Episode #113: Mitochondria with Dr. Bryan Rade, ND

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Why You Should Listen

In this episode, you will learn about the importance of mitochondria in recovering from a complex, chronic illness.

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About My Guest

My guest for this episode is Dr. Bryan Rade. Bryan Rade, ND is a graduate of the Canadian College of Naturopathic Medicine and is the current President of the Nova Scotia Association of Naturopathic Doctors. His practice focuses on complex chronic illness, persistent Borreliosis and coinfections, mold illness, integrative oncology, the biomedical treatment of autism (“DAN” Doctor), PANS/PANDAS, neurological disorders, SIBO, and chronic pain. He utilizes a wide range of tools including prolotherapy, prolozone, neural therapy, perineural injection therapy, platelet-rich plasma, ozonated clot matrix therapy, Low Dose Immunotherapy, Low Dose Allergen Therapy, ozone therapy, CranioBiotic Technique, hyperbaric oxygen therapy, intravenous laser therapy, chelation therapy, and many others. Dr. Rade owns and operates the East Coast Naturopathic Clinic in Bedford, Nova Scotia, where he is joined by a team of innovative naturopathic doctors, as well as naturopathic residents. In addition to his clinical practice, Dr. Rade regularly speaks at medical conferences. He lectures at the Canadian College of Naturopathic Medicine on persistent borreliosis and coinfections, as well as on injection therapies for pain management.

Key Takeaways

- How many ATP are in a given cell in various tissue types in the body?
- What are key symptoms of mitochondrial dysfunction?
- What is the connection between mitochondria and adrenal or thyroid dysfunction?
- Is there a connection between our nervous system, sympathetic dominance, and mitochondria?
- Do microbes and toxins impact mitochondrial function?
- How does ATP as the danger signal in the Cell Danger Response model fit in with an attempt to support the mitochondria?
- Does mitochondrial dysfunction play a role in dysautonomia, POTS, or EDS?
- Do antibiotics or other medications such as Disulfiram or Cholestyramine negatively impact mitochondria?
- Can mitochondrial support improve those with damage from fluoroquinolones?
- What options are available for testing mitochondrial function?
- How is mitochondrial dysfunction treated?
- How can mitochondrial biogenesis be stimulated?
- What is the role of NAD in supporting the mitochondria?
- Does reduction of blue light at night benefit the mitochondria?
- What non-supplement tools may help support our energy production?
- How can autophagy and mitophagy be upregulated?

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Transcript

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[00:00:01] Welcome to BetterHealthGuy Blogcasts, empowering your better health. And now, here's Scott, your Better Health Guy.

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[00:00:35] Scott: Hello everyone and welcome to Episode Number 113 of the BetterHealthGuy Blogcasts series. Today's guest is Dr. Bryan Rade and the topic of the show is mitochondria. Dr. Bryan Rade is a graduate of the Canadian College of Naturopathic Medicine and is the current President of the Nova Scotia Association of Naturopathic Doctors. His practice focuses on complex chronic illness, persistent Borreliosis and coinfections, mold illness, integrative oncology, the biomedical treatment of autism PANS and PANDAS, neurological disorders, SIBO, and chronic pain. He utilizes a wide range of tools including prolotherapy, prolozone, neural therapy, perineural injection therapy, platelet-rich plasma, ozonated clot matrix therapy, Low Dose Immunotherapy, Low Dose Allergen Therapy, ozone, CranioBiotic Technique, hyperbaric oxygen therapy, intravenous laser therapy, chelation therapy, and many others. Dr. Rade owns and operates the East Coast Naturopathic Clinic in Bedford, Nova Scotia, where he is joined by a team of innovative naturopathic doctors, as well as naturopathic residents. In addition to his clinical practice, Dr. Rade regularly speaks at medical conferences. He lectures at the Canadian College of Naturopathic Medicine on persistent Borreliosis and coinfections, as well as on injection therapies for pain management. And now my interview with Dr. Bryan Rade.

[00:02:12] Scott: I used to think that mitochondrial support was something that came later in a treatment protocol. But over the past couple of years, I'm starting to understand how important it is as a foundational item in order to provide the body with the energy it needs to participate in the healing process. I'm honored today to have Dr. Bryan Rade on the show to talk about mitochondria. Thanks for being here, Dr. Rade.

[00:02:32] Dr. Bryan: It's my pleasure, Scott.

[00:02:35] Scott: Tell us a little bit about how you got into treating complex chronic illnesses. Did you have some personal health journey or experience that led you to doing the work you do today?

[00:02:45] Dr. Bryan: Well, personal health issues were definitely what launched me into naturopathic medicine. When I was a teenager, I developed these severe debilitating digestive issues. Went to my family doctor basically said it's IBS you're going to have to learn to live with it. And thankfully for me, I have a wonderful mother who's wonderful for many reasons, but she's incredibly open-minded and was a very early adopter of complementary and alternative medicine. She's also a bookaholic. So, we have these troves and troves of complementary alternative medicine books, things like on homeopathy and ayurvedic medicine and old school Bernard Jensen like crazy detox protocol books and things like that. So, just out of desperation, I started looking through some of those trying to find answers for myself, and thankfully, you know, found out like well, gluten is kind of bad for me and dairy is kind of bad for me, etc. And so, started piecing some things together, but it really just fostered a love and appreciation of complementary and alternative medicine. And so when I learned about the existence of the naturopathic profession, it was just the perfect fit for me.

In terms of getting into complex chronic illness, it just really all started with one patient about nine years ago in my practice, she had been diagnosed with Lyme down in the States. She was on Doxy for two weeks, and she wasn't anywhere near all the way better. And at the time, there was just no one else in my province or surrounding area that was treating Lyme or coinfections. So, she couldn't afford to go to the states or go out of province. So, she was stuck with me. And so I just hit the books and basically, very quickly found Dr. Klinghardt's work, Stephen Buhner's work, ILADS, Richie Shoemaker and then just realized like, "Oh my gosh, there's this whole world of complex chronic illness out there." And so just yeah, kind of came into it that way. And my practice has really snowballed in that direction.

[00:04:33] Scott: Beautiful. Yeah. And having had the opportunity to get to know you at some of the conferences, I know you have a deep passion for helping your patients and so that clearly comes across the work that you do. Give us the Mitochondrial 101 in terms of what mitochondria do for us, how many are in each cell? Does the number in each cell vary based on the type of tissue? And then how is ATP produced, how much ATP is produced, kind of give us an overview.

[00:04:58] Dr. Bryan: So, with mitochondria, they are very, very complicated. And as I've learned more and more about them, and as I've had the chance to dig into the research, it turns out they probably do a lot more for us than what any of us ever learned in biology class, once upon a time. But really the main thing that mitochondria do for all intents and purposes is they make ATP, so they make cellular energy, adenosine triphosphate, and we need ATP to run every single system in our bodies. So, a question mark would be like, well, in what kind of scenarios or what kind of systems are mitochondria so important? It's like, well, every single one because every cell needs ATP to function, whether it's a neuron, a hepatocyte, you know, a myocyte, whatever it happens to be. So, with mitochondria, the main job that they have is to make ATP or cellular energy. They're also very intricately involved in the antioxidant systems in the cells. They're involved in telling the cell when it's time to die. So, that relates to apoptosis. So, not surprisingly, there's a really big role that mitochondrial function or more importantly, dysfunction plays in cancer cell propagation, which is a whole other topic for, you know, another day or another podcast. But the long and short of it is they're very, very important.

So, with mitochondria, you know, knowing things like how much ATP is produced in the body and how many mitochondria are there per cell, etc., those are things that I am really interested in. What's interesting and slightly disappointing is that those definitive answers aren't readily available. Looking on a scientific literature. there's some data but it's not as detailed as I'd like it to be. And that might be because there's approximately-- Well, I'm sure some research dollars but close to zero research dollars going into enumerating mitochondria, unfortunately. But what we do know for example, is that in the substantia nigra, which is this region in the brain that produces a lot of dopamine. I'm sure many of your listeners will know that when that area becomes compromised or damaged then we get Parkinson's Disease. What I think is amazing is that every single neuron in that substantia nigra region has 2 million mitochondria per cell, which is a staggering number. What's further very interesting is that the neurons in the surrounding tissue actually have much lower levels of mitochondria, suggesting that their energy-producing needs are probably quite a bit lower, and those cells are completely spared in Parkinson's.

So, just kind of really drawing the correlation between neurodegenerative disorders and mitochondrial dysfunction. But you could take that a step-down, let's say and say well even say brain fog, which many-- I know many of your listeners are suffering from, or have loved ones or people they know that are suffering from that, or patients that are suffering from that. It's something that's very likely related to that too. So, neurons in the substantia nigra, at least 2 million per cell. Cardiomyocytes, so, the muscles in our heart, about 30% of the cell volume is mitochondria. So, kind of that old school concept of mitochondria that I know I had in high school and probably in undergrad and probably in first-year naturopathic school is that oh, there's only one mitochondrion per cell. It's like no it's a lot more, especially in certain parts of the brain, etc. In terms of how much ATP is produced in the body, that's another question that I was hoping the answer will be forthcoming in the literature. And it's really not, there are some interesting extrapolation models, but the estimate seems to be somewhere between for the average person, say, like a 60-kilogram person, let's say, would produce anywhere between 24 to 60 kilograms of ATP per day, which is, I think, insane. That's a staggering amount of substance being produced in the body. So, it just really speaks to the importance of ATP and how our bodies need to be making this stuff all the time to keep us running and functioning. So, when there's compromise there, go figure that certain body systems might start suffering.

[00:09:00] Scott: So, we think of ATP then essentially as fuel for the car. And so from your clinical experience, how important is a focus on producing ATP on supporting the mitochondria in terms of being able to move your patients towards health?

