Summit Introduction and Mitochondria Focus

Welcome to the Reversing Chronic Crohn's and Colitis Summit. I'm your co-host, Doctor Christine Schaffner. And today, I'm delighted to introduce Dr. Hamel Patel.

And we're going to be talking all about the role of the mitochondria and health and healing, especially related to inflammatory gut issues. So welcome, Dr. Patel.

It's always an honor to interview you. Hey, great to see you, Christine. Yeah. So we're going to, you know, dive in. And as anyone who's listening, I hope it, gets this idea that when we're looking at the gut, we have to look at the body as a whole.

And we are, you know, our whole organism. And when one area of the body is injured or needs attention, we don't just look at that system, but we have to look at, you know, the energy of the body and the information and the communication in the body.

So, Doctor Patel, why don't you just share? I'm sure many, audience members know your work, but why don't you just share? your kind of like how you became really passionate in mitochondria research.

Yeah. so I did my PhD back in, from 1999 to 2002 at the Medical College of Wisconsin. The the broad topic of the lab was protecting the heart from ischemia injury.

And so this is where you deprive an organ of oxygen and nutrients, and it dies, right? It doesn't respond when you sort of resuscitate it because it's lost that that capability.

Turns out that one of the key features in creating resiliency in an organ, when it's deprived of oxygen and nutrients is preserving mitochondrial function.

So typically, organs will go into a hyper native state when they're deprived of these raw resources. when you flood the system with resources, that's typically when the injury happens.

And they call this reperfusion injury. And it's driven by this flooding of oxygen into the system. And the mitochondria are now dormant. And they haven't had to live with that oxygen.

So now they get flooded with it and they make a lot of oxidants.

Dr. Patel's Research Origins

And so the the thesis that I focused on was how opioids of all things actually create this protective paradigm where they allow the mitochondria to become resilient, where they can take in during reperfusion, more oxygen and do more with it to create less injury and more energetics.

Right. And so this is where it started. So I as a grad student, and then when I started my postdoc at UCSD, it sort of evolved into this looking at the membrane as a target for how to regulate cell biology.

And we made this early discovery that it turns out that the membrane actually sits in close proximity to mitochondria. And there's this mitochondria are symbiotic organisms, but it turns out there's an even bigger symbiosis that happens with our membrane structures and mitochondria and how they respond to stress adaptation.

And so now you have a conduit of how to get information from outside of you, right? Things that or organelles and

all these things respond to the outer environment.

And so you have drugs, chemicals, toxins that bind to stuff on the receptor. They now manipulate this microenvironment and they change the energetic profile.

And so this is what my lab's been focused on for the last 15 years is to look at that that micro relationship of membrane to mitochondria and ultimately how that regulates whole energetic systems within cells, organs and then the organism as a whole.

Yeah that's fascinating. I mean I'm no of of course about your work, but as you talk about that, I'm just thinking for people in that chronic illness world, especially if they've had mold illness, which, we've looked at mold and its relationship to, you know, energy and hypoxia and then usually down regulates probably gets the mitochondria in this hibernation state that you're talking about.

and one of the tools we use to heal people from toxic mold is using phospholipids. So using cytochrome i.v. Or, orally or even rectally for some people.

And just thinking in my, you know, brain, I'm thinking that's doing way more, you know, than I think it's doing. Because by, you know, healing that cell membrane, you're getting rid of, toxic ions lodged into that membrane or, you know, basically oxidation of, fats that are in that membrane.

And getting out of that restores not only the receptors, but also, allows the mitochondria to communicate better and, restores the mitochondria as well.

Can you help me understand that? Yeah. So there's an old, old, sort of evolutionary story around this, right. So the thought is that when organisms left water and came onto land, one of the biggest pressures that they adapt to was the oxygen environment.

and so the, the evolution's sort of answer to adapting to living in a high oxygen environment was putting cholesterol in your plasma membranes. So cholesterol actually binds oxygen.

