell her publication about the endosymbiotic theory that we all know today.

This is a consensus. Absolutely. Mitochondria used to be bacteria, independent bacteria that entered into the cell during the evolutionary process. One and a half billion years ago, the cells start to develop.

The nucleus wasn't there yet. It was just DNA flying inside the cell. And now this bacteria enters into the self. Usually, when a bacteria affects a cell, they will multiply until they'll kill the host, and then they would live to infect another cell and so on and forth.

In this case, mitochondria entered into that cell but they did not multiply into infinity. just a little bit and then they actually became an independent part of the cell and they transitioned most of their DNA into the nucleus and maintained just the portion of it within the mitochondria.

Now they can no longer live independently, they have to reside within their cell, and this is the symbiosis, What the mitochondria did was actually enabling the cell to survive in an oxygen-enriched environment, which is what we have here on Earth, right?

It's an Oxygen-Enrich environment. And before that oxygen was toxic to cells, but now mitochondia consume the oxygen and the nutrients and produce energy for the cells.

And this is not chi. This is a chemical molecule that is required for everything that happens in the cell. It is called ATP, or adenosine 3-phosphate.

And it's a molecule, that it is needed for, everything the cells is doing. Every pump, every enzyme, Every protein formation, nucleotide formation. every DNA replication, Everything requires this ATP.

and that's how life began. And now there are creatures on this planet that consume oxygen, produce energy, thanks to the mitochondria that we have residing within our cells.

My aha moment, the second one that I promised, came when I suddenly realized that actually mitochondrial will vary selfish, they behaved like a selfish gene.

They wanted to integrate their DNA into the nuclear DNA to protect it. And now the nucleus produces most of the proteins for the mitochondria. This is the perfect symbiosis, and this is why I love it so much.

My aha moment was, can we use mitochondrion to fix diseases? Can I isolate mitochondrial from one cell type? and apply it to another diseased cells. Will they go in like they did during evolution?

And that's it. That's how Minovia started. It was a crazy idea, but I just had to test it and it was true.

Endosymbiotic theory and mitochondrial origins

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 your personally, but unfortunately I can't. 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. What was that scientist's name you said in the 1960s and who didn't publish her paper and was it a guy?

So Lynn Margolis was a scientist in the 1960s, and she suggested the fact that there were endosymbiosis happening between different species. It is also the chloroplasts inside plant cells, which are also evolutionary.

They used to be also germs and the mitochondria in our living cells. So she suggests that that is a structure that enforces these two species to come together in order to supplement a missing function.

That missing was the use of oxygen and the outcome was creation of the energy, the ATP. She tried to publish this in 15 different journals and she was rejected.

In the 16th time, that was accepted. And eventually, it's a common practice for everyone. This is the endosymbiotic theory. When you look at the mitochondria under the microscope, they look like bacteria.

They are the size of bacteria, and they contain their own DNA, just like a bacteria and their DNA is circular, like bacterial DNA. So everything about them says that they used to be bacteria they actually can replicate independently from the cells multiplying.

Yeah, they do the fusion and mitobiogenesis, and they're very independent within the cell. Yeah. No, I love mitochondria. I think they are the cutest organelle personally.

Surprised you don't have a big poster on your wall. But I have them all over. Yes. You're going to have to get mitochondrial jewelry and stuff. And I have a mitochondrial jewelry, actually a very special stone that was brought all the way from Africa, from Ethiopia.

And then an artist created a Mitochondria for me from that stone. I actually should have worn that. One of my partners actually created that jewelry for it.

It's beautiful. So let's talk about how understanding this endosymbiotic origin of mitochondria, how should that affect how we think about chronic disease?

Or does it come into play in terms of how were thinking about chronically? Because it's so interesting, right, what you said earlier, that you've got these incredible branches like fertility, chronic disease and mitochondrial disease specifically, but just any chronic.

And then just the pure process of aging. But when we're talking about chronic diseases, does this endosymbiotic origin of mitochondria have an impact on how we need to be thinking about it?

So the first thing, of course, the fact that they are consuming oxygen and using the nutrients that we eat to produce the energy, we need to be mindful for the quality and the amount of oxygen that you are breathing, and how important is that.

And it's not to wonder that when we are doing sports and when you were doing yoga and We are singing. We're just loading our bodies with oxygen. It makes us feel good.

That feeling good is actually boosting our mitochondria with energy. And eventually that's the outcome. When we are feeding our Mitochondria, there are huge differences between if we're absorbing now carbohydrates, glucose, for example, or if we are now feeding our mitochondria with free fatty acids, because you know that the number of molecules of energy that you produce from a carbohydrate versus from free-fatty acid is completely different.

You can get up to three or four-fold higher numbers of particles if you consume free fat acids versus glucose. So I think the mindfulness of what will cause our mitochondria to be more active or more efficient in energy production, this is super critical.

And we do know, I mean, we all feel the drop of energy and the start of frailty as we age. We know that in chronic diseases, people feel weak, they are less active.

So how do we know? This is mitochondrial dysfunction, eventually. We feel the lack of energy, we feel tired, you feel exhausted. This is practically mitochondrial dysfunction.

And why is it happening? Because mitochondria carry, as I mentioned, their ancient DNA that came from their bacterial origin. That DNA is circular and exposed.

It doesn't have the three-dimensional structure of our nuclear DNA with all the proteins that are protecting it, and it is very, very protected.

How mitochondria shape chronic disease and metabolism

It gets wound up and it has to unwind to get to work. Yeah. And there are systems to fix errors within our nuclear DNA that doesn't exist in the mitochondrial DNA.

So the fact that it is open, very sensitive and for instance, from the environmental instance and that there is no machineries to fixed damages in DNA of the microchondria actually cause it to be very, prone to mutations in mitochondria.

Now each mitochondria have multiple copies of mitochondrial DNA, and in each cell you have many mitochondrias, so even more copies, of the mitochondrian DNA.

And sometimes if it's just several of these are mutated, you won't feel anything. But with time, The process, the natural process of selection of the good mitochondria is harmed with the age.

This is the mitology, so it's going down. And what you see is accumulation of damaged mitochondrion within the cells. Those mutations in the mitochondrial DNA accumulate more and more as we age, And those mutations are what's causing us to produce less efficient energy and eventually even more reactive oxygen species because of the non-efficiency of energy production.

And that further circulates and increases the damage within the cells, all eventually connected to mitochondria. So that's the origin of why chronic diseases eventually evolved from this DNA that is so sensitive.