[00:09:16] Dr. Bryan: In my experience, it's been really, really important with the patient suffering from complex chronic illness, there's such a spectrum of patient cases, you know. There's the patients where you just get them on the right antimicrobials or the right detox protocol, or start killing off the mold or detoxifying the mold. And you know, they just hit the ground running and their physiology just rallies and they're doing great. And then there's the patients where you do all of those things, you address everything, you seem to leave no stone unturned, and certain symptoms just get better at a snail's pace or they seem to be perpetually plateaued. And so supporting the mitochondria to help make more fuel for the cell to make more ATP, in some cases, it's pivotally important. I'm really an empiricist at heart like I love being able to see like, which-- what a given treatment or a couple of new treatments introduced at one time actually do for a patient. I really want to see like does X treatment produce, you know, Y results or whatever it is. And so over the course of time, I've had the opportunity to work with many patients who are maybe plateaued or otherwise not getting better at the rate that we're kind of hoping they might, or conversely, it's like they're getting better, but it's like, well, like, you know, maybe let's throw something else into the mix here to see if we can get you better faster. And what I found is that, more often than not, you know, probably, if I had to throw a number on it, maybe 70% of the time, if the case seems to be moving a bit slower than we'd like it to, then mitochondrial support will oftentimes be helpful, assuming that it's in a well-selected patients, which we'll talk about how to select those patients coming up, I'm sure.

[00:10:52] Scott: We hear in the news about cases of mitochondrial disease, what is the difference between mitochondrial dysfunction and mitochondrial disease?

[00:11:01] Dr. Bryan: So, mitochondrial disease is genetic. It's relatively rare. My understanding is it's about one in 5,000 people would have a mitochondrial disease. There are a few hundred different mitochondrial diseases out there and some of them are incredibly debilitating. A number of them, people will have congenital manifestations of that. So, they might be born with seizure disorders or a shorter stature or different types of developmental differences. And then other mitochondrial dysfunctions don't become-- or diseases rather don't become apparent until later on in life. So, for example, some people have a mitochondrial disease, so a genetic deficiency in their ability to make optimal amounts of or functional ATP synthase, which is involved in energy production pathways in the mitochondria. And so going through their childhood and maybe young adulthood, if they don't have too much stress and they're well-nourished and this, that and the other thing, they might feel totally fine.

But then maybe getting into 30s or 40s or beyond, as other physiological insults start to accumulate as happens in life sometimes, then they might start getting tired for otherwise unexplained reasons, and it might be not manifest until later on. So, there could be some patients where we might say, "Oh, it sounds like you have mitochondrial dysfunction." I.e. a sub-optimal functioning of the mitochondria due to the presence of toxins, insufficient nutrients, etc., different things that we'll talk about coming up. But they might actually have mitochondrial disease, if they have kind of like a-- not super, super impactful, not really super obvious kind of mitochondrial disease.

[00:12:41] Scott: You mentioned brain fog is one of the symptoms that might lead us to think about mitochondrial support. What are some of the key symptoms that you hear from your patients that kind of make you go "Oh, I need to think about mitochondria here."

[00:12:53] Dr. Bryan: So, in my mind, I have it kind of broken down into two different sections. So, there's kind of the direct symptoms of mitochondrial dysfunction, and then what I kind of classify as indirect mitochondrial dysfunction symptoms. So, the direct ones would be ones that are directly related to a lack of adequate ATP being produced. So, in other words, an inadequate amount of cellular energy being produced. So, fatigue would be a hallmark symptom, reduced stamina or endurance. So, those folks who, you know, like I can do things around the house and I'm okay but I go run an errand and I pay for it for the next you know several hours or days. Or you know, I can like lay in bed but if I do anything more than that then I'm just completely kaput. So, reduced stamina would be a symptom also muscle symptoms. So, if people feel a heaviness in their muscles and muscle weakness, or I had a number of patients where they report saying like you know, every time I go to the massage therapist or my partner you know, goes to give me a massage they say like you're just-- your muscles are made outta granite or marble. And it's like, well, once we start supporting their mitochondria, we'll oftentimes see that getting better or going away.

And what's interesting there is that, well it's not intuitive, but what is factual is that it takes more energy, more ATP for a muscle to relax than it does to contract. And the glaring example of that not a very pleasant example to think of is the example of rigor mortis. So, a person dies and then of course, they're not making energy anymore. So, as their energy levels, their ATP levels go down, then their tissues contract and then it isn't until things eventually start to kind of, you know, degenerate that then everything goes soft again. That's a symptom that I've seen in a number of cases, brain fog, as you mentioned, also people feeling really cold. So, oftentimes we'll think about coldness or the chilly extremities, you know, maybe as a circulatory issue as it can be, but also thinking about, say the thyroid gland. At the end of the day, our body temperature is-- the potential body temperature in our bodies is really regulated or dictated by the number of chemical reactions happening in the body. Our metabolism, the definition of metabolism to my understanding is it's the sum total of all the chemical reactions happening in a person's body. And many of those chemical reactions require ATP to work.

Every time the chemical reaction happens, there's always a little bit of heat lost as a byproduct of that, and that's ultimately going to make our tissues toasty inside. So, when a patient comes in saying, you know, my hands and feet are cold, or I'm always chilly, or I get chilled, and I just can't shake the chill, "I'm thinking what's going on with your mitochondria?" And lo and behold, I've had patients who have been chilly people for years, like blanket aficionados, even like the first day of September, which depending on where you live, like you're in Nova Scotia, it's still pretty warm the first day of September. And they are like, "Wow, I'm finally warm again," like a normal person once their mitochondria are on track. So, those would be the direct symptoms. And then just briefly, the indirect symptoms would be things like if a person has osteopenia or osteoporosis because there's some interesting connections between mitochondrial dysfunction and the development of bone loss. Sarcopenia which should be a loss of muscle tissue, also insomnia because melatonin is so very, very important for mitochondrial function. And I've had patients where it isn't until we get their mitochondria working better that they can actually finally sleep properly. And there's a couple of other miscellaneous ones, but those are some of the ones I think about.

[00:16:20] Scott: One of the things that I hear a lot, particularly in those with chronic fatigue syndrome, CFS/ME is, you know, they're doing better but they do just the smallest amount of exercise and then they have a crash or this post-exertional malaise. And so I'm wondering, is that something that you see can be improved or resolved with a focus on mitochondrial support?

[00:16:40] Dr. Bryan: Yes, yeah. It's a huge tell for me that we're dealing with some mitochondrial dysfunction.

[00:16:45] Scott: Beautiful. Talk to us about what happens when mitochondria are not functioning optimally. Do they get removed similar to a apoptosis with unhealthy cells? What's the life cycle of mitochondria, and can they be removed independently of the cells in which they reside, or is it that the entire cell has to be removed?

[00:17:06] Dr. Bryan: So, mitochondria experience something called mitophagy, which would be analogous to autophagy. And where ultimately it's kind of this, like housekeeping that's done. So, if a mitochondria or mitochondrion is too dysfunctional, then the cell will say, “Oh, it's time to just basically envelop that in a little membrane, and then just basically digest everything inside and recycle the mitochondrial body parts essentially. So, if enough mitochondria are compromised, then I mean, I'm sure eventually there'll be a critical point where the cell couldn't function and the whole cell will apoptose. But mitochondria can be broken down independently, the whole cell doesn't have to die thankfully. In terms of the life cycle of mitochondria, just like the number of mitochondria per cell, and the amount of ATP produced in the human body per day, not a lot of really definitive information about that. In rat studies, they identified that most mitochondria and most tissue types seem to have a turnaround time of about two to four weeks. But they also speculate it's probably much faster in something like a hepatocyte, which would be dealing with so many toxins and whatnot. So, they have a pretty short lifespan in the grand scheme of things, which is, you know, probably ultimately a good thing because when we start intervening with things to make mitochondria healthier then, you know, hopefully, we can just have stronger and healthier mitochondria coming back pretty quickly.

[00:18:37] Scott: When the body produces new cells do those new cells have a higher number of healthy mitochondria, or are the new cells similar in mitochondrial number and function to the older unhealthy cells that they're replacing?

[00:18:51] Dr. Bryan: So, I think highlighting the importance of mitochondria for cellular physiology is the fact that a cell can't replicate or won't replicate with too many dysfunctional mitochondria. So, what happens in cell division, lots of things happen but from a mitochondrial perspective, what happens is mitochondria will join together in a process called mitochondrial fusion. And then those mitochondria once the cells have split, then they'll start breaking into smaller mitochondria called mitochondrial fission, and then the new cell goes off and does its thing. So, when that fusion has to happen pre-cell division, the mitochondria-- healthy mitochondria won't fuse with an unhealthy mitochondria. So, if a mitochondrion is too unhealthy to warrant fusion with a healthy one, then that mitophagy will kick in and the cell will just break it down. So, when our cells do divide to my understanding, they have to have a-- they have to have healthy mitochondria being divided with them.

[00:19:56] Scott: Let's talk a little about aerobic respiration, and how we can produce 36 to 38 ATP versus anaerobic respiration, which my understanding yields only two. So, when would the body be in one state versus the other? How do we shift the system more towards aerobic respiration to maximize the production of energy and ATP?

[00:20:17] Dr. Bryan: So, I mean, the short answer, like the high school biology textbook answer would be if there's oxygen available in the cell, then the cell is going to make that 36 to 38 molecules of ATP per unit of glucose going into the energy production pathways. I would say that the expanded answer would be that's going to happen, you need oxygen, absolutely. But then you also need all the other myriad cofactors required to run all the reactions of glycolysis, the Krebs cycle, the electron transport chain. So, I guess summarizing that, it would be as long as there's adequate nutrients including oxygen, then you're going to make the higher amount of ATP from a sugar molecule. What I think is really interesting to think about is that at the end of the day, it just really all comes down to energy. Because ultimately, energy from the sun is captured by plants when they're chloroplasts, which are their version of mitochondria. So, they have them too and it’s all very fascinating, I think. And so then they harness that energy to make, you know, energy for themselves. So, like sugar molecules, essentially. And then they can make vitamins and minerals and amino acids, and all the stuff we can't make, and then we eat those plants to get all that stuff that we can't make, or we eat the animals that have eaten the plants to make the stuff that they can't make.