And these membrane domains that we study are called cavalry. They are enriched in glycol single lipids, cholesterol. And they create this structure in the membrane that's very different than the rest of the membrane.

And so it's literally a platform for signaling to happen. And so this creates a sense of how efficient biology is. Right. So when we see we see instantly it doesn't take protein randomly colliding to create this effect.

It's an instantaneous thing. And so by having this molecular machine assemble in a very tight space in the membrane, now you have everything you need.

When a compound comes in to bind those receptors, all of the effector molecules, all of the things that are there. And so it turns out oxygen becomes a ready made molecule that supports energetics and other things. Right.

Your mitochondria use oxygen to make energy. And if you had an unopposed flow of oxygen into the system, mitochondria wouldn't know what to do with all of that oxygen.

So the hypothesis we have is we think that these lipid structures that are created in membranes actually bind that oxygen, and they deliver it in a very tempered way so that your system becomes hyper efficient.

Membranes, Cholesterol, and Oxygen Signaling

So one of the side effects for the first generation statins that were out there, that that lower cholesterol in your body were cataracts. And so it turns out the lens fiber in your eye has the highest density of cholesterol in your body.

And so if you lower that, this is where you have actual direct contact with oxygen. You oxidize everything. And so

you create these cataracts. And so it suggests that there is this crazy, amazing relationship between cholesterol and oxygen in this environment.

The next biggest place where you see these structures in your body or in your lung, right? Your lungs have a ton of cavalry that are super hyper enriched in cholesterol in these micro domains.

Because this is how you're exchanging oxygen from the outside environment and putting it into your body. And so we really think that there's this intimate relationship that the membrane creates with the energetic systems in the body.

and targeting membranes is probably one of the first things you want to do with chronic diseases. you know, I think mitochondria is something that everyone understands that energy use and, and sort of generation become, sort of dysmorphic in a lot of diseases.

But the primary culprit in all of this, I think, is really this membrane degradation, which then leads to this demise of mitochondria. Because if you can't keep the outside environment from the inside, you're going to create a lot of disturbances in a cell.

Yeah. No, I, I hadn't heard that before. and that way so that you know, reiterates because again, one of the treatments that I find, you know, when people are really like fried, they're nervous system, they've had a lot of toxic and exposure, congested liver.

They're just they're, you know, they're just very sensitive to like, we give them IV a central line, which we get from Switzerland. And, I can't tell you how many people respond.

And then there's this other product, called plasma allergens on the market that are a little more precise fatty acids that can kill membranes and myelin is what they say.

have you worked with that in the lab? The we haven't. So we, we do like a single lipids and cholesterol. but we the other way to sort of rebuild this structure is the protein, that was discovered in the early 90s, around the cancer diagnosis, actually, it was a substrate for SAQ, which is a kinase that activates in certain cancers.

It's called Caveolae And so it turns out this Caveolae in protein is a membrane marker protein that creates this and vegetated membrane structure where cholesterol accumulates.

And so it turns out if you just make more of this protein in the membrane, the the lipids come and reorganize into that shape. And so we're using this as a therapeutic for all kinds of neurodegenerative diseases.

We have a product currently hopefully moving into a clinical trial by the end of the year for ALS that's built around this idea of overexpressing this Caveolae and, and protein to create this new platform for all of the lipids in the signaling to come back.

so it's, would it be just to reiterate and summarize for the audience, is caviar all in a when it's high, it's batter high, it's actually therapeutic.

And remodel it in the membrane for and. That's the crazy part of biology. It's both right. It depends on the cells. So if it's if it's. And then this is we were on the cover of a journal called Molecular Pharmacology a couple of years ago, where we wrote this review article on the good and the bad of, of these Caveolae proteins.

So it's a yin yang kind of thing. So in organs where they are susceptible to oxygen deprivation. So the brain and the heart upregulate Cavehill and becomes a therapeutic right.