What are the things people can do to support mitophagy, mitochondrial biogenesis? What other things we can to do that? But I want to go back to something that you said earlier, and you talked about the difference in ATP production between glucose as a substrate fuel versus free fatty acids and then ketones.

Do you have a position in terms of whether people should follow more of a ketogenic diet in certain states? Or is this just, or does this more speak to maintaining metabolic flexibility so that the body, like the problem is most people live in sugar burning 90, 100% of the time, right?

Because they taste good, they make us feel happy. And the worst part is when we get sick, we go there because it makes us, it gives us… I mean, I've just gone through something.

I can tell you, We do that when we are stressed, right? And we feel bad. Well, because my cortisol is high, my adrenals are taking a hit. You know, like, I feel like going keto right now might not be smart.

But maybe that's a trick. Maybe I should be thinking more on the keto side. And I'm just curious in if you have an opinion in these chronic diseases or in situations where the mitochondria is really impaired, is there an argument there that says that either introducing the right exogenous ketones at the time or maybe following a ketogenic diet, this might be a use case scenario for it?

Yeah. I think ketogenetic diets are very dramatic and very drastic. Yeah, they're problematic, like they are not perfect. Yeah. And I think we should, but definitely I do believe in the concept of more free fatty acids versus carbohydrates.

There is a very simple explanation to that. This is why I so strongly believe that what happens is that when we consume carbohydrates, our cells immediately, there is huge responsibility to remove the glucose from the blood, right?

It cannot stay around. That's what we want. Once it goes into the cells, it becomes a source of energy. And then it will create ATP in a non-efficient way, but through the glycolysis, you will actually get ATP produced.

You'll get about nine molecules, I think, of ATP. Then if it go into mitochondria, we'll have 36 molecules of ATP if the pyrovert goes in. So the responsibility of removing the glucose from the blood and now creating energy through the glucose is huge.

And if you consume both free fatty acids and glucose in your diet, you now eat a hamburger, okay, with the bun and the cheese and burger itself. So you have it all mixed in.

What the cell will do, it will use the carbohydrates for energy production. and the fat will go being stored because they don't need it now. They have enough energy from the carbohydrates.

If you eliminate the carbohydrate from your diet, now you are educating yourselves that they need to use the free fatty acids in order to produce the energy.

the cell will multiply the mitochondria to the amount needed to face the flow of free fatty acids that you are now consuming, and then they will not be stored.

What happens is currently when you start a paleo or a keto diet, you're educating your cells now, to increase the content of mitochondria so they can face the amount of free fatty acids that currently are consumed.

They will not be stored, they will actually start using your fat tissue because they don't have the glucose now and there is a requirement for energy that is coming from the fat.

And this is why this re-education actually shifts the balance and people start to lose weight and it's crazy. It's completely against what we are being taught.

However, there are carbohydrates that you have to consume through vegetables, of course. There are a couple of carbohydrates everywhere. When you're doing ketogenic, you'll have cut on everything and that have their consequences because you need the vitamins, you needed fibers.

This is why paleo is more of a diet that I believe in because it's mostly about natural food, consuming good free fatty acids. I'm vegetarian, so it is mostly nuts and avocado and olive oils and butter, which is great, and coconut oil, etc.

Those are good, free-fatty acids that supply me with great energy and very low-carb diets. And it is about metabolic flexibility. You know, it's so interesting to hear you speak this way because it really reinforces in me this idea that, you know if you're heavily on a car, if your on the glucose, pure them, cure the head of the Glucose Carrying Parade, that process of, and it re-educating your cells because your cell's know.

They just haven't had to, its almost like the factory's been shut down for however long. Right. And so now we need to restart the machinery and it can be ugly at the beginning.

But if we can get to that world where we kind of flip back and forth, it allows us to get the best of all worlds. So going back very quickly to the, and we're not going to stay on endosymbiotic for much longer, but can we talk a little bit about this symbiosis influence, how it it would influence modern immune signaling because the immune system, we were talking about this offline earlier on another subject.

Again, like we don't think of it as a mitochondrial issue, you know, as foundational as mitochondria is to the cell. immune proper immune balance and function is foundational to our ability to be healthy and age well and not have chronic disease.

So I don't know how many people know that, but the function of immune cells really depends a lot of mitochondrial function and actually the different cell types that we have in our blood and in systems also differ in their mitochondrial function, they differ under mitochondria content, number, quality, so it's very different.

So for example, if you want the cell to stay in a stem kind of senescent state or quiescent, the mitochondrion are actually not active. They only rely on glucose for metabolism.

Once the cell needs to differentiate to different cell types, then they start activating their mitochondria. And now different types of cells will carry different amount of mitochondrion.

Actually, they are controlled, their differentiation state are control by the function and the number of my mitochondrial within those cells. So it really is critical.

to have good and functional mitochondria in this stem cell that will eventually produce all the immune cells that are required. When there is an immune requirement in the body, either there's a pathogen that is inflating or whatever, then the mitochondrion needs to be very active and this function is being tested.

An antigen producing cell needs tons of mitochondria, okay? This is a fundamental requirement for the production of antibodies or the presentation of antigens all requires energy and a lot of energy.

So the function of mitocon are actually fundamental for immune system function and very, very important. And when we talk about immune function. It's not only about protecting the body from invading pathogens, it's a virus or bacteria, is actually protecting our tissues from damaging damaged components.

For example, while we are aging, there is an increase in the presence of senescent cells. Those are like dormant cells within the tissues. The fact that they are there, not functioning just just there and secreting their factors is a damaging response.

What should have happened is the immune system should've come to that organ and cleared them away. Because the new system is not functioning well, you get them accumulating within the tissue and inducing further damage.

So the function of the immune system is not only against foreign pathogens, it's also against damaged, dead senescent cells within our tissues. They have to be removed by theimmune system.

So there is a very strong connection, for example, between immune dysfunction and Alzheimer's disease, immune disfunction and metabolic diseases, pancreatic diseases kidney insufficiency.

There are many connections between these two organ systems, the and further organ dysfunction mediated by mitochondrial dysfunction. And you had mentioned earlier, before we started recording, you'd mentioned something about the immune cells started over producing It was overproducing mitochondria or do they start over replicating?

Immune function, signaling, and mitochondrial transfer

I can't remember. Yeah. So it really depends on the type of the cells that are produced because the immune system is composed for many, many different cell types.