So, ultimately, we're-- for all intent and purposes, we're harnessing energy from the sun and then through food, it ultimately gets into us. And then it's up to our bodies to break those food molecules down so we can get the energy out of it so we can make ATP. So, it really all comes down to ATP in some senses. And so, ultimately, our bodies had to evolve to the point where we can make that much ATP because we have just these really big bodies, relatively speaking. We have like, I've heard at least a trillion cells in the average human body. Some people say 100 trillion. Either way, it's a lot of cells, and we need that much ATP. And when we don't have enough, because we have an inadequate amount of oxygen, or an inadequate amount of nutritional cofactors, then we're going to start to suffer and we're hopefully not going to die. You know, most of the time, that doesn't happen due to a frank ATP deficiency unless you asphyxiated or something crazy like that. But you can certainly start not functioning quite as well. So, as a person's body is kind of falling off the rails like I'm feeling tired, I'm feeling brain fog, I'm feeling crummy, my body's not healing as well, bruising easily like all these different things that some of our patients are suffering from. Well, you're probably not making just two ATP at a time, but you're probably not making 36 to 38. You might be somewhere in the middle. It's kind of a mix between oxygen-dependent and oxygen-independent energy production. And at the end of the day, the person's alive but they're not nearly functioning as well as they should.

So, to try to optimize oxygen levels, I think that of course, you know, well breathing is important and trying to optimize breathing as best as possible. And whether that's tapping into things like, you know, the Buteyko Breathing, you know, mouth taping, that kind of fun stuff. Or if it's doing deep breathing, or it's trying to spend as much time as possible in places that have the least amount of air pollution, so you're hopefully getting a higher constant of oxygen coming in. Or you're using fancy pants things like hyperbaric oxygen therapy, or ozone therapy, or exercising with oxygen therapy, or all these lovely things that are out there. I think you can artificially jack up your levels more, but I think that in general just trying to breathe and you know, exercise and that kind of thing, that's probably the-- typically a more than adequate way to get enough oxygen for the average healthy person. People with chronic illness might need to do some fancier things to get themselves on track faster.

[00:23:55] Scott: I love my mouth taping at night while I'm sleeping.

[00:23:57] Dr. Bryan: Awesome, me too.

[00:23:59] Scott: Another good reason to mouth tape. When people have low energy or fatigue, the first thing I think we think about is thyroid and maybe even more so adrenals. So, what's the interplay between the mitochondria and the adrenal or a thyroid in terms of their function?

[00:24:18] Dr. Bryan: I think that's a really fascinating area to think about because as a naturopathic doctor, we certainly talk a lot about these adaptogenic herbs that are ultimately working to help support the hypothalamic-pituitary-adrenal gland axis or HPA axis, or you know, what we used to call it was just good old adrenal fatigue. And then now people will tease you if you call it that because it's not technical enough, I guess. I've been teased by students nowadays who say HPA axis support. Anyways, either way, it's something that I've seen in my own clinical practice, especially before I started treating patients with complex chronic illness. And even as that part of my practice was starting to build, just treating more like standard cases, less complicated cases of people saying like, “Yeah, I'm just kind of tired. I'm kind of depleted. I'm working too many hours, this, that and the other thing. Those types of adaptogenic herbs like Rhodiola or Schisandra, or…or Eleutherococcus rather, aka Siberian Ginseng and others, can be really helpful. I've seen adrenal support or HPA axis support, herbs helped so many patients over the course of time. And then similarly, with thyroid support, sometimes that can be really helpful too.

In my opinion, in my understanding, there's a lot of overlap between herbs that support the thyroid versus herbs that support the adrenal glands. But in terms of, you know, different nutritional supports and iodine and things, sometimes that can make a big difference. As I have, over the course of time, as I was treating more patients with complex chronic illness, I found that those herbs wouldn't really pack as much of a punch. Sometimes they wouldn't help at all. And I would say that in the average population, if it would help, you know, say 75% of people with energy levels, at least to some extent in a complex chronic illness population, I'd say you know, maybe like 20-25%. And if it was helpful might be like, “Yeah, like my energy was the three, now it's like a three and a half or a four.” So, I found that it wasn't really getting great returns. And so I wouldn't always use them all that regularly until a patient was at least, you know, halfway better, let's say and then maybe we'd start getting more traction with something like that.

What's really fascinating though, is when you think about adrenal gland physiology and thyroid gland physiology and their hormones, cortisol, and adrenaline and thyroid hormone, they ultimately all work through stimulating the cells to make more ATP through the mitochondria. So, cortisol, for example, the main thing that it does is it helps to regulate the amount of available glucose, sugar, for the body to be able to make more energy on demand in different tissues in the body and the face of stress and that's all done through the mitochondria. So, an adrenal gland or adrenaline or cortisol doesn't directly make energy, it needs the mitochondria to do that. Similarly, with thyroid hormone, it also helps to liberate different nutritional factors so that the cells can make more energy. You know, whether it's from glucose or fat or amino acids, it helps to improve absorption of nutrients in the intestinal tract so that the body can make more energy on demand. It is something that certainly has a direct impact, like on heart rate and things like that. But in terms of actually making energy, the thyroid gland ultimately stimulates the tissues to stimulate the cells and their mitochondria to make more energy.

What is interesting with thyroid hormone, T3, specifically, the active form of thyroid hormone, is it's a big driver of mitochondrial biogenesis. So, when adequate T3 is being produced, then there's going to be more mitochondrial replication and we're going to have more mitochondria per cell, which is a good thing. So, what I further think is that well, why is it that over the course of time I've heard so many stories about people with say like, you know, a so-called Wilson's Temperature Syndrome or sub-laboratory hypothyroidism, where it's like “Oh that sustained-release T3, like it was a miracle. It's amazing.” And then I'm sure many cases get all the way better and stay better. But then I've heard of other cases of patients in my

practice where yeah, the T3 or the desiccated thyroid, it really, really helped. I had this honeymoon period of several months or maybe years, where it was great. And then it just stopped working. It's like well, if there was mitochondrial dysfunction, and then you ramped up the T3, and then you're feeling better, but you're kind of like, you know, trying to beat a dead horse here. It's like you got a little more life out of those mitochondria, but then eventually, you know, they're just not an inexhaustible resource, you really didn't get to the root cause there. So, I think that if a person is feeling fatigued, and or brain foggy, what have you, and adaptogenic herbs or thyroid support isn't really doing the trick, or it's only partially helpful; one would want to be looking further, I guess upstream in this case, to see what-- is there may be a mitochondrial dysfunction component contributing to the lack of efficacy there.

[00:29:04] Scott: Good stuff. Let's go on a little bit further with the adrenal conversation in terms of how the mitochondria might be impacted by constantly being in a sympathetic dominant state. So, does the body need to be in more of a parasympathetic state to really improve mitochondrial function, and is there any connection between our nervous system and our mitochondria?

[00:29:26] Dr. Bryan: So, there's definitely a connection. And it really kind of-- the answer comes down to two categories. It's well, what do we see clinically? And then what do we see actually on, a more of like a basic science level, like on you know, actual physiology studies. From a clinical perspective, any clinician who's been at it for more than six months and is living in our society in this day and age is going to be able to say like, "Yeah, people who are in that sympathetic dominance, they're going to tend to get burnt out more easily. They're probably going to be more tired. They're going to be more prone to crashing. And the people who do try to have a more balanced lifestyle, try to do the deep breathing, they' trying to get better sleep, this type of thing. If they're kind of promoting a lot of those sympathetic nervous system functions, if they're doing vagal nerve stimulation and all the fun things that are out there now, then those people are generally going to probably feel better. They're going to heal better that type of thing." So, clinically, where, once again, energy levels are pretty much synonymous with mitochondrial function because mitochondria make ATP, energy is, you know, ATP, basically, in your cells, then there's definitely a clinical connection, in my opinion, and in my experience.

On more of a basic science level, there have been some studies done showing that when there's more sympathetic stimulation happening in animal models, then the mitochondria don't function as well. And then conversely, in certain models where they cause you know, some kind of like heart injury or cut off the blood supply to some poor animals in the study and then there's, you know, the cells are dying. If they're stimulating the vagus nerve, which is basically synonymous with the parasympathetic nervous system, if they're stimulating that at the same time, then the amount of mitochondrial damage and subsequent cellular damage is drastically mitigated. So, there definitely seems to be basic scientific evidence and certainly clinical evidence that they're, yeah, promoting the parasympathetic tone is important, and then too much sympathetic is probably bad.

[00:31:22] Scott: Let's talk about some of the key items that stress the mitochondria. So, I've heard some suggests that heavy metals like aluminum can impact our mitochondrial function. Mold toxins or mycotoxins potentially play a role in suppressing mitochondrial function. What about chronic infections? Let's talk about, for example, Lyme or coinfections. What is the role of these toxins and microbes in terms of our mitochondrial function and how important is it to treat those in your treatment protocols in order to improve mitochondrial function long term?

[00:31:56] Dr. Bryan: So, everything that you listed there, there's evidence indicating that they're bad for the mitochondria. Starting at the top of the list with heavy metals, every heavy metal you can think of whether it's, you know, cadmium, lead, aluminum, mercury, what have you; all of those have research showing that they are deleterious to the mitochondria. They seem to have kind of this like one-two punch really double whammy effect where the presence of those metals in the mitochondria will react with the plenitude of oxygen that's in a mitochondria, hopefully, a plenitude and can easily create free radicals from that oxygen. So, they're free radical generator, free radicals being you know, also known as pro-oxidants, or the opposite of antioxidants, so damaging the tissues. So, the presence of heavy metals in the mitochondria jack up the levels of free radicals which are damaging to the mitochondria and damaging to the cell in general. And then also heavy metals, in general, will bind to selenium and sulfur residues in the tissues as well. And virtually every antioxidant producing enzyme typically has selenium and sulfur residues in it.