It drives the, the ability of the cell to survive in or in systems where you already have hyper survival. It turns out they hijack the capsule and mechanism to do this.

So cancers, certain cancers actually upregulate capsule and and so in those you want the opposite. We've so in our lab we've developed tools to not only upregulate Cav but also knock it down as well. Okay.

And so you can literally cell specific target the susceptibility in unique ways. So in cancer we can deliver RNA molecules that will knock Cavehill and out in heart and brain cells.

We can actually deliver the product that will actually upregulate the expression. Right. So you can do a concerted up and down. Do you know if clinically that this, marker is available for lab testing or is it just research based?

It's not. I mean, we've been playing around with this in in blood plasma as a surrogate for measuring global levels and things like that, and it hasn't really gone very far with it. Yeah.

Well maybe that's your next test. And then we'll talk about their tests that. Yeah, Patel has developed. So okay. So, so some of our patients unhurried, like hibernation or the community rather, hibernation of mitochondria.

Caveolae, Therapeutic Remodeling, and Disease Context

And they thought immediately. So danger response is that is that, something else that's happening, in the state as well? Possibly. I mean, the the idea when your system goes into stress is it typically shuts down, right?

It doesn't have the resources it needs to do its normal activity. And so the, the cell responses to shut that system down and keep it dormant. So it's not creating oxygen pools and other things.

So mitochondria do two things very well. Right. The energetically feeble reaction is that they make free radicals which then cause injury to lipids, proteins, DNA, RNA, those sorts of things.

The less favorable reaction is that they actually couple in a, in a tight way, electron transport across the electron transport chain to create this hydrogen gradient, which then the big payoff at the end is making a ton of ATP relative to glycolysis and other processes that go on.

and so it has to control this in a unique way, and it needs oxygen to move and create this efficiency. So if oxygen is deprived these have to go into a stasis like sort of quality.

One of the things that that I've read a lot of the literature on, when I was looking at ways to protect the heart from a schematic injury, is the hibernation world, right?

There are organisms that go into a cave, and there's a trigger that causes their physiology to basically shut down to the point where they seem like they're dead and they live in that dormant state for months, and then they wake up as if nothing happened.

Yeah. and so that we think is one of the dynamic features of figuring this out. Right? So if we could figure out how these organisms basically go into stasis and ultimately how they wake up and their physiology reanimates to the point where it's completely homeostatic, we could answer a lot of questions about how to adapt living organisms that are non hibernating to this process, as well.

And I think the key to that again is two things, right? One observation that has been made is bears and other organisms. And I don't know who goes in to bleed them right before I go. And yeah that's what I was I was like.

Wow. Right. And so what they've noticed is when you look at their blood plasma, it moves from like a yellow color, which we typically see if we were to take yours and mine to this opaque milky white plasma.

So that would suggest that there's a lot of lipids right before an organism goes into this hibernation induction trigger. So this is where these lipids come back. Right.

So there's this notion that that there is something that the lipids do to put this into a stasis kind of phenotype. and

then the other aspect is mitochondria.

Right. The mitochondria create this energetic pool. They shut down and then they reanimate. And so to understand the connection between lipids and mitochondria could be a core feature of this, one of the other unique ways we've started looking at this.

And we were collaborating with a group on campus. So there's a couple of blood borne cancers that are very responsive to chemotherapeutic agents. These nib compounds essentially knock out that leukemia and it's gone.

so then the idea is, well, then it's gone so I can stop taking the drug. And so when people stop taking the drug, it repopulate. And so we were trying to figure out, well, what is the cause of that repopulation and why isn't it completely knocked out?

it turns out that these compounds work because they antagonize mitochondria in the peripheral cell. But the stem cell environment where this cancer propagates from is protected in this lipid rich environment.

So you never get access to this space for that drug to knock it out. So as soon as you stop taking the drug, that lipid rich environment which is kept, these progenitor cells essentially repopulate.

And so the target for a cancer like that, to really create a once and done therapeutic is to figure out how to create this loss of cell in this lipid rich environment that then supports that survival.