And eventually the types of cells will be produced is dependent on mitochondrial content and function and special characterizations of that mitochondrion within that cell type.

When immune function is overactive, when you see the immune over… Autoimmune disease or… It might be due to impaired mitochondrial function in a certain cell type within the system.

It's important to look also on the specific cell types, yes. The headline that people know, if anybody knows about mitochondria, they know that they produce energy, right?

That's going in position. You, I think you and many other scientists talked about the micellular sentinels. They also sense the environment. they also make decisions.

Basically, can we talk a little bit about how they sense the environment? What are the other things that they're sensing for? There is a close relationship between what happens in the nuclear environment and the mitochondria.

The sensing that we talked about is mostly environmental sensing. For example, the nutrients or the oxygen that currently exists within the cell environment.

And then the Mitochondria sends signals to the nucleus. There are many proteins that are on the mitochondria in their day to day job. And then when there is a stress signal, they will shift that protein into the nucleus signaling that now the Nucleus needs to react.

What does it mean to React? The nucleus can now induce gene expression. of proteins that are required to stand in stress conditions to allow the cells to be more active in certain directions versus others, to eliminate reactive oxygen species or to increase the number of mitochondria.

That's what needed. I've just said, for example, when you're controlling the nutrients and now there is more free fatty acids, you need more mitochondrion.

Let's restart mitobiogenesis. If there's now a reduction in the needs of energy, let's start mitophagy and eliminate some of the mitochondria. So all of that is a crosstalk between the Mitochondria, the environment, and the nucleus.

It all goes back eventually to gene expression and protein production, et cetera. The mitochondrial behave like sensors. There is something very new recently in the past few years that speaks about mitochondrion also as censoring organelles between cells.

So now there's a phenomenon known as mitochondrial transfer. Cells can produce these tunneling nanotubes, like

tunnels between cells, and they can transfer mitochondria to neighboring cells.

Come on. That's incredible. I know. This transfer is actually induced by stress coming from the cell that is in demand. So one of the stress signals is that I don't have enough mitochondria, I have a dysfunction in the mitochondrion, and now the donor cells will create those nanotubes and transfer the microchondria to the demanding cells.

We see that in cancer between immune cells transferring mitochondria into cancer cells, the cancer is using this mechanism to actually extract mitochondrial from the immune system in order to work in a more efficient way.

This is maybe a mechanism of how in the future we will target cancer and try to cure That's one element. We see transfer of mitochondria in the brain through macrophages from the immune system, transferring mitochondia into microglia cells in brain and rescuing function.

There are many systems where mitochondrial transfer has been shown and it is a pure signaling between cells. It's beautiful. So that's incredible because that almost like in a healthy cell or in cell that not fully broken yet, it's an emergency that says, I don't have the energy to make my own new mitochondria.

Can somebody send me some over so I can survive this, right? And then if that doesn't work, then I guess at that point, that's when the cell would eventually become a senescent cell and become more damaging cell.

But how do you shut that down for, how did you set that pathway down, for cancer and not for the systems that need it, is then the question. I had a question here and I think we can now establish that at least from your work and from everything I'm hearing you say, we it's less of a chicken there to the egg thing.

It probably starts with mitochondrial dysfunction versus the mitochondria dysfunction as a downstream effect. Although my guess is once you get into that spiral, it doesn't matter anymore, but the way to fix it is going to go So can you talk a little bit also about how mitochondria communicate danger to the rest of the body?

You talked a bit about it at a cellular level. Is there another mechanism that is more macro or does it all happen through the nucleus, the genetic expression, Is it basically that process?

So there are actually also messaging of mitochondria between cells that are usually delivering stress response or damage responses. Those are called exosomes.

So cells usually secrete content from the cells. And they contain fragments of mitochondria and those exosomes are secreted out of the cell and they are actually serving as signals to the immune system, for example, or to to brain.

So there are multiple elements of cross talk between the internal inside cell mitochondrial to what happens in the environment. And it's not fully resolved and this is not my field of expertise, but it is known that exosomes carry damaged mitochondria as a signaling to the immune system to say this cell is currently damaged and needs to be cleared.

Wow. That's wild. I mean, guys, I hope you're geeking out on this. I'm fascinated. This is so interesting. Okay. Let's move into mitochondria and rare disease.

And then I think what I want to, and then we'll talk about mitochondrial aging. So let's go micro and we're going to go macro. A lot of your work started with rare diseases, right?

Do you want describe a few of the conditions that you kind of, are there a a conditions you really focused on initially? Yes. Yes, absolutely. Our therapeutic approach is actually to harvest stem cells from the blood and then enrich them with mitochondria.

We are doing this outside the body. So collecting from blood outside of the bad and enriching with healthy and

functional mitochondrion. Those are blood stem cell.

It means when they infuse back to the bladder, they will engraft in the bone marrow and start producing healthy cells of blood in immune system. So the first disease that we looked into was an ultra rare disease, that starts with a bone marrow failure.

So, the stem cells in the bone of these patients are failing, their mitochondria are fading. And that means that the lineages of blood cell formation are disrupted.

This is the disease we started because we realized we first need to show that, we can fix the blood system. What happened later on, I will tell you, but the disease that we chose was a very special disease, that first of all is not inherited from the mother.

It's a genetic disorder that is non-inherited from mother, and the patients are born with a huge deletion in the mitochondrial genome. And they are suffering, of course, from lack of energy and many other functions of the mitochondria, which means that the first symptom is that they're not growing like their age-matched children.

So they have full failure to thrive. They have a bone marrow failure. And then we said, if we'll harvest the stem cells from their bone mirror and we will augment them with mitochondrion coming from…

Their mother is actually an identical mitochondrial genetic source. But without the deletion. That was the concept initially. And it's not about the business model because there are only about a hundred patients in the world with this disease.

It is called Pearson syndrome. But it's about understanding. You're almost inducing endosymbiosis. Exactly. That's exactly what we are doing, replicating what happened back then.

But Pearson is also a multisystemic disease. When you see these children, they are all dying during childhood, but as the disease progresses, it started moving from the bone marrow into other organ systems.

So they start having kidney insufficiency, they started having diabetes, have muscle weakness, narrow degeneration, cardiac issues. And when you think about it, there's just like an old man in a young boy's body.

It's a replication of the aging process in the very clean genetic background of a mitochondrial dysfunction. So we actually realized that this is the best disease to study the impact of our therapy in a multi-organ failure, just like happens in aging.