So, things like glutathione peroxidase and superoxide dismutase and catalase and so that will interfere with the way that the enzymes work, and so you'll wind up not producing as many antioxidants. So, you get this double insult where the heavy metals are inducing more free radicals, not as many antioxidants and so then the mitochondria can just really start getting damaged and the cell can start getting damaged. In terms of mycotoxins, every mycotoxin that's been studied at least in vitro has been shown to be damaging to the mitochondria and either interferes with its function, their function or it actually will trigger the mitochondria to promote just the overall death of the cell. So, mycorotins are just, they're just awful. And I know I'm preaching to the converted on this one, but like they're just awful and they're really bad for your mitochondria. And it's quite interesting because when we get our mycotoxin panels and like look at all these different mycotoxins, every single one of them on the panels that I've seen have all been studied, and they're all deleterious to the mitochondria.

In terms of Lyme and co-- Borrelia and coinfections, diving into the literature on that, there really aren't a lot of studies, there really aren't many studies at all, drawing connections between direct mitochondrial toxicity in the presence of those microbes. There was one study looking at patients with Borrelia like persistent Borreliosis, and they found that they had higher levels of superoxide, which is this really, you know, pretty nasty, free radical, and lower levels of calcium in the mitochondria of the white blood cells that were taken out of these human subjects. And so those two things indicated that there's more damage happening in the mitochondria and more inflammation happening because the lower calcium in the mitochondria is indicative of that. You see that in other inflammatory models and other types of studies. So, that was from Borreliosis.

In terms of Chlamydia pneumonia, Ehrlichiosis, Anaplasmosis, maybe Mycoplasmolysis; there are some studies that show that there is some degree of mitochondrial dysfunction with those coinfections, but it's not really super cut and dry, whether that would have-- it's not really cut and dry exactly what impact. And then I wasn't able to find any literature looking at Babesia or Bartonella on direct mitochondria impact or not. So, from a basic science perspective, there's really limited research there. Of course, from a clinical perspective, you know, like, do they cause mitochondrial dysfunction? Obviously, they do because they can induce fatigue and brain fog and muscle issues and all this stuff that is directly related to mitochondrial function. But there's a deficiency of research, unfortunately, definitively proving that. What was the very last part of your question, Scott? It was a very long question, a great question but very long.

[00:36:03] Scott: How important is it to treat those toxicities or infections in order to improve mitochondrial function?

[00:36:11] Dr. Bryan: It's very important in my opinion, and one of the things that I've seen in my practice is every so often I might have a patient where I think, well, like you know, you're really depleted, really fatigued, but you don't have a really really strong picture suggesting that there is necessarily really active ongoing infection. Or you're really depleted and fatigue but you were in that moldy environment for a long time and you've recovered you know, quite a bit since then, but you're still just feeling really tired or whatnot. So, let's just maybe start you on some mitochondrial support and like you know, if you don't have money to do any testing and this, that, and the other thing I'd say like, let's just start on some mitochondrial support. And sometimes that goes great and sometimes it's just a big like swing and a miss. Not a swing and a miss but kind of a swinging a, like very anti-climactic like, and I've been on this protocol for a month and I feel no different. It's like okay, there must be something going on that's still interfering with your mitochondrial function. And so we're going to have to start digging a bit deeper. But in virtually every case, I'm thinking like, "Yeah, let's work with mitochondria but we have to remove all of these roadblocks to your optimal mitochondrial function, which could be Lyme, coinfections, metals, mold, stress, etc, etc.

[00:37:21] Scott: When we look at broader categories of microbes, so parasites, viruses, fungi, or bacteria; have you observed any particular group being more impactful to the mitochondria or is it pretty similar?

[00:37:35] Dr. Bryan: You know, I think it's kind of like I've heard you asked a number of your guests over the course of time, Scott, you know, like, which coinfections for example, like seemed to be the most impactful or you know, it's like mold, like mycotoxins versus coinfections or Lyme or whatnot. And I think that, you know, from a clinical perspective, it's probably like, you know, the Bartonella or mold illness or Babesia that maybe that would…Mycoplasma that maybe pack the biggest punch because stereotypically, they might be the most fatigued provoking. But there's been no research directly telling us that so it's all kind of conjecture.

[00:38:11] Scott: We know that our environment has become exponentially more polluted with electromagnetic fields, electromagnetic radiation. Is there a connection between EMF or EMR exposure and mitochondrial function? And if so, what are some of the things that you recommend for your patients to mitigate that potential mitochondrial toxin?

[00:38:29] Dr. Bryan: So, the research on that is really complicated, because of course, it has to-- you have to take into account which species of you know, animals being tested. You know, is it a human with a long lifespan versus a rodent model with a relatively short lifespan? What's the frequency with the amount of exposure there's all these different variables. But there are a couple of review articles out there on this and the general take-home message seems to be not a big surprise, but that they're bad for our mitochondria. It seems to result in more reactive oxygen species production. It seems to interfere with some of the complexes in the electron transport chain, which is an important part of the energy production pathways in the mitochondria. And interestingly enough, it can also induce an… EMF/EMR can induce the formation of this, what's called the mitochondrial transition pore. And that's something that seems to be, it's kind of an important part of mitochondrial function. But if this pore this gap, if you will, on the inner mitochondrial membrane is open to at the wrong time, then it can cause to my understanding really significant irreparable damage to the mitochondria. And so that's just an extra way that EMR might be that much worse.

So, what I think is that there may very well be a synergistic relationship between EMR and mycotoxins, heavy metals, microbes, because if the EMR/EMFs are able to create this increased permeability of the mitochondrial membrane and maybe the cell membranes as well, then I'm wondering if it creates just an easier access for the microbes to go intracellu or for the toxins to get intracellular. And then that ultimately exacerbates them. I know of course, Dr. Klinghardt talks extensively about how important EMR mitigation is in his patient population, and how that might be part of why we're, as we're seeing ever more EMR exposure to our species and our respective countries at least, and others, of course, that we're seeing people like you know, getting maybe sicker and seeing more complex chronic illness and seeing slower recovery time. And I know it breaks my heart but I see more and more younger patients now having conditions like you know, Chronic Fatigue or Fibromyalgia which of course can many times be related to infections and mold and etc.

And just, you know, younger and younger and I just really wonder what impact the EMR has. In terms of mitigating exposure, you know, trying to, of course, you know, not put our ce phones or like our smartphones right next to our heads, trying to, you know, speak on speakerphone, trying to, you know, do the best job possible to shut off the breaker at night. So, just keeping the bare essentials, running, you know, available overnight, shutting off the Wi-Fi, and then people who are really electrically hypersensitive, then of course, you might need to do more drastic things than that. But those would be some basic things I'd recommend.

[00:41:32] Scott: Let's talk a little bit about the Cell Danger Response that Dr. Bob Naviaux has talked about. First, maybe at a high level, kind of what is it but then in that model, where ATP is the danger signal; I've wondered if supporting the production of ATP too early in someone's recovery potentially reinforces that Cell Danger Response. What is the connection between Cell Danger Response and the mitochondria?

[00:41:57] Dr. Bryan: So, the Cell Danger Response is basically where some type of threat and you know, a virus is what Dr. Naviaux talks about, for the most part. But my understanding is things like metals and possibly other toxins and I would say probably anything that goes intracellular could trigger it. So, like pretty much Lyme and coinfections for the most part, mycotoxins So, basically, when the cell's under threat, it essentially goes into lockdown mode. And it really down-regulates the production of ATP by the mitochondria. It down-regulates methylation, it down-regulates a bunch of different functions in the cells. With the general-- my general understanding or the way I explain it to patients is that the cell is just saying like, we're going to go into lockdown mode so that the virus or the microbe or whatever just can't use the cellular resources too for its own benefit to replicate. So, it's like let's just kind of lock it down and then kill it off and then move on. One of the-- when that doesn't happen like when the threat is not neutralized or eliminated quickly, then part of what will happen is the cell will release ATP outside of the cell. And to my understanding the concentration of ATP inside of a cell is about a million times greater than the concentration of ATP outside of the cell. Every cell in the body, according to what I read from Dr. Naviaux has ATP receptors on the outside of it. So, it seems like it's this universal signal that if the cells say, "Hey, why is there more ATP outside of the cell now? There must be some danger, I better go into lockdown mode too."

So, it's a really good question in terms of, you know, is there a time when you shouldn't support mitochondria, we want the ATP production to be kept lower. And I've certainly seen in practice that sometimes mitochondrial support started too early or at the wrong time or too aggressively can definitely flare people up. Where it gets kind of complicated is that for many of the agents that we'd use to support mitochondrial support-- to support mitochondria, rather whether it's what I would consider to be like super-comprehensive mitochondrial support, which is what I would usually recommend. Or it's more of like a, I'll say partial mitochondrial support I'm using, you know, certain key agents or some of the ones that are emphasized the most in the literature. You can almost always tie those agents to some other physiological process that might be accounting for the detox. So, for example, in my mitochondrial support protocol, there's fairly high dose taurine in there. And taurine is wonderful for mitochondrial function, but it's also involved in bile elimination things in bile, you know, phase 2.5 detox pathways. So, I wonder well, if you're flaring on that, is it because you're flushing your toxins out too quickly, and we don't have enough binders or whatever it is in place? Or is it that the mitochondria are being supported too quickly, and it's exacerbating a Cell Danger Response?

So, unfortunately, until somebody studies it, which I don't know if anybody ever will. But at least clinically, you can definitely start a protocol too quickly or at the wrong time. And I do wonder if it's in part related to that ATP too quickly. In certain cases I'll say, well, if we're building up your mitochondria support protocol and you can only tolerate, say a 25% dose and then you start to flare beyond that. Maybe we'll keep it there. Now let's start ramping up your antivirals because we did some serological panels and we saw that there's high viral titers. So, let's start building up on that. And if you hit a plateau on that dosing, because otherwise you're Herxing, now let's build up your mitochondrial support. And so I've definitely seen those types of give and take sort of protocols. And so clinically, I think that there are probably cases where jacking up the mitochondrial function too quickly is going against that Cell Danger Response ultimately, to the patient's detriment, so I'm always very careful with those types of cases.