Right. And so this is why these critical cells, like stem cells sit in bone marrow is because they're surrounded by lipids, which gives them that protection that you don't have in other cell types right there in this rich environment that gives you fuel resiliency, resource and likely this hyper native component.

So I guess we in a sense hibernate as well, right? Certain cells in our bodies sit dormant. And these are these stem cells. Yeah. Yeah I know that's fascinating.

And you know really laying the foundation for, you know, this really dynamic, you know, role of the mitochondria and that's, you know, very complex. And after Patel is here to really, help us to honor that and also, you know, make a clinical gateway for you to understand kind of the role of your mitochondria, especially if you're in a state of inflammation and chronic illness.

and so it kind of leading to that Doctor Patel with, you know, thinking about like, chronic inflammation states. Right. states that people have, you know, we're talking about gut health.

So, you know, inflammation in their colon or their whole digestive tract. And, you know, usually there's a trigger kind of the, you know, stance we're taking into some that there's a it's not just purely genetic, but there is a maybe a genetic propensity.

And then there is a trigger either environmentally or infection borne or trauma event.

Hibernation, Stem Cells, and Chronic Inflammation

So when you think about, you know, inflammation, especially pertaining to the gut, you know, what have you learned about the mitochondria that you haven't shared?

And then you shared some really awesome. Yeah. And, I mean, in my sort of limited understanding of Crohn's, I think it's mostly Western developed countries that see large elements of Crohn's.

I think Asia and Africa have very little diagnosis of Crohn's. So there's something about the European population that then migrate into the West that has this. Right.

So I suspect that there is some genetic component to this and some sort of tracking with environmental exposure

as well. mitochondria become interesting in this tracking, right?

One, I gave this lecture recently at a biohacking conference where you can actually track migration patterns of humans based on mitochondrial DNA because you inherit them from your mother.

So all of your mitochondria come from the egg. The sperm uses all the mitochondria to sort of basically swim upriver. And then those mitochondria exhausted because they're overworked, they tend to create a lot of mutations.

So Biology's figured out a way to not take those into the new organism. And so now that you have the genetic lineage of the mother, you can actually trace patterns of how people came out of Africa and went to Europe and divergence into Asia.

So be interesting to look at the mitochondrial etiology in that population. And I'm sure someone's done it with Crohn's and inflammatory diseases. and so I bet you there's a tracking pattern along with that, with genetic mutations that exist in the the mitochondrial versus the genome component, and so that they become sort of this interesting conduit for, for identifying disease etiology in terms of the gut one of the things we've been thinking about is the interplay between your gut microbiome and your mitochondria.

Right. one of the big questions is will, how does the gut communicate with the rest of the body? the big idea is that everyone now fixates on the gut as the true mind.

Right. And that this is what's now regulating the gut to brain axis. The gut deliver gut the heart. I mean, it interacts with all of your other organs in the body.

I have this sort of idea that the way it does this is it communicates with what it knows. And gut bacteria, if they're going to communicate with the known, should communicate with your mitochondria because they're old primordial bacteria that get incorporated into our cells.

And so I think that's the communication pattern that we need to understand is how does the gut regulate mitochondrial behavior in the cell? Most people think of mitochondria as being just the powerhouse of the cell, which is true.

They make a lot of energy and they keep this balance. The thing that's emerging now is that they do so many other things in a in a cell and in an organ.

They're involved in dynamic shifts. They're involved in signaling, they're involved in buffering. they become a toxin sink as well. the other thing that most people don't think about is this is mitochondria work.

Cholesterol is synthesized in your body. This is the building block for all the hormones you have as well. Right? So the hormonal balance likely comes from mitochondria pools and stores as well.

So if you have dysfunctional mitochondria this could explain why you have all these imbalances in hormonal dynamics as well. All of this then I think ultimately feeds into this inflammatory phenotype.