So, we started treating these children and what happened was that we improved more than just the blood function, the hemoglobin or red blood cells production.

Mitochondrial therapy for rare disease and aging

We started seeing that these kids started gaining more energy. and the children who were at six or seven-year-old in a baby stroller or in the wheelchair started walking and gaining more energy, being awake more hours in day, the kidney insufficiency stopped progressing and it was mind-blowing.

So we can actually input with the same therapy that only enriches the blood stem cells with mitochondria, by the way, causing the immune function to be better.

Now we see an impact on distal organ systems, on the brain, the muscle, and the kidneys. It was a disease where we can really learn so much about the potential of the therapy moving forward.

Currently, we are in phase 2 for these Pearson patients. Because it is so rare, so probably only a few patients to get us through the approval process because there are just no more patients, but then Obviously when we started, everyone looked at us and said, you're going to transplant mitochondria in aging, right?

And then reverse aging. You're gonna do anti-aging. It's going take a while, but the targeting of a very specific mechanism, which is the mitochondrion, not by trying to improve one function, because mitochondria have thousands of hundreds of complex, but by replacing the entire organelle.

That was the approach that we took. And then we said, we have to be able to measure this. As I told you, there are no blood tests to measurement mitochondrial function.

So we developed them. And then we could validate the biomarkers in the patients with the mitochondrial deletions and then tested whether before and after treatment, you see an improvement in mitochondria function.

So that was very important for us. It was just this person with maternal-derived mitochondria, and this is really non-scalable. When you think about us moving into aging, we can't really have this mitochondrial from our mothers, right?

They are no longer a source, so we developed. a bank of healthy functional mitochondria that are actually coming from donor placentas. So women who give birth, within Minovia, everything is very female oriented.

The mitochondrion only from the mothers, the donation of mitochondra comes from placenta, from women that givebirth. We are female founders and more than 75% female in the company.

I love it. Yeah, very, female-oriented. But now we have a bank of healthy mitochondria that we can use across the board for anyone that will require this treatment.

But we didn't go into aging yet, because maybe the impact we see in children is because they are young and they're regenerating easily. like going to an age-related disease and demonstrating impact also in elderly people.

Again, we looked for a disease where the bone marrow is failing, the stem cells of the bones, and that was myelodysplastic syndrome, or MDS. And those are people that are 60 years old and older and have a severe anemia.

They are blood transfusion dependent and straightforward thing we'll do to improve their stem cell function and to see an improvement in an anemone. And that was indeed what we started seeing.

So this is currently in phase one and retreat patients, but those patients have other symptoms of aging, of course. For example, one of these patients had the kidney insufficiency with blood creatinine that is high and he was already scheduled to start dialysis and then three months after treatment, you start seeing a decrease in the creatinine levels in blood and currently he's on normal creatineine level.

So not only his blood improved and he is no longer blood transfusion dependent, also his kidney function improved. It makes so much sense, right? You're going at the stem cells in the bone marrow.

If there is a fountain of youth in a body, if there's a foundation of regeneration and repair, it's those stem-cells. And I think you said this earlier, they will differentiate into many different cells that are applicable to many systems.

So it is the elegance of this is getting it at such a foundational level that then the body just decides what the next priority is on the list. Exactly.

That's wild. I just want to go back to that childhood disease, just because it's my own curiosity. Would that disease not also be a really good candidate for some kind of gene therapy someday to fix that deletion in the DNA?

Many tried, of course, so the complexity of that. So we know how to fix genes in the nucleus. In the mitochondria, it is way more complex because the DNA resides within the two membranes of the myocardium.

You need to deliver components for fixing the dna to pass through these two membrane. And then you not only have one or two copies of DNA, you have multiple copies.

Yeah, yeah. To fix that is more of an issue. But you're just keeping them alive until somebody figures that problem. Maybe, yes. And even then, they will need more mitochondria we will supply them with.

There's another accelerated aging disease where it's not the same one. Progeria. Is there an application for progera with mitochondrion too, or is it a different system?

There is a specific gene that is mutated and causing the disease. I think a gene therapy in that case will probably be applicable. So within the context of this discussion here, is there something to be said about mitochondrial resilience in all this?

I think we have a way to control our mitochondria resilience. I wouldn't wait until we age and develop chronic diseases. We can definitely behave while we are young and maintain our function.

Getting that into our awareness, will actually start resonating very early in our lives, and it will impact everything we do. It's the clarity in your brain and how we think, it's sleep quality, our metabolism and our ability to absorb food, how do we behave when there is an infection, when our body needs to activate our immune system.

So we cannot predict when a whole pandemic or when our immune system will be challenged, we need to be prepared to that. And being prepared for that, meaning our mitochondria need be resilient to all these immune cells.

I think many of the surroundings, the air pollution and the water that we drink and food that eat, and medications that take, they have a very strong effect on the mitochondrion and not the positive one.

So being aware to that all the time and making sure that we consume only things that benefit our mitochondria is something that you need to take in mind if you want to age healthier.

Yeah, I love that. I have a question. This is a little bit out of left field. Do you have an opinion on non-native EMFs? On what, sorry? non-native EMF, electromagnetic frequency.

A lot of people talk about the impact of 5G on the cell. Basically, this new input that our bodies have not had to evolve. The earth has electromagnetic efficacy, like natural, of course.

And you may not have thought about this. Maybe this hasn't come across your desk yet, or you're so busy. So we can talk about that the next podcast, because there will be another one.

All right. Let's talk a little bit about aging. At one point in life, Do you think we start to feel or experience that decline? I've always heard at the age of 30 is when things start.

Up until 30, our ability to regenerate and repair is really good. And 30 is an arbitrary number. It could be 32, 31. Is it that 30-year-old where we start to really experience that decline?

And is that backed up by mitochondrial science, do you think? Or is it later, earlier? What are your thoughts? So we're currently measuring those mitochondria functions in different ages.

And I'm sure that in two years, I will have a precise answer for that. But actually, people say that starting 20, we started to age and decline. Okay, it's very depressing.

I know, I'm sorry. Oh my God. Yeah, but I think it is very different and very personalised between people and definitely different between males and females.

As we know we age very differently. So I think when we will do our analysis using our mitochondrial biomarker to determine the health of mitochondria in different ages, we'll take into consideration also the different sections and then try to build those mitochondrion scoring systems.