[00:45:40] Scott: We hear more and more about people struggling with dysautonomia is like POTS, hypermobility syndromes like Ehlers-Danlos Syndrome. Is there a connection between the mitochondria and POTS or EDS?

[00:45:54] Dr. Bryan: So, in my clinical experience, yes, because I've seen mitochondria support be very helpful for some of those patients. And especially in the case of EDS where, of course, there's the connective tissue issue, what's really interesting with mitochondria is that the-- as a backup fuel source, mitochondria will burn proline, the amino acid proline. And proline is actually really interesting in that it's not just a fuel source and it's not just for collagen synthesis. But it's also involved in redox balance, and it's involved in electrolyte regulation in the mitochondria and it's kind of fantastic stuff. And so just where Proline is one of the three primary amino acids in collagen, I do think that there's probably a special connection there with EDS. What is interesting is I've had patients with EDS and you know, oftentimes with lots of comorbid symptoms as well, and sometimes they will…some of the biggest mitochondrial support flares I've seen have unfortunately been in patients with EDS. So, I think there's probably something really significant there, and whether it's because they're harboring Borrelia in their connective tissue and then as we're getting that on track, it's flushing it out. Like I don't really know, but it is something I'm extra cautious in those EDS patients with mitochondrial support.

[00:47:15] Scott: Anesthesia is a known mitochondrial toxin. So, how can we protect our mitochondria if we do need a procedure that requires anesthesia? And then is the negative mitochondrial effect of anesthesia, is that short-lived or can it persist longer term?

[00:47:31] Dr. Bryan: Yeah, I mean, I don't think that I-- I mean, I've never-- That's not true. Actually, I had one patient who had a like post-anesthesia syndrome, and it wasn't anything too too major. But I did see that one time, and I don't think it was really mitochondrially mediated per se. I haven't really heard a lot about that like nothing near like, you know, people getting floxed or something crazy like that. Or even like post like, you know, gadolinium toxicity stuff which is rare, but I've heard some cases of it. So, it's not-- I'm not sure how significant it is. But I think that if one-- well really, if someone's going in for a procedure where they'd be requiring a general anesthetic, obviously with, you know, certain exceptions, but as a rule, I'd usually recommend going in on some things that would ultimately be supportive to the mitochondria because we want their bodies, in my practice at least, I want-- assuming it's like a surgery as it would be, we'd want their bodies to be loaded up with all those nutrients that are going to be required to heal, so that once they're, you know, post-operative, they're going to heal quickly. So, I would usually recommend that loading up on mitochondrial support nutrients for say, at least a couple of weeks leading up to the procedure, depending on their baseline state of health. And then once they're, you know, post-operative, they're no risk of any interactions with, you know, whatever medications they might be working with, then probably put them on that protocol again. So, I think that would just be sort of-- that's kind of just inborn in my surgery support protocols. But that's what I would generally recommend.

[00:49:06] Scott: Given that mitochondria evolved from bacteria, do you think there are some potential downsides of long term use of antibiotics in the treatment of Lyme disease, for example, in terms of longer-term mitochondrial function, and potentially fatigue and energy-related issues?

[00:49:23] Dr. Bryan: Yeah, I think it's this really amazing concept. They're not concept. They're really amazing reality that, you know, mitochondria being these remnants of these ancient bacteria that created this symbiotic relationship with these other cells. And we kind of carry these little bacteria remnants around, you know, and the numbers of hundreds to thousands or maybe millions in a substantia nigra neuron in our bodies. And so, over the years, I've thought many times about it, where it's like, wow, like we got these bacteria. So, if

we're doing things that are bad for bacteria, maybe they're bad for us because we have these mitochondria that are bacterially-drived. Where, you know, looking at the literature, antibiotics are as a rule bad for our mitochondria. But it just really kind of begs the question in my mind, is that actually clinically relevant? I think that it really comes down-- Because what happens in a petri dish doesn't necessarily translate into what's actually relevant in clinical practice. Because in a petri dish, there's not all these compensatory mechanisms and pathways and things like that to maybe circumvent the negatives from antibiotics.

So, I think that my general take would be that if a person has a condition that they need antibiotics to save their life or to significantly reduce the likelihood of them having a crazy issue, like say, if you have a kidney infection, like you probably want to be on antibiotics, then I think that it's a good idea to go on them. And if one had any post-antibiotic symptoms, aside from maybe some indigestion or yeast infection or something like that, they're like, “Oh, wow. I went on an antibiotic and I feel tired.” It's like “Yeah, let's support your mitochondria to get you back on track because maybe you had an issue there.” But I think in general, for patients who are working with say more of an antibiotic centric or heavily influenced approach for treating their chronic infections, I think that it's just important to kind of think about how those therapies are affecting them. If it's, you know, kind of a transien Herx, then maybe that's just the cost of doing business in that type of model, but if it's like I'm on these antibiotics, and I've been on them for months or years, and like, I'm just feelin like more dragged out than I did at baseline. Or my pain’s going down and my crazy neurological symptoms are going down, but my brain fog and my fatigue are just really not comi around. I'd be thinking, well, maybe your mitochondria are, you know, having a hard time recovering because maybe the antibiotics are having a deleterious effect. Fluoroquinolones are a totally separate kettle of fish, but for general antibiotics, that would be my thoughts.

[00:51:52] Scott: So, let's talk about the fluoroquinolones. So, things like Cipro or Levaquin which used to be used more commonly in the Lyme and coinfection realm particularly wi Bartonella. There is some thought that these potentially poison the mitochondria and lead to as you termed it floxing, so people that have these very significant issues with tendons fo example. What effects do we think these medications have on the mitochondria, and then once someone has been fluxed, have you observed that a focus on mitochondrial support can reverse the damage that those drugs have potentially cost?

[00:52:26] Dr. Bryan: So, yeah, there's some just for clinicians listening. There are some interesting review articles looking at fluoroquinolone toxicity. And I think they're really interesting reads. With fluoroquinolones, they are definitely toxic to the mitochondria. And what makes them kind of special and not in a good way, but unique from other antibiotics is that their structure is such that they have a negative charge on one end of the molecule and a positive on the other, which is something called zwitterion and I just like saying that word. So, I thought I'd just have a chance to say zwitterion the second time now, but those zwitterions are able to bind up metals that have a 2+ valence. So, like a 2+ the atom atomic symbol as written out. And so that includes iron, iron can be a 3+ as well, but can also be a 2+, zinc, copper, and manganese. And so that's really problematic for the mitochondria because they need copper to make cytochrome C, which is part of the electron transport chain. They need manganese to allow superoxide dismutase type 2 to work, which is the main type of superoxide dismutase in the mitochondria.

They need iron as a building block of all of the complexes in the electron transport chain and, you know, heme synthesis and all the things that iron is really important for. And then in terms of zinc, it's a cofactor in just so many different enzymes in the body. And so by sequestering those metal ions, it's going to get in the way of how well the mitochondria can function. But the really big issue over and above that is that at a cellular pH level, fluoroquinolones are not very soluble. So, they get into the cell and then they kind of sediment there. And they don't solubilize easily. And so they just kind of stay stuck there and so it's kind of like you've got, you know, like just a really bad roommate that just won't leave like it’s just they're sequestering all these resources, and this doesn't go anywhere. And some of those resources are very important not just for general mitochondrial function, but also for tendon health and integrity. And so seeing these connective tissue issues and tendon ruptures and all the crazy stuff that happens.

So, fluoroquinolones are, you know, can be a really really big problem for people that bioaccumulate them more easily. In terms of mitochondrial support in those patients, so looking back in hindsight with the people with fluoroquinolones toxicity that I've seen, mitochondrial support interventions were probably the most successful things that helped with them. With that being said, you know, I think a really comprehensive mitochondrial support would probably be even more useful. But it's something that would have to be managed really, really carefully. Because if you start mobilizing things too quickly, it could-- like these patients can be really, really sensitive. So, it's something that you really have to handle those cases with kid gloves in my experience.

[00:55:34] Scott: With some medications: Disulfiram, for example, becoming very popular in the Lyme community for treatment of Borrelia and Babesia, Cholestyramine very commonly used as a binder for removing mycotoxins. These and other medications may have negative effects on the mitochondria. So, what are your thoughts? Are the benefits of using them to address or remove an infection or remove a toxin from the body that might have an effect on the mitochondria as well; are the benefits greater than the potential downsides from a mitochondrial perspective?

[00:56:10] Dr. Bryan: I think they are, yeah. I think that, you know, looking at the literature on those particular agents, with Cholestyramine if memory serves, the literature is a little bit mixed on that, like, I don't think it's really a slam dunk like, yeah, it's definitely bad. I think that it's a little bit more like case report based as opposed to like, “Oh, it’s definitely bad.” Disulfiram on the other hand, like yes, like it is definitely not great for the mitochondria, but it's just that cost benefit ratio that I believe you alluded to. What I do speculate on though, and I got a really great schooling on Disulfiram from the interview you had not too too long ago. So, that was great. And one of the things that I took away from that was that sometimes patients are just really tired when they're on the Disulfiram, and I do wonder if that's something that might be related to the fact that they've got this mitochondrial-- there's a deleterious effect happening on the mitochondria. And I know not every patient would have that with Disulfiram but-- So, I think it's kind of like the antibiotic question, you know, if someone was like, “Well, I'm on this medication, this treatment, and I'm really just getting tired and dragged out.” It's like, “Well, maybe it's not the best fit for you, or we should be supporting your mitochondria concomitantly.” But I think the benefits probably outweigh the negatives.

[00:57:25] Scott: Let's talk a little bit about testing. So, what are some of the better tools for assessing mitochondrial function? How do we know if our mitochondria are weak or strong? What are the things that you use in practice? And then are there any of these tests that actually lead to actionable steps?