I think where this has been pushed and new hypotheses, these have really been developed is long Covid, right. There's a paper that came out, from, I believe, a European group that suggested that all of it has to do with mitochondrial dysfunction.

They took muscle biopsies and did some phenotypes. And we're working with a couple groups on campus here where we suggest that the dysfunction actually starts in immune cells with their mitochondria.

And this is what derails the phenotype. It creates this chronic fatigue, brain fog, pots and all these other things that you see in long-covid kinds of things.

And I think there is an inflammatory component to all of that, how that all integrates and connects, I don't know, but

I think mitochondria are a central part of all of this.

Yeah. No, I, I could absolutely agree. I mean, I've always known about mitochondria and I think I'm always treating them indirectly. But when, long Covid came on the blog, it was like very important.

like up my knowledge and strategies to, repair and support people to, help my dog improve mitochondrial function. And that's really getting a methylene blues and then, then the melatonin.

So the cocaine got very popular during this time. And I think for the good I think, you know, it's the the leakiness aspect as well. And I think mitochondria are really drivers in maintaining that that barrier functionality as well.

Yeah I hadn't thought about that because of the if there's just biotic, flora in the gut and that's affecting regulation on a, my own level that can affect cellular.

The no that's a really great point. Yeah. I want to, circle back to something. You said that, mitochondria are, toxin sinks. I think you saw it and said, like, they're, they they kind of absorb, intracellular toxicity. Can you just.

Yeah. So, you know, I, I went to grad school at a time when med school students and grad school students were together. So I met my wife, and I know each other forever.

And so we were she was in medical school. I was in grad school. So we took the first two years of courses together. Oh yeah. And the bane of my existence was physiology.

And so I still remember to this day one question I got wrong on a physiology in which which ion does the cell regulate? At the tightest level, it turns out it's calcium.

So the level of calcium outside versus inside the cell is the largest gradient you will see in the system. Mitochondria are sinks for calcium, right. They suck all that calcium out of the system.

And so they create this buffer for when your mitochondria dysfunctional. That calcium is now available to the rest of the cell. So all the things that calcium activates become hyper activated and do untoward kinds of things.

And we we've shown studies where you use nanoparticles and things like that as a way to tag and target specific things.

Mitochondria, Gut Microbiome, and Toxicity

All of them tend to start accumulating in the mitochondria, the really sticky organelles. And so they'll suck all these things out of the regular cell environment.

Right. And they become this hyper sink for these toxic kinds of things, which then creates two things, right? They become this sink that then allows you to survive and live because those toxins get eliminated.

But then ultimately, when they accumulate to a too high of a level, they start impacting mitochondrial function. So then they feed back into the system to then create disarray.

Wow. Yeah, I, I, I, I thought that could be the case, but the way that you explained it and then with the calcium, I mean, there's some models, like for some modern day toxicants, flooding, you know, cells with more calcium, the more and more, you know, there's a model of like, EMF exposure and why that is so problematic.

just or, you know, we're trying to create many ideas and models for that. And. Yeah. Yeah. Marty Parham, popularized the idea of increasing intracellular calcium.

So that indirectly is of course affecting the mitochondria. Yeah. Yeah. So we're doing some studies around this, Beatrice Golem, who is here at UCSD and at the Salk, where she's very interested in EMF toxicity as well as

environmental toxin exposures and things like that.

So you're starting to look at mitochondrial phenotypes in this. And we're really using our new technologies around mitochondrial testing to do this at scale.

Right. Which is yeah, allows us to do a lot of yeah. That gives me such great hope that, you know, I, a researcher is starting to look at that because I think that's honestly why people are so sick today.

is that this ever increasing, environmental toxic exposure is happening over a year. And so then in the backdrop of that, our bodies, like our immune systems, get impacted.

No Condra. Right. We can have more models. hopefully that site change. And sadly, I mean, we have models where we can study this, right? So the the place that Beatrice is doing this primary study right now is in East Palestine.