Yeah, health and disease, and then they'll have a better answer. Yeah. There's recently been some noise about these two inflection points, particularly in women where aging accelerates.

And I think one of them was at 40, which makes sense because that's where menopause, you know, give or take a couple of years. That's when it starts to kick in.

Again, I'm asking for opinion. I think this is an area that you're going to actively be studying, but how much do you think that this mitochondrial drop in function might be playing exactly into those?

Is the loss of hormones affecting mitochondria health? Absolutely. A hundred percent. And actually the decline, the mitochondria could be the clock that caused the declining hormone production, and that's where you see, so it's what starts, right?

And I believe that the Mitochondria, that are kind of the trigger for that, because I told you about the involvement of mitochondrion in steroid hormone protection.

The cholesterol, I will never forget that. At 60, it's already a multi-factorial event. It's when the mitochondrial dysfunction starts circling back on different pathways.

You will see telomere shortening, you will reactive oxygen species, So many dysfunction of the immune system, exhaustion of stem cells, all of that is happening already at 60. So it's already too late.

This is why I mean that you need to intervene very early to prevent dysfunctional mitochondria, which is one of their earliest things that So I was going to ask about stem cell exhaustion.

Talk to me about STEM cell. Is it a thing? What is it? And do you think it's coming from the mitochondria? I mean, listening to you now, I'm like, everything's going from.

Of course. Talk to us about stem cell exhaustion, because it's a term that I think gets thrown around a lot. And I don't know that people fully understand what it really is and what isn't.

Then let's tie it back to our mitochondria. Yeah. Eventually, you measure the outcome. For example, we know the ratio between lymphocytes and other cell types within our blood system is changing with age.

Why is that? You see just the outcome eventually, you see a very poor responding immune system, but it starts with your stem cells. This is how we know that.

So exhaustion of stem cell means cells are actually lacking the energy to start differentiating Okay? Because that's what you need as a trigger to start producing new blood cells.

New blood is meaning new immune cells and different subtypes of immune sense. So as I look at stem cell exhaustion is their ability to renew themselves.

A stem cells has to be able to, to renewal itself and all to stop producing you daughter cells that are differentiating. So the adoption affects two pieces, the renewability and the differentiation capability.

And the lack of energy is something that just puts cells into senescence. Just like you said, if I don't have energy, now what I will do is just hibernate, right?

I don't waste energy. I'm sleeping now. Don't bother me. And not bothering me, meaning I am not doing my job. i'm not replicating, I m not differentiating, i m producing the cells that I should produce.

Stems also, of course, have a role in regenerative capacity. So when they migrate into a tissue, they will need to renew that tissue. Every renewal process requires energy.

The production of proteins within a cell that is one of the most important piece of this cell needs to do either secreting factors or producing enzymes for the cell to function, it all requires the energy and mitochondrial function.

I think eventually this is what exhaustion is all about, renewability, differentiation and regeneration. Yeah, no, it's foundation. It's foundational.

Like the immunosenescence has got to be fundamentally, you've described it three times now. Yeah. And just to go back to the zombie slash senescent cell, I've heard them described as grumpy old men.

They don't even just sit there and hibernate. know, they secrete cytokines and inflammatory molecules. And so in the absence of a neighbor that will create one of those nanopores and donate mitochondria so that they can live to fight another day, then they are like, okay, fine then, I'm just going to take everybody down with me kind of attitude, which is You know, interesting.

Okay. You've referred to neurodegenerative diseases a couple of times. How foundational do you think mitochondria role, like, is it a foundational role?

Okay, because, you know we talk about accumulation of plaque, we talked about the death of neurons, which I'm sure you're going to sit there and explain to us in a very clear terms is everything to do with not enough mitochondrion.

But, but, in treating these diseases, should mitochondrial treatment fundamentally be one of the pillars? Because as you said a few times, in a lot of these chronic diseases, it's not just one thing, right?

It's never going to be just on thing. But should addressing mitochondria function foundationally be on of pillars of dealing with these things, whether it is slowing them down, reversing them or avoiding them in the first place?

What you need to do with brain cells is really prevent their death. What happens in neurodegeneration is either the cells stop functioning and they remain stuck in the tissue, like we said, or they are dying and again remain in stuck tissue and are inducing further damage.

When we talk about induces mitochondrial function, it's actually preventing the death of neurons. and prolonging their life and activity within the tissue, because neurons are not regenerating.

We are born with amounts that will keep us alive all our life. And the amount of energy that the brain demands is huge. It's enormous. Where does it get this energy?

Mostly from the mitochondria. It's more than 80% coming from mitochondrial function.

Biomarkers for measuring mitochondrial health

So, of course, neurodegeneration, especially in aged individuals, is due to mitochondrally dysfunction. We see that greatly. As we age more, more components join the party, and of, course it's accelerating everything.

but the earliest signs is really mitochondrial dysfunction. The problem is how do you solve mitochondria dysfunction in the brain? That's really problematic.

You have to only handle what's in there. you cannot deliver anything new. It's very, very difficult. Even drugs are difficult to inject, to to brought into the brain.

So it's a huge challenge. When we have treated our patients, they also suffer from neurodegeneration. I mentioned Pearson syndrome. There are also other more neurological diseases like Karen-Siren syndrome and Lee syndrome, all our primary genetic mitochondrial diseases.

And we observed an improvement in neurocognitive function. We don't really know how to explain it, I have to tell

you, because if you remember, we treat the stem cells in the blood, how does it influence the brain?

But we have seen it because it's like, again, the immune system and the signaling What you're doing is getting in at such a foundational level that allows the body to do what it does.

So you don't have to get to the brain. The body is like, we can get there. All you got to is give us what we need. It's so incredible. I'm going to use the word elegance again because It is such a, and I mean, nothing about this is simple, but it is truly so simple that if you can get to that first piece where the body is producing, it's what it needs, then it can do what needs to do.

Like that is just wild to me. But we are scientists. I mean, and we have this curiosity to understand how things are working and drug development. If you want to succeed, you need to.

Understand how they're working. This is what initially we only focused on the bone marrow and the blood system to show improvement. But eventually the impact that we see in the patients, that's our trigger to go way far and beyond.

just as rare diseases and the genetic disorders really try, we feel very brave now. First of all, our therapy is safe, so it's not, do know how, first of, and then the multi-organ improvement.