[00:57:43] Dr. Bryan: Yeah. So, there are a few different tests out there. And what I think is important to note is that where the mitochondria are so complicated, you know, there's so many reactions you know, probably about a dozen or so reactions, maybe eight reactions, leading up to the point of entry of the Acetyl-CoA into the mitochondria through glycolysis. So, those are important. And then with the Krebs cycle and probably should have counted these before this interview, but it's like maybe another eight or nine, you know, intermediates in the Krebs cycle. And then there's several steps along the way with the electron transport chain. So, there's all those components, and then that's to say nothing of the redox balance. So, the antioxidant pro-oxidant balance, and certain transporters, like, say carnitine transporters, get fatty acids into the mitochondria for beta-oxidation, etc, etc. So, there's so many moving parts. And so when we look at some of these profiles that might look at things like, say the MitoSwab, for example, which is this cheek swab that looks at the integrity of the different complexes of the electron transport chain, like well, that's really interesting. And if that looks abnormal, and it correlates with symptoms, then that's probably useful to know about that. But if the MitoSwab looked fine, it doesn't necessarily mean that the mitochondria are totally perfect, because there's so many other moving parts that could be off kilter. Or in an analogous way, say the organic acid test, which measures certain organic acids, so certain components of the Krebs cycle or glycolysis, or certain amino acid metabolites that relate to certain complexes in the electron transport chain. Same thing if they look abnormal, and it fits the picture well, that's probably relevant.

But if organic acid profile looked fine, it doesn't mean the mitochondria are perfect. Dr. Sarah Myhill’s panel, which I think is really really interesting, looking at different ATP ratios and different cell types, and the presence or absence of certain nutrients and then measuring certain other markers in the blood. I think it's really, really fascinating in her paper, which was-- I can’t remember how many years old now. It was like, “Oh my gosh, they look at 98.5% predictive value in picking out you know, control subjects versus patients with ME/CFS.” But then earlier this year, there's a paper that came out basically refuting the results. And now it's like, she hasn't had a chance to really formally refute, but kind of okay. Well, that's disappointing but just, I think that what it comes down to is that of the different tests that are available, they're very interesting. And I think that having all those tests done would be great. But I know in my practice, where a lot of my patients have limited resources, and in Canada, we don't have any extended insurance policies that pay for any testing at all. I understand it’s a little bit different in the States with some plans, and I don't pretend to understand US healthcare in any way, shape, or form. But basically, it's something where we don't usually have the luxury of running multiple tests.

And so what has really come down-- What it’s boiled down to in my practice is trying to be as clinically savvy as possible to know when to pick up, when should we go after mitochondria, and how do we know if they're getting better. And I have this, you know, amazing screening test, it took me a long time to figure it out. But basically I just asked the patient, do you feel tired and depleted? And if they say yes, then I'm thinking their mitochondria probably aren't functioning optimally. And so that's kind of my screening test. And then if we can do an organic acid test, or we can do some other type of tests, then that's great. And there does seem to be some useful clinical correlation with organic acid testing. It is a

test that I do recommend for my patients. It's the most accessible one out of the ones we've talked about so far and it does seem to be useful. And I do find that patients where they're getting better, when people are getting better they never want to retest, but on rare occasions they do. And then we'll see like, oh, yeah, those markers are actually looking better. So, it does seem to be useful, but there really is no perfect test out there.

[01:01:47] Scott: Let's move on to talking a little bit about treatment. So, what are some of the favorite tools that you use? I know you have some well-known ones and some lesser-known ones in terms of either individual materials like PQQ, for example, or if there are specific formulas or products that you use in your mitochondrial protocols.

[01:02:06] Dr. Bryan: Yes, So, my mitochondrial support protocols have evolved or changed quite a bit, evolved in my mind over the course of time. When I first started using mitochondrial support, I would just basically pick, you know, different formulas that were prefab by different companies. There's, you know, seems like most major companies have their mitochondrial support formula. And it's like, okay, like you're feeling tired or like we're, you know, getting some good headway with most of your symptoms with, you know, the herbs or detox or whatever we're working with. But let's start, you know, trying to get that energy level up more and so, you know, bringing in some mitochondrial support to help with that. And I found that for the most part, it was pretty much a swing and a miss almost every time. Like I didn't really see a whole lot of change happening, and maybe it was not quite the right product or not quite the right timing, and maybe it was, it might have been my fault, I don't know. But at the end of the day, I just really didn't find there was much traction there.

So, for, you know, years, like I just didn't even really think about the mitochondria as far as it goes like, you know, maybe we'll throw it in there. But it was just not something I really ever saw anything being terribly exciting, didn't really hear colleagues or presenters talking about anything that was super exciting. It's like "Oh, I put everybody on CoQ10." "Well, does it make a difference?" Like "Well, I'm not sure because they're doing million and one other things." Like it has never heard a compelling enough story to say look at definitely that. But over the course of time, I took a couple of different runs at mitochondrial support. And what I've found over the course of time, is that by working with a really comprehensive support, it seems to be-- it can be very, very helpful for patients. I've seen a comprehensive protocol crack a lot of cases that I was otherwise struggling with, and that's always what's the most exciting for me, because, you know, it's kind of that number that people talk about at conferences a lot where it's like, yeah, you know, 80% of the cases, you know, like they go pretty well and we're going to figure it out pretty efficiently. And then there's the 20% that are like oh man, like, you know, keeping me up at night or pulling my hair out trying to get them figured out.

And so what was very exciting to me was that once I figured out what was in my mind a comprehensive mitochondrial support protocol, then I started trying that on some of my, you know, my 20 percenters like the ones that just weren't getting better as quickly as we'd like them to or they were moving really slowly. And I started seeing it improve them really really nicely and really consistently and I got really excited about that. So, I started using it more and so oh wow, like they're actually kind of as you maybe touched on it I think at some point in our interview probably like oh, yeah, like that's kind of like a later on in the process kind of thing that doesn't really have a place until a person's most of the way better let's say or a lot of the way better. And say no, like now I'm-- I have cases where I put them on my antimicrobial protocol for their Lyme and coinfections and if, not for everybody but in well selected cases, comprehensive mitochondrial support and see like, "Wow, like you're getting better, like twice as fast or three times as fast." Like first follow up, "Like what? Like, all your symptoms are gone, you're already 90% better. Really?" Like because in an analogous case, it would take at least like three months to get there if we're like, you know, really lucky. So, it's by no means when I say that mitochondria support is a panacea, but what I've been finding is that in appropriately selected cases, it can be really helpful.

So, in terms of what that looks like, you know, when I say comprehensive mitochondrial support, so what that looks like is basically every single one of the vitamin and mineral and amino acid cofactors required in glycolysis, Krebs, and the electron transport chain, and in addition to that, working with antioxidants that are supportive like Alpha-Lipoic Acid or Resveratrol, which helps to support mitochondrial biogenesis and things to that effect. So, kind of my phase one support is putting patients on all of those things. And when I say it like that, it sounds like I'm sure most of your listeners are thinking like so, like the whole kitchen table is covered in bottles. And it could be, but thankfully, we have a really great compounding company in our community. And they put together a custom formula for me where basically I did the math on it. And before we had this custom formula developed, the patients would have had to take about 60 capsules a day, it would have been, you know, about five times as much as what the cost of this product is. But where they put everything into one formula, we keep it in a powder, which doesn't taste great. Everybody complains about the taste, but it's 20% of the cost and it's not 60 capsules, it's two teaspoons twice a day. Plus a high potency multivitamin, multimineral, so like a formula that has you know, good dosages of methylated B vitamins and the active forms of other nutrients and you know, good doses of trace minerals and whatnot.

So, between those two formulas together, that covers the vast majority of the foundational bases needed for supporting all of those reactions that I mentioned. And virtually all of those nutrients are at really good therapeutic dosages. So, what one might get, say in a multi-nutrient we might need like 10 times the dose of say Benfotiamine, for example. So, that custom formula that we have, it has 10 times the dose of Benfotiamine or has a, what I would say like a full like multiple gram therapeutic dose of taurine and carnitine and Vitamin B5 and some of these heavier hitters. There's other nutrients in there that I'd like to see in higher dosages, but we just have to kind of keep the cost-effectiveness in the stratosphere for patients. And so things like nicotinamide riboside or the dosage of Alpha-Lipoic Acid in the formula or CoQ10. Like I'd like to see them higher. At the TFIM, I'll be talking about specific dosages for clinicians there and whatnot. But the long and short of it is that we have really robust dosages and a really comprehensive formula. And that seems to be packing a heck of a lot more punch than what I'd see in the other more like partial formulas that maybe only had, you know, three ingredients at a decent dose and then a couple, maybe four or five other ones at tiny dosages which makes the label look really good, but it doesn't actually necessarily pack a whole lot of punch.

So, that's kind of my, what I described on my mind at least is kind of my like basic foundational phase one, if you will, comprehensive mitochondrial support. Taking it a step beyond that, if needed, would be adding in things like Creatine and Ribose. Ribose being a building block of ATP, having done some studies in animals and humans showing that Ribose supplementation directly translates into jacking up ATP levels in the cells and then Creatine also very, very fascinating as a molecule. It is involved in mitochondrial biogenesis, it's involved in redox balance, and then it's also a backup support for ATP. It's kind of our backup like last-ditch energy-storing molecule in our tissues. And so especially in patients who have a lot of muscle tightness, muscle fatigue, reduced stamina, I think clinically, like on paper and clinically, the Creatine can be really, really helpful. And then phase three would be, you know, potentially getting up to more of like a tailored type of protocol where it's like, yeah, we're going to maybe add in the PQQ, or we're going to add in higher dose CoQ10. Or we're going to get more redox support or something like that. But at the end of the day, it's the comprehensive like a phase one protocol, we've got it figured out to a very concise and in the grand scheme of things like very cost-effective approach for patients. So, it's really exciting because it makes a comprehensive mitochondrial support very widely accessible for people. And that's kind of the overview of what I've been using in practice.

[01:09:46] Scott: Is there a role for lipids like phosphatidylcholine and phospholipid blends in terms of supporting mitochondria?