That chemical spill. And so we're deploying our Cod technology where now we can get samples from people at different sites. We're also testing, animals that were exposed at those sites as well.

Wow. And so now you can look at really the human versus their companion, like dogs and things like that, and their companions and really look to see if there's a relationship between how close you are to an exposure site, time of exposure and all these other things that play into the epidemiology of all of that.

Yeah, yeah, yeah, no tragic incident, of course, but I'm glad people are studying the impact, of course. And that is always a clue. Sometimes some people have a sick animal.

I find that they're kind of the canary before the person within something that you're studying. Both. So a big reason, why women to have you, the summer, not only for all the awesome information you just shared, but, one of the things that I think is going to change, really how we approach, mitochondrial dysfunction in the chronic illness, community is, you know, looking at, really the mitochondria, through this lens that you created, this test called the Me screen.

So why don't you just walk, straight through, kind of like where mitochondrial testing was and then kind of this whole and, and maybe you've created. Yep. So the gold standard has always been direct muscle biopsy testing.

And so we do a lot of these on campus. you go in, to the research study and subject comes in in the morning. their leg is numbed with the local anesthetic.

You take a gigantic needle, stick it in that leg, pull it out and we get about 200mg of muscle tissue that comes out. We can then do lots of phenotyping of that muscle.

So we have a system called the Or borrows, which allows us to look at direct mitochondrial function in that tissue. You do have some anesthetic effects, but the controls and everyone goes through the same thing. So you control for all of that.

it typically takes us about six hours to analyze in pretty fine detail mitochondrial function. In this we have a pretty well-equipped lab. So it takes us about six hours.

And we can look at three samples a day. So there's really no way to scale this outside of research studies that we're doing. So we can do, you know, a 200 person human clinical trial over 3 to 4 years pretty easily.

And it typically you'll get a grant for about 4 or $5 million to do that. So there's no way to get this to the consumer. So the next iteration of this, we were involved in the NASA twin study, where the twin went up into space for a year and his counterpart stayed on the ground.

We had access to blood samples before flight, during flight, after flight. And NASA's challenge was we can only

give you plasma without cells, right? So platelet free plasma, could you predict time and organ of dysfunction?

during flight and where things will go sort of south. And so the thought I came up with is plasma has your metabolites, your exosomes, your proteins, everything, your cells are being bathed in is captured in that that micro capture of that blood.

we can capture this fairly easily at scale. I mean, we were able to do it in space for a year. Essentially every three months, Elon Musk would send a rocket up and send the samples back down.

so the idea was to basically do an adoptive transfer experiment. So we capture your environment and your exposure in this sample, we transfer that onto a plate that basically has human on a plate.

So we we can buy cells from a commercial sources that represent every organ of the body. Yeah. Those cells now were exposed to that plasma environment.

And now we can see how their mitochondrial function changes in real time on the systems that we're assessing. Right. So you can basically look at muscle dysfunction immune dysfunction neuro dysfunction cardiac.

We have all these cells that manipulate this. So we were able to predict unique time points where you'd see muscle endothelium immune dysfunction. And so it became a way to to really see could we commercialize this to the rest of the world.

The fact that you had to do the blood collection still creates a barrier, right? You'd have to go to quest or Lab core or something. It have to be processed in a certain way.

So how do we get rid of that to then really scale to the next level? So we came up with these cards that lots of companies like every well and other companies use.

Ours is a bilayer system. So you drop a couple drops of blood in this first window. the bilayer wicks the serum into the second window. The car dries.

We've shown stability of this card to two months at room temperature. So this gives us the ability to literally get a sample from anywhere in the world, that you can capture fairly easily.

six drops of blood match. Right. so once this arrives at our facilities, we punch that serum out. we reanimate the serum in our proprietary buffer, and then we're off to the races.

Mitochondrial Testing and the MeScreen Platform

We essentially do the NASA type assay, but now with the serum that we've archived on this card, anywhere in the world, any person who can poke a finger and give us a blood sample, we can give you an assessment of your mitochondria.