We had a patient with severe epilepsy and stroke-like episodes on a monthly basis. that for six years we prevented those after a single treatment. It was like unbelievable.

So we really try to understand how to deliver this therapy way broader. The brain is very interesting but very challenging. we will just have to face the clinical outcomes to see them.

And eventually that will be the proof. I don't think we'll ever understand everything about how it's exactly working. But I think that's the most important thing, because if you save the life of a child with a Pearson syndrome, but you didn't fix his brain, I mean, that So yeah, you didn't achieve the ultimate goal.

So we can say that metabolic disease like diabetes, non-alcoholic fatty liver disease, cardiovascular diseases, are going to, at the very minimum, have a mitochondrial component to them, if not be foundationally linked to mitochondria under this malfunction or under performance.

Are there, do you think, are we misclassifying diseases that are actually mitochondrion in origin? I mean, fundamentally, the question is, Do you there's a disease that isn't founded in mitochondial dysfunction?

You can try and search your engine. Any disease with mitochondria, you will find a connection. I tried this before. You could go ahead and do that. But definitely, I mean, whether it started a disease or it's an outcome, this is a question.

What I can tell you for certain, any disease will benefit from improved mitochondrial function. Okay. That's because fundamentally, if you get more energy, you can fix anything within the cell.

Talk about you spoke about diabetes insulin production is an energy energetic process. It has to be coming with improved mitochondrial function, and then you'll get insulin production, controlling of insulin, production controlling glucose metabolism.

All of that is energy dependent and mitochondrial dysfunction will actually induce further damage into the cells. If you improve that, you have a better chances to fix the, even the initial problem that the cell has.

Yeah. cell accumulates mutations, for example, and now you improve mitochondrial function, you have better chances to initiate a DNA damage response or to improve the function of those machineries that will fix the cell.

And if it's not, mitochondria will induce the apoptosis, the cells suicide to remove and eliminate the bad cells, which

is also fundamental. Yeah, I think there is even…

I tried it, even one disease that doesn't have mitochondrial function, eventually. And those diseases will probably benefit from mitochondria function.

Yes. Yeah. I would think so. Even anything in the eyes, as we see the decline in eyes. The eyes are so dense in mitochondrion. Before our call, we had a full discussion about different diseases that could benefit for mitochondrian transplantations.

You name it. All right. Let's move into mitochondrial biomarkers, measuring the invisible. So this is what your work really becomes also about. There's obviously the mitochondria transplantation, but as you said, in order to quantify what you're doing and to understand the needs and the outcomes, it's really about measuring something that nobody really has been able to directly measure so far.

And that's a big aspect of your work. So clearly, I mean, we don't measure them because we can't, or we haven't developed those strategies. What are the most promising biomarkers in mitochondrial biomARKers you're seeing coming out today?

First of all, you cannot treat, but you can not measure. Okay, this is fundamental knowing that drove us to develop those biomarkers. We wanted to developing therapy, But we realized there is no way to measure our success in this therapy development.

So let's develop biomarks. But then when we were exposed more and more into this world, of mitochondrial diseases, we realize it is way beyond just the primary genetic.

And there is a whole world of secondary mitochondria diseases and a world age-related mitochondrion diseases. You see people that look completely perfect.

They go to the doctor, they say, I feel very frail, very tired. I don't have the power at the end of the day to climb my stairs to go home. Your blood tests are perfect, nothing shows that you have any disease.

Exactly. So we are not measuring, obviously, what drives our mitochondrial dysfunction. The most important things. Mitochondrial disfunction is tricky, right?

Because I just told you, they have so many functions. Which ones are the ones we should pay attention to? So the number of mitochondria are critical. Even if you have some of the mitochondria dysfunctional, but you can increase the content, you could eventually increase content of good mitochondrial, get enough mitochondrion to get energy.

So the number of mitochondries are really critical. The function that we are looking at is, of course, the ATP production. But ATP could come from glycolysis too, right?

So we're coupling the production to the respiratory chain activity of because eventually you need to know what is the specific ATP produced by the mitochondria per single mitochondrion within the cell.

So we're doing all three of them together. And those are the three biomarkers that we are using to calculate the mitocondrial scoring of a healthy individual versus a child or an adult with a primary mitochondrial disease.

Now, every person that will come and be tested with these biomarksers, we can say, your score is somewhere here, Your health healthy age-match control is here so you're doing great or your age match control.

Is here. You're here primary genetic medical diseases are here you can go further down or you get the therapy and move up. the scale into your edge mitochondrion.

You can also go up the tail into a younger phenotype, which would be fantastic. But that allows us to really measure the quality, content, and function of the mitochondria in different people.

We are also looking, of course, at the Mitochondrial DNA because we want to know the mutation load in the mitocondyl genome, Which is fundamental to all this non-functioning mitochondrial.

And there is also another biomarker that is released from distal organs. that is called GDF-15. GdF15 and mitochondria, this function goes hand in hand.

So overall, five biomarkers are currently used in our clinical studies to first score different people, different ages with their mitochondrial scoring, and then test how our therapy improved the function or the levels of those biomARKers in the blood of these patients.

Wow. So it's like a biological age test for your mitochondria. Exactly. It's a biologic aging test your biological age, period. Exactly. This project, I have to say, was funded by the foundation called Countdown for a Cure.

I've just returned from Atlanta from an event, the annual event of the Count Down for Cures, where the people who drive this foundation are people will look completely normal.

When you speak to them, they're telling you all the symptoms that they are suffering. As to a secondary mitochondrial disease, young, beautiful, healthy people, they look very healthy.

Eventually, when when you test them, all the blood tests are fine. There is nothing, but they cannot climb the stairs in their house. They can come to come through a situation where they can't even feed themselves and they need help.

So it's unbelievable. It's mind blowing how needed those biomarkers are.

Safety, combination therapies, and practical support

And this foundation eventually identified our project as one of the most funding and they gave us the grant to develop those bio markers way far and beyond than what we initially intended to do.

I told you in two years we will meet and I will tell you exactly what should be the score of mitoconate at each one the ages and then we can start screening for it.

Wow. Wow, wow, okay. So we talked a bit about the transplantation that you're essentially in a very very simplistic way, trying to induce endosymbiosis outside.

You're using donor mitochondria from cord blood. So essentially, unless we get into a world where we all bank our mom's mitochondrial when we're born so that we can use, you know what I mean?

Like later on in life, I can see on a prenatal level, at some level that mitochondrion support for the mother would be wildly important. I mean, it's wildly through your entire life.