[01:09:53] Dr. Bryan: Yeah, that would be like, you know, the phases aren't set in stone. So, yeah, phosphatidylcholine, I'd definitely bring that into the mix in some cases. When I was playing with different permutations of this, I used to have like mandatory like, yep, there's got to be some sunflower lecithin or just separate phosphatidylcholine, whatever it was in the mix. And what I found was that just clinically, it didn't always really make an obvious difference. And everybody complained about the taste of liquid because I wanted non-GMO stuff that had to be sunflower. And anyways, there was a compliance issue. But it didn't really seem to notice a huge difference. So, if there's a patient with a lot of neurological stuff, or if they have liver issues, and I really want to load up that phosphatidylcholine, then I would definitely recommend it more. Or sometimes I'd multitask and if I wanted them on glutathione, which the liposomal ones are usually in a base of lecithin, so they're kind of multitasking a little bit with that. But even things like glutathione like where yes, on paper, absolutely like that should be part of a comprehensive support. But what I found is that whether we brought in the liposomal glutathione or we didn't or the phosphatidylcholine or we didn't, or certain other fatty acids, it just for a lot of patients, it didn't really make much of a difference.

And so I've been trying to keep it as lean as possible. And then like phase two or phase three, if we need to go there, we might tailor it to someone else. I am really intrigued by some of the formulas out there that really emphasize the phospholipid blends for mitochondrial support. And I've, you know, really only heard anecdotal reports from clinicians. And it's something I'm really intrigued by, but it's not something I really played with, mostly because of the price tag associated with those products. And it's a little tricky to import them into Canada at a reasonable rate and stuff. So, the long and short of it is, I'm really intrigued by that. And my mitochondrial support protocols are certainly an evolving process. So, it might be something that we wind up, you know, I might find it's really crucial down the road.

[01:11:54] Scott: I've heard some practitioners in the past say that when they attempted to implement mitochondrial support in their patients they didn't see a lot, they got discouraged. But later they came back around and realized they were dosing things too low to really get the effects that they were looking for. So, the counter to that argument is if you dose too high, the mitochondrial machinery may start working better, they may start detoxing, potentially, then they have mast cell reactions for example if there's metals being

released, and so on. So, when you put somebody on these mitochondrial support protocols, is it a slow ramp or is it you know, right at the beginning there at the target dose of these things?

[01:12:31] Dr. Bryan: In my practice, it's-- Well, it depends on the patient. But if they seem like they're already doing quite a lot better, and they have a nice robust constitution and they've you know, quote-unquote, only been sick for say, eight months or nine months or less, then I might put them on, you know, full-blown right out of the gate. But for patients who are sensitive, like especially patients who seem to have histamine intolerance or MCAS issues, I will definitely start them really slow. If the maximum dose of this combo powder that I mentioned is say two teaspoons twice a day, I'd probably start them off at like maybe a quarter of a teaspoon once a day and then build it up every day or two. And to my good fortune, if you will, or my pleasure, my joy is that a lot of patients seem to tolerate it really well. And I think one of the really interesting things with the mitochondrial support unless there's like a phase 2.5 blockage, or maybe there is a Cell Danger Response thing or some other exogenous or extraneous variable; I think that by supporting the mitochondria so comprehensively, and where they are, you know, sort of at the root of-- I would argue at the root of human physiology, it seems to go pretty well like a lot smoother than say, if I was ramping someone up on an HPU protocol. Like that can be just like pulling teeth trying to get someone ramped up on that if they're really sensitive or toxic or even trying to get someone ramped up on a detox protocol. But the mitochondria support seems to go pretty well.

[01:13:58] Scott: Let's talk a little bit about NAD. That's a popular topic these days in the mitochondrial world. So, NAD, NMN, Nicotinamide Riboside which you mentioned, I personally have benefited from a compounded topical NAD cream. I'm curious, what are your thoughts on the role of NAD in supporting the production of ATP from the mitochondria And then are there potential risks of using NAD?

[01:14:22] Dr. Bryan: Yeah, So, I think that you know, Vitamin B, Vitamin B3, in general, is important. Looking at the literature on it, my understanding is that there are certain downsides of using different types of B Vitamins like niacinamide, for example, which is non-flushing, which is nice and inexpensive, which is also nice at high doses can inhibit certain substances in the body like sirtuins, for example, to my understanding. Something like Nicotinamide Riboside, there's not as much-- there's not a ton of research out there on it. There are some interesting pharmacokinetic studies, bioavailability studies. And you know, unfortunately, the best paper and most thorough paper on that, to my understanding looking at, I would like to read the conflict of interest statements on papers. And my understanding is that there is a notable conflict of interest with kind of one of the best papers supporting how Nicotinamide Riboside is so much more bioavailable, not to say it's not valid. Like conflict of interest are what they are, but they just don't think that there's super, super definitive research or information in the literature showing which type of Vitamin B3 is necessarily superior. I do think it's really important because Vitamin B3 is a crucial component of mitochondrial, like ATP production. So, I think it's really important. But at this point in time, I really haven't had the opportunity to play around with it saying like, well, you're on this mitochondrial support protocol. We've kind of got, you know, things are, you know, you're improving at a certain rate.

Now, let's throw in some extra, you know, NAD or NADH or NMN or NR or what have you and then let's see if it's really made a difference. What I have done, I guess, kind of in that vein, is we have run quite a few intravenous NAD treatments. We've also run NADH, which we can still access here in Canada. And the long and short of it is that I did have some patients who felt better on those treatments. But I found that it was, you know, I probably ran them on maybe 30 different patients, let's say, and some of them are like, “Yeah, I love that stuff. It's great.” And I think most of them that just didn't really notice much of a difference. And I know that there are certainly like, in the realm of like addiction, like I understand that megadose NAD can be super, super valuable, but in my patient population, at least, just didn't really see enough traction with it to really stick with it.

So, whether I didn't have them on enough mitochondrial support beforehand, or whatnot, I'm not really sure. But I just found other tools were more valuable, high dose IV NAD just takes forever to run, you can get certain side effects of higher dosages. So, you know, to address the question of safety, I'm not aware of any issues aside from the higher dose niacinamide potentially inhibiting sirtuins, or a higher dose niacin has been, you know, loosely associated with liver damage potentially and nobody wants the flushing anyway. So, who the heck takes regular niacin for the most part? But in terms of those other compounds, I'm not aware of any safety issues, but the IV can certainly cause like, some chest discomfort and breathing stuff and dyspnea and it's just a little bit of a pain in the neck to work with.

[01:17:37] Scott: You mentioned earlier the importance of melatonin in supporting mitochondria. So, can we take that further to say that blue light exposure at night is potentially bad for the mitochondria and do you generally recommend avoiding blue light in terms of sleep and melatonin production?

[01:17:54] Dr. Bryan: I definitely do recommend that. I love my blue blockers on my devices and on my blue blocking glasses and all that stuff. So, I think that's really important. Melatonin, as you said, is incredibly important for mitochondrial function, it is probably super relevant. Just I'm pretty excited about melatonin. And so just to share a couple of quick, interesting tidbits. To my understanding, based on the literature, Melatonin is a molecule that actually has existed since you know, the primordial goop of, you know, ancient, ancient Earth, the most primitive bacteria have this melatonin molecule in them, and the molecule has been largely unchanged from then until now. So, when there's something that's that ancient, and something that has not changed, it suggests that A, it's probably important and B, it's kind of perfect because otherwise, nature-- evolution would have changed it along the way.

So, what's also interesting is that they've done animal studies and they found that when you look at a cell, the highest concentration of melatonin is in the cell membrane. The second highest concentration is actually in the mitochondria. So, it's really important for the mitochondria because Melatonin is a very strong antioxidant. And the mitochondria are kind of like these little combustion engines where they're making energy but they also make free radicals as part of that process. So, they need a really good balance of the antioxidants and the pro-oxidants, so melatonin being super important. So, I think that preserving melatonin at all costs is a really good idea, and blue blocking would be a part of that strategy.

[01:19:25] Scott: Talk to us a little bit about the role of diet in mitochondrial health. Can a ketogenic diet potentially increase the number of mitochondria? What are the strategies you recommend from a dietary perspective to optimize mitochondrial function, but also biogenesis?

[01:19:41] Dr. Bryan: So, there are studies showing that caloric restriction can-- that helps to induce mitochondrial biogenesis, a ketogenic diet does induce-- support mitochondrial biogenesis both in, definitely in animal studies. I can't remember, I think in human studies as well, if I'm not mistaken. And so those seem to be the most important things to do. Now, that being said, there's research showing that things like quercetin and resveratrol and I'm sure other antioxidants as well help to promote mitochondrial biogenesis. So, I think from that one could extrapolate the eating lots of, you know, good, like healthy fruits and vegetables and things like that kind of in a Dr. Terry Wahls inspired sort of methodology is probably a really good thing to do for our mitochondria as well. With that being said, in terms of what I actually recommend for my patients, I generally recommend working with whatever diet is the most sustainable for them, and that helps them to feel the best. So, I'm not leery but I'm certainly cautious around the ketogenic diet.

And that, in part because some of the studies like they'll show like “Yeah, there's benefit.” But it's like, “Well, how long was this study? Was it a 10-year long study, or was it a week long study in rats?” And so just how long those effects are going to be beneficial for and the body can adapt. So, are the cells going to keto adapt, and maybe it's like wow like I really struggling through this, you know, ketogenic diet and I could really, you know, go for some carbs right about now, but like, I want to keep my mitochondria biogenesing like crazy. That's not a word. And then, you know, it's like, it turns out, that like, “Oh, yeah, like it, you hit an adaptation point at three months, and there's been no further benefit, and you've got an ApoE4 you know, unfavorable genotype and you're clogging your arteries. And it just-- I think it's really confused or well it is confusing, it's definitely convoluted. And so I think that it's not something that I really push a lot with my patients. But if a patient needs to lose weight, then you know working with a ketogenic diet temporarily like could be a good multitasker.

[01:21:43] Scott: Beyond supplements, what are some other tools that can stimulate or support the mitochondria? I know I'm a big fan of photobiomodulation or red light therapy. What are some other tools that you bring into your patient protocols?