And so basically now we do an adoptive transfer experiment that serum has your metabolites exosomes, proteins. We transfer that onto a muscle cell. And we see how that cell's mitochondria behave in unique ways.

We can look at a mitochondrial stress response. We can look at homeostatic. What type of energy systems are you driving, how much mitochondria, how much glycolysis.

We can look at reactive oxygen species generation. And then importantly, what we talked about as the signaling component is we can now start looking at the dynamics of your mitochondrial network.

Because we created a cell line where all the mitochondria glow green. And we can see how your environment now shapes that that network dynamics. And so we really give you with a few drops of blood, a real assessment of your function structure and dynamics, which then gives you some deep insights into how to move that number to the

next level.

we've seen some interesting results that have popped out. and we've really been able to, to show people this is where your mitochondrial status is for providers.

It gives them a way to track what things that they're doing and if they're making a benefit in their mitochondria functionality, and if they're not, it gives us some ways to tweak that in a unique way.

And we can give you some suggestions of where to start. Right. And in terms of my biology and how I think about it, it's what you started off with is you got to fix membranes first.

For most people. After that, it's to activate these, my top AG autophagy pathways to clear out all the death and decay in the cell. And then finally, I think you want to go to antioxidant on board.

And if you can manage those three elements, most people will do well with their mitochondrial functionality. But there's a thousand ways to achieve that.

And I think it works differently for everyone. So it's to find that balance of what works for everyone. Yeah. No that's incredible. And it will have of course, you know, you share, you know, all the ways to get this really innovative test.

It's one of the tests I've been most excited about in the last few years. there's always specialty labs coming up. And, you know, while they're helpful, they don't always have a clinical application.

So this is, really exciting to me. what kind of. I mean, of course, all hypotheses, unless you've seen this data, with people in this community with gut inflammation, inflammatory bowel, do you have any, like, hypotheses of where their mitochondria will be, where that will be?

most likely break down. Yeah. So I think with those individuals, it tends to be something that starts with their gut microbiome. Right? I think really it's a dysfunction of the gut microbiome that then propagates these bacteria that are opportunistic and create an inflammatory environment.

This then feeds into the metabolite pool that's circulating around your body, which then basically allows that phenotype to migrate away from the gut to the rest of the body and creates sort of this global phenotype.

So in that case, I think there's an element of understanding that energetic dysfunction that you see, which then really gives you some insights into how to manage the gut microbiome.

and I think the best way to couple our test is to look at gut microbiome information as well, and then to look at the metabolite pools that you're creating.

And I think having an integration of those three really gives you a unique window into your overall health that ties to this microorganisms that live in our gut, and how the micro organisms that live in our cells interact globally.

Yeah. One of the things that we're like, really, attuned to clinically and the treatment of chronic illnesses and, gut just biotic, you know, patients, we look at bio toxin, secretion that happens especially when people are taking like an antifungal or acidic antibiotic.

And a lot of our strategies are to mop up the bio toxins because usually people feel better, you know, that treatment. So, I'm curious of one of the drivers to the mitochondrial kind of dysfunction is, bio toxins, you know, in that metabolite pool.

So, yeah, I mean. Possibly possibly I mean, one of the things we do see in a lot of individuals that take our test, the first thing we run is a baseline respiration.

And so it's this muscle cell that's cooking away. And we add that environment onto this cell. In an ideal individual

nothing should happen. Right. That cell shouldn't be impacted in any way.

But we see in a lot of individuals is they actually have a decrease in baseline respiration or they have an increase in baseline respiration. So with this suggests is that there's something inside their their serum that's either shutting mitochondria down.

So a possible toxin that's creating this non resiliency pool. Or there's a toxin that's hyper activating their mitochondria. Because you see this increase in basal respiration.

Typically in those individuals you'll find some dysfunction downstream in the other parameters we run in our test as well. The ones that tend to not move and have the sort of the baseline that stays average.