Is there anything in the transplantation trials originally that surprised you? I think the brain, the impacts on the and the kidneys as well, because usually they say that once kidneys fail, you cannot reverse kidney insufficiency because of the fibrosis that happens.

So I think that was really, really surprising. But overall, knowing how important mitochondria are, I wasn't that surprised if I really planned this amazing mitochondrion.

And I also knew that the placenta are very unique in their nature because they just fed a baby for nine months and now we are using it we're like behaving like it's trash and all of a sudden taking that very very healthy young organ producing the mitochondria it was mind-blowing it is way more active than blood mitochondria, for example.

So if you compare mitochondrial from placenta to blood, mitochondrion, you will see that they're 10 times more effective than the blood. Mitochondria and they contain more mitochondal DNA than black mitochondra.

Overall, it's like a super organ for mitochondry. And that I knew from my PhD already, that's very unique. And we found something to do with this trash.

So very happy about it. Are there any safety concerns that have to be solved? Do you think like it could? I mean, I guess in this case of an existing cancer, you've already said that the cancer is already hijacking sometimes.

healthy cells, mitochondrial stores. Are there any other safety concerns that have come up for you that you've seen so far that, you think, well, we might really need to?

Definitely. I mean, there are always safety concern around a new therapy and intervention with some evolutionary conserved processes that was always frightening.

This is why we started, by the way, first from the maternal mitochondria into the children, because we didn't want to go first into an allogeneic donor.

But today, the main safety concern were, can we really mix different mitochondrial in individuals? Can I take or borrow mitochondrion from one donor and use that?

Will I have any immune responses? We get rejection. And eventually, we didn't see any immune response. Again, the mitochondria, there are no anti-mitochondrial antibodies that are produced due to this process.

So overall, in 27 patients so far, it seems to be safe and tolerated. Those risky aspects have been overruled. The cancer, I think, is always an issue.

Will we induce cancer because of this mitochondrial augmentation? But actually, We went to one of the world's experts. We focused, if you recall, we are focused on one age-related disease called myelodysplastic syndrome.

And MDS had the risk to develop into leukemia, to AML. So we went one the of world expert before we started those inpatients with MBS. He has a mouse model.

This is a lab in Memorial Sloan Kettering. in New York, led by Dr. Omar Abdelwahab. So he has a mouse model that is accelerated ager, has MDS, and develops AML.

And all the mice die from AMl. When we took the stem cells of that mouse and we transplanted them in a healthy mouse, all of the mouse died within four weeks.

If we augmented those stem cell with mitochondria, that was supposed to be only for safety. The question was whether we induced faster the leukemia. Do they die in a week?

The answer was they actually have an extended lifespan and it makes a little chemical progression. So that was comforting. I said, okay, we are safe to go into humans now, because not only we're not accelerating leukemic conversion, but we actually protecting them and we delay the leukemia.

Well, based on the conversation we've had, you're re-engaging the immune system to do what it does, which will, I mean, it sounds to me, again, oversimplification.

through Z over here. But based on this entire conversation, that makes complete sense. Okay, we're down to the last couple of questions. Stay with me.

I could keep you here all day. Because I would imagine that down the road when this has been refined and tested and this is going to become at the least as common as stem cell therapy, but I imagine this would augment stem-cell therapy.

This would underpin, like did you know, we're seeing so many amazing things coming out of stem cells, exosomes. But now if we bring the work crew, the stem cell with the exo zones, and the instructions, but now we are also

providing extra energy to the cell to actually do the works.

You can imagine that this would explode in terms of efficacy or am I missing something? I love it. Yes, exactly. We all think about how to combine therapeutic approaches in order to reach the maximum capabilities of a human body.

And this is exactly it, so if you harvest now stem cells from an elderly individual and you infuse them back, they're actually old. The mitochondria are old, it won't be enough.

Taking those stem cell and now augmenting them with young and healthy mitochondrion will boost their activity. That's exactly what happens also when you grow cells in culture in order to produce exosomes.

Growing cells endlessly in the culture causes the cells to age. Their mitochondria are aging. Now if you want to create healthy functional exo-zones, boost the cell with mitochondrion to get more exozones and healthier exotones.

It's everything. People are trying to reprogram cells. to come back to their young nature, like iPSCs, pluripotent stem cells, to go back and be embryonic-like, right?

Those are the newest things with gene therapy, trying to over-express the Yamanaka factors to make cells young and healthy again. But how do you fix their mitochondria?

You cannot do that with the monaca factor. So let's augment them with mitochondrion. And then you see how this combination therapies will solve multiple problems at the same time.

It's not one plus one, it's exponential because of the impact of mitochondrial. Do you have any thoughts on NAD precursors, supplements, in terms of their importance, something that people can do to support mitochondrial function at a foundational level?

And is there any other supplement that stands out to you? Yeah, so NAD, of course, is one of the important substrates that sells mitochondrial function.

Super important, yes, and actually available and consumable by the cell. So it's something that relatively was well studied and it seems like to produce a good results.

In addition to NAAD, you can see coins and Q10, very important ubiquinol and not ubiquinole. and a structure that can be consumed by the cells and be actually available for the mitochondria.

Coenzyme Q10 is one of the most important co-factors for mitochondrial energy production and important to know it is blocked when we are taking statins.

So anyone who's taking to statin to reduce the cholesterol is actually blocking the synthesis of Coenzium Q 10 within our cells. And this is why supplementing with Coensin in that case is a good thing.

And one of the things that I spoke about, you know, raising your free fatty acids, that induces your mitochondrial activity and mitobiogenesis. This is important, easily controlled by the food that we eat.

sing, consume oxygen, do yoga, because it's so good, yes, for mitochondrial activation. And one thing I tried on cells, on culture and is working, a compound called resveratrol.

It is found in grapes, red grapes only, and actually works. So again, the source is important, but the Zveretrol is something that induces mitochondrial biogenesis, so the increase of mitochondria content within the cells.

I know, I tried, it's working. Are there any lifestyle or other interventions that actually you believe really do move the needle on mitochondrial biogenesis?

I think free fatty acids is gonna be one of them just because of the energy. Is there anything else? So I'm gonna throw a couple of things at you and you can give an opinion if you have it.

There's red and near infrared light, infrared saunas, hyperbaric therapy, fasting, Hyperbaric chambers, yes, induce mitochondrial function, by the way, increase also stem cell content within the blood.