[01:21:54] Dr. Bryan: The main ones that I would use would be photobiomodulation, the version of that that I like using the most is intravenous photobiomodulation. So, if anyone's interested, you can pop on YouTube and just punch in IV laser therapy and see videos of this. They've been doing human clinical trials with IV laser therapy since 1981 starting in Russia and there's-- Yeah, it's like it's a huge thing now in certain circles, but I've been using it in my practice for about-- I live in a bubble, Scott so I don't know…

[01:22:25] Scott: I was gonna say that's way fancier than mine.

[01:22:29] Dr. Bryan: Oh, no. Oh, no. Well, it's something that we've been using for about five years in our clinic, and it's a German machine. And yeah basically, it's just like, it sounds like you're hooked up to an IV catheter and then there's a little sterile single-use fiber optic cable that's fed into the catheter, it's taped down attached to a laser module and we're literally shining laser light into a patient's bloodstream. And what I think is really fascinating is that to my understanding, there are photoreceptors on all of the tissues in our body,

not just our retinas and our skin and things like that. So, meaning that there are light molecules like photon receptors in all the tissues in our body, the darkest recesses, which is fascinating for many reasons. But one of which is that hmm, by shining a light literally inside of a person's body, can we see physiological effects? And the research says, “Yes, we can.” And then clinically, we also see a lot of benefits with our patients too; there's other physiological impacts besides supporting mitochondria. But basically every-- Well, as you would have kind of alluded to there with photobiomodulation being supportive. One of the ways that it's supportive is that the complexes in the electron transport chain will absorb certain wavelengths of energy. So, you're literally irradiating those complexes and putting electrons through photons into those complexes to then go ahead and make more energy. So, it's almost like well, I don't know, maybe there's some way to rig up a photobiomodulation system where you wouldn't need to eat anymore because you can make energy that way but food so good, hopefully, nobody was would do that.

So, the IV photobiomodulation is something that we use a fair bit of. It's also anti-microbial and a bunch of other effects in our chronic infection patients, we use it all the time. Intravenous ozone therapy is another method that we use to help boost up oxygen levels in the tissues. IV ozone therapy is really quite fascinating in that it helps to stimulate antioxidant production pathways and it helps to promote oxidation of the tissue. So, we kind of get the extra oxygen to make the energy through oxidative phosphorylation, but also these elevated antioxidant levels. And then it also helps-- it has antimicrobial activities, etc. We also use hyperbaric oxygen therapy to help get more oxygen into the tissues as well. So, if patients don't like needles, then you know, or just for whatever reason want to do more than one thing, then we'll use that as well. But really, anything that's going to help get more oxygen into the tissues, that's going to directly get photons/electrons into the electron transport chain or therapies that would help to boost up nutrients. So, you know, high dos nutrient infusions, maybe NAD in certain patients, maybe IV antioxidants to help with redox balance. Those could all be additional tools outside of just oral supplements or whatnot.

[01:25:16] Scott: So, for people listening, is the IV photobiomodulation is that the Weber laser?

[01:25:22] Dr. Bryan: That's the one that we use. There are other ones as well, but I've heard some of them aren’t available anymore. But yeah, the Weber one is actually the one.

[01:25:31] Scott: We know that exercise is important for increasing the number of mitochondria. We've talked about the fact that some people can't exercise because of post-exertional malaise and so on. So, do you find that once you've implemented some of the mitochondrial support that then people are able to start exercising in order to move things even further forward?

[01:25:49] Dr. Bryan: Yes. Yeah, absolutely. Yep.

[01:25:50] Scott: Perfect. Let's talk about removing dysfunctional cells. So, if we have dysfunctional mitochondria, are there certain things we can do like intermittent fasting or autophagy support or sauna? How can we accelerate our body's ability to remove either dysfunctional mitochondria or dysfunctional cells?

[01:26:13] Dr. Bryan: Well, I think that to my understanding there isn't definitive research saying exactly how we can do that. I think that anything that would support autophagy will, by default support mitophagy as well. So, the things that you mentioned would all be probably useful things. What I think is that were, well our bodies, in general, are always striving for homeostasis. That's true on a cellular level and that's true on a mitochondrial level as well, in my opinion. And so I think that when we support the mitochondria, and we're working to try to remove obstacles from the mitochondria working well, so if there's mycotoxins, we flush those out and kill the mold if there's colonization, there's heavy metals, we flush those out. If there's other toxins, we flush those out, try to minimize exposure. Try to get the person sleeping well, so their melatonin levels are optimal, maybe load them up with some extra melatonin, get rid of the Lyme and coinfections. So, we deal with all of those things, and then I'm just a really big fan of tracking, you know, how a person's feeling. So, if a person says, you know, on this protocol, and I'm doing better and better then we’re probably doing a good enough job of if they're getting better at a fast enough rate. If they say like, this is getting better, but this is kind of plateaued said, okay, well, what are we missing here?

And if it's like, well, like, I'm getting better, this is plateaued, I'm getting really itchy like, well, maybe you need to sauna or do some dry skin brushing or, you know, some lymphagogue herbs or whatever it is. But I just really, I'm a big proponent of listening to what's happening with patients’ body, their body will basically talk to you and tell you where you need to go next. But as I said, all those tools that you mentioned are probably very helpful. And it kind of brings the question up of, well, what's the, you know, when you're feeling good, then how do you keep your mitochondria nice and happy, and it's probably all of those things. Which I think most people in my experience, present company included, if I'm feeling good as I do most of the time, and if I am doing some dry skin brushing or if I do some saunas or do this or that, like I just feel better. And I think that's my body's way of saying, “Yeah, keep it up, you know, keep up that mitophagy and the autophagy stimulation and whatnot.”

[01:26:16] Scott: I know in kind of wrapping this all up that you had a specific case that you wanted to share to kind of illustrate the role and the timing of the mitochondrial support in moving someone forward from a chronic illness. So, can you tell us a little bit about that case?

[01:26:28] Dr. Bryan: Sure. I'll give the Coles Notes version of that, Scott. So, the case that I was referring to, so it's a patient I've been working with her for probably about a year and a half. And, you know, fatigue, brain fog, other neurological symptoms, a lot of itchiness, a lot of high histamine symptoms, etc. And the long and short of it is that over the course of time, by working with antimicrobial herbal protocols, largely Stephen Buner inspired addressing mold colonization in her sinuses with essential oil nasal spray, she was on binders and detox support and what else did we do with her? I had her on adrenal support and possibly some other and it's maybe just another you know general detox support. But basically we were working with a lot-- Oh, we did LDI, we did LDA, Low Dose Immunotherapy, Low Dose Allergen therapy.

So, we worked with a lot of different things. She was very dedicated, she was coming in for intravenous infusions. We were doing the IV photobiomodulation just you know, every-- throwing most of our tools at her. And she was doing better overall, but her energy levels would really oscillate. Like sometimes she'd be like “Oh, I feel like a six” and then like the next visit it would be two months later like she's down to a four again. And also her brain fog and her memory just never changed for the whole time that I was working with her. And so finally I said, “You know what, like, you're doing pretty well overall, but why don't we put you on some comprehensive mitochondrial support. Let's, you know, wean you off like your herbs and binders and all that stuff.” Like she just seemed to have really hit a plateau overall. So, we really just emphasize the mitochondrial support, so the multi-nutrient that I mentioned, the separate powder with all the extra bells and whistles in there, maybe some creatine, I can't remember now. And within six weeks, she's like, “Wow, like my energy's like doing a lot better.” I’m like, “Oh, that's great.” Like a seven.

I said, “Awesome. Let's not do a happy dance until your next visit, because sometimes you're up and down.” Comes back in a couple months later, “Yeah, I'm like, an eight, some days a nine. My brain fog is lifting my memory’s better.” Everybody's noticing a difference like wow, like that symptom didn't ever change at any point. Energy is more consistently better and now we're probably like, eight months out from her starting that and she's still doing consistently better. And with her, you know, it's like, Okay. Well, now let's maybe focus a little bit more on like a specific mitochondrial detox protocol. Like I've got a couple of protocols to do that type of thing. Like melatonin loading protocols and different things like that. So, we're still playing with her case, but her case is a really good example of where you know, we put-- We worked with a lot of different things. She had certain things, just never really improved, certain things were oscillating and then the mitochondria support seemed to be what was missing. And everything else was-- we’d largely stripped away everything else. She wasn't on the herbs or sprays or this, that or the other things. I think her body was really missing that mitochondrial support.

[01:31:18] Scott: Beautiful. You know, that's really exciting. So, the last question that I have is the same for every guest and that is what are some of the key things that you do on a daily basis in support of your own health?

[01:31:28] Dr. Bryan: That’s my favorite question, Scott, and yeah, I do a lot of things for my health on a daily basis. So, I try to follow a very clean diet. So, kind of somewhere between like a keto and a paleo diet for the most part. I do something called targeted dry skin brushing, which there's a website called stop chasing pain that people can check out if they want to learn about that. It takes about two minutes a day to do that. I do something called controlled articular rotations to help keep my muscular-skeletal system very-- Have you heard of it before, Scott?

[01:32:02] Scott: No.

[01:32:03] Dr. Bryan: Okay. It's something that takes about two minutes a day as well, something that's easily searchable online to keep my musculoskeletal system nice and healthy. I take mitochondrial support on a daily basis because I have three kids and a busy practice and way too many side projects so that's pretty much mandatory to keep me as healthy as possible. And yeah, trying to minimize the EMFs. And yeah, doing periodic detoxes, doing the work that I love. Being married to a perfect wife is really helpful as well. And yeah, there's probably other things too Scott, but yeah, just trying to really practice what I preach and staying healthy so I can keep doing this work because I love it.

[01:32:46] Scott: Yeah, I can tell that you have a huge passion for what you're doing and for helping patients. This has been a super fun conversation. I feel like I just got the whole crash course on mitochondria, and I think it's something that again, I haven't really talked too much about in past shows. And so I think it's going to give people a lot of food for thought and then being able to carry that conversation into their next conversations with their practitioner. So, lots of good information. I appreciate your time today. Thank you so much for all the work that you do, and I look forward to talking again soon.

[01:33:14] Dr. Bryan: Thanks, Scott.

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