They tend to look pretty well when you look at other parameters. so that would suggest that there is some activating or deactivating toxin that's sitting there.

Yeah, yeah. I know with Clostridium difficile, or Clostridium special species, it's like p cross, all that is very inflammatory. And so I'm sure there's, something, you know, there's some contribution probably not the full story, but, but yeah, no, I, I think it's a really brilliant idea and model to go after because when you fix, you know, the mitochondria.

you know, we talk a lot about regulation. And by regulatory medicine, we think about the autonomic nervous system and the fascia. But really, the mitochondria are regulatory, you know, organelles at the root of it because, you know, they're you know, not only energy, oxygen, all of that, but the whole apoptosis, senescent cells, you know, all of that, which is regulation in my mind.

So, yeah, it opens you keep expanding my ideas and model. So thank you. so, Dr. Patel, anything before we share, like how to get this test, how to connect with your company, any other, URLs that you want to share with this?

population right now? Yeah. I mean, I think, you know, people ask a lot about who should get tested for this. I mean, if if you're living and breathing, you should have a minute.

You should know what your mitochondrial number is. Right. And I think that our test allows you to get that baseline. And if you're well to it every six months to a year, just to make sure you're tracking along.

And if you're not, I think knowing where you sit really is going to give you deep insights into where to move.

Who Should Test and How to Access MeScreen

and it's all about energy, right? People want to live well, resilient lives. And one of the things that I've seen in this, in the spaces that we've started interacting with wellness, is most people are trying to hack health and longevity through the nucleus.

The nucleus is not where the answer is, right. This is an organelle that's designed to kill us. genes are selfish. What they want to do is move to the next generation, and they're designed to get rid of that first generation.

So you don't take resources away from your children. and this happens evolutionary in every organism. And so the way to hack health and longevity is something outside of the nucleus.

And it really comes down to your membrane because this is what you have to keep active for the rest of your life outside too, inside separation. And then the energy balance, the thing that ties the energy to all of that.

Right. So it's really membrane mitochondrial health. I think that's going to give you resiliency against chronic diseases and to live a long, healthy lifestyle overall.

That's resilient. Yeah. Great. Great great points. why don't you share you know how people can get their own

movie screen. And if they want to get a consultation as well, if they don't have a a doctor who's doing this test yet.

Yeah. So ideally they come to someone like you who is part of our larger network. and, and, you know, it's available through providers. we do have a direct to consumer model that we're working on right now.

so me screen.com is where you can land on and find information about the test. the model will be that, that, you know, if you go through the direct to consumer, we'll give you a consultation about how to interpret that test result.

And the goal really is to give people an insight into their mitochondria. The new landing page that we're developing, we'll have an educational arm as well.

So not only will you be able to see all your numbers, but you get to learn about mitochondria and why they're so important and all the different tests, features that we have and what basal respiration means versus your mitochondrial potential versus your rest level.

And I think it's to educate that population. The other thing that we want to drive in this is I assume consumers are going to be interested in this because there's a buzz around mitochondria right now, which is a great thing for people to know what they want.

But the, the, the way we built this company was really going after the providers. First, we wanted to get a network of providers across the US that were starting to engage in to teach them about this.

So then when this direct to consumer population comes, we can funnel them to people who really understand how to look at whole health, which mitochondria is a big part of, but then really give them conduits to change the health in a dramatic kind of way.

And so there's lots of ways to do this. So providers or go to mescreen.com Awesome. as I mentioned this is a test I'm very excited about. And I've used personally as well as clinically.

And I think there's just going to be so much more information to really, I'm always saying in my process that we're like really trying to get the most elegant path for healing for people who've been suffering for, for so long.

And I think this is going to be a really big piece of that puzzle. So I want to just thank you so much for your contribution and really your, work in creating this test and also furthering this knowledge.

And it's always so lovely to speak with you. Have a great talk with you. So thank you. All right. All right.