Great science behind it, totally agree. I don't know about red light therapy, I'm not sure I haven't seen anything, but I think I am all about the science eventually.

And the last one, what did you mention? or fasting, fasting. So, and I would also figure out your ketosis, right? So. Absolutely. Yeah. Ketosis mitochondrial function, yes.

Exercise? Again? Exercise. Absolutely. Okay. When you build muscle, what you're actually doing is increasing the mass of mitochondria within your fibers, muscle fibers.

That's all what it is. This is how you, this is where you need protein in order to expand mitochondrial within the muscle fibres. And what do you see is more energy, the more mitochondrion in the muscles, more Energy and then more tolerance and exercise tolerance.

Excellent. Also, when you exercise, of course, you consume more oxygen. You feel better energized. It's all over. Everything. it's great. Love it. Okay, last two.

What is one mitochondrial myth you wish would just disappear? Oh, that's a good, well, you know, people think that mitochondria are single. Actually, mitochondrion are plural.

So it's mitochondriion for one. Mitochondria, are plenty. And what people, every time they are surprised, oh, we don't only have one mitochondrium within ourselves?

No. We have tons of them. This audience, they know. Yeah, it's okay. Last one. And this is something you mentioned at the beginning of the podcast that I think is so important.

You talked about emotion. you talked About joy, you talk about the impact of our, of something that is really not tangible, but that impact on mitochondria.

Do you want to speak to that a little bit? Because I I want the audience to walk away with something that they can start tomorrow, right? Or today. And just, I'm a scientist who's been so deep in this for so long.

I'd love for you just to share with the audiences your thoughts on our emotional, spiritual inputs that can they affect our mitochondria. I think when we all feel good, I mean, we feel energized, you feel so powerful.

Lifestyle, supplements, and emotional health

And I go to visit my parents and my father is now 84 years old and unfortunately suffers from a third age epilepsy and consumes drugs that really harms his mind.

When I come to him, what I tell him That is seeing, seeing when you're seeing it, it's like you float your body with all your energy and you feel so good.

And now my mind thinks, why is that? And how come one of the most surprising things I've ever met in a scientific conference was a group coming from, from Canada, from Toronto.

They are from a psychiatric hospital. And they presented data that I was just shocked and overwhelmed. Schizophrenia, mania depressia. All these bad diseases of the soul are eventually bound to mitochondrial dysfunction.

Iwas shocked. I never knew that even. So our mental health really depends on the quality and health of our mitochondria. That was shocking to me. And eventually, exactly, everything is eventually bound together.

Why should we live those long lives unless we feel good, we fell healthy, feel energized, and with good health, mental health as well? So they presented data showing that 30% of bipolar disease patients carrying mutations in the mitochondrial genome.

It's like it's crazy. Okay. So I think there's so much to it of how we, we recapture our life, our health and how much of it is in our control in mitochondria.

This is the case. We can really control it by simple. Yeah. And that, that was, I was getting at like in a person who is not chronically ill or has a serious condition, is it possible that bringing attention to living in joy and gratitude and in a positive mind can also have a very powerful impact on how we age and how our mitochondria behave or thrive.

Is there anything people can do when they're in the position that they are ill and they have to take medications that are in a negative to the mitochondria but saving the system.

So you know what I mean? Like sometimes we're in the situation where we need a medication to get us through a crisis. Have you seen or are there things that people can do when they're taking medications that are fundamentally toxic to the mitochondria, like a statin, for example, you mentioned CoQ10.

Are there any other interventions you can share with the audience that they might want to consider and talk to their physicians about that could be helpful to supporting the Mitochondria which ultimately could support their recovery in some small So, you know, for our mitochondrial disease patients, there is a list of drugs that are forbidden for mitochondria disease, patients because they are harmful for the mitochondrion, they can further deteriorate.

Of course, in some conditions, it is not possible. but there are drugs that are more toxic to mitochondria than others. For example, I mentioned epilepsy that my father suffers when he started on valproic acid, which is the first line of drugs epilepsy gives.

He started deteriorating like crazy. And then when I was reading about it, I actually, it is known that those drugs have mitochondrial toxicity. So if certain physiological condition, there are multiple approved drugs.

I would look into the literature and see which one of them has less mitochondria toxicity, Of course, we can supplement, just like people who are taking antibiotics are also taking probiotics in order to prevent the death of our gut microbiome, We can also supplement our mitochondria as mentioned with the supplements that we've discussed minutes ago.

in order to improve the mitochondrial function and to rescue that. There are certain antibiotics that are also harmful to the Mitochondria. I would try to avoid those.

Again, it is available. It's a data that is throughout the networks. You can easily know which antibiotics have more mitochondria toxicity versus others.

So just being in attention into our mitochondrial health will eventually allow us to ask the right questions either our physician or sometimes it's just information that is available through the network and you can really find out so much more about your health.

My goal is that any drug that is being developed in the first stage of development, the drug will be tested for mitochondrial toxicity. I was shocked to know that about 50% of the drugs that we use today have known mitochondria toxicities.

This is crazy. You can prevent that. It's so self-defeating. You're keeping a person alive and denying their body the ability to do any kind of repair work.

It is wild. All right, listen, we are, you know, this is like when you're the longest goodbye. I have to let you go so that you can go answer all of these incredible questions and bring your incredible work to market, which I think is going to be so unbelievable for the whole, everybody, so Natalie, please share with us any information that, anything that where people can learn more, where they can follow your work If there's anything anybody can do to help, I think just please share with us anything that people or just even follow the work.

Yeah, of course, we have our website, MinoviaTX.com. We have LinkedIn and Instagram and we are also on Twitter. So I think, you know, just follow our activity.

we're trying to be as helpful as possible for the entire community and to implement the importance of mitochondrial health. As you've just heard, it's not just about getting a therapy or a drug approved.

It's being mindful to our mitochondria very early on in our life as soon as possible and just bring it forward because eventually what we learn and experience on our bodies could be beneficial to others.

So share that information, follow what We do. And give us feedback and send questions. There is an info at minovia.tx.com. Feel free to write to us and ask any questions we find the time to answer everyone.

So do that. That's amazing. Thank you so much. This has been I can really, you know, I'm always interested in my topics, but this one got me. So we will reconvene, Natalie, maybe in a year or two when you've had your next couple of breakthroughs.

And I think people will be waiting for that episode. I know I am.