[00:00:00] **Dr Mike T Nelson:** Welcome back to the Flex a Diet podcast. I'm your host, Dr. Mike T. Nelson. On this podcast, we talk about all things to improve body composition, add muscle, improve your longevity, all done in a flexible framework without destroying your health in the process. Today on the podcast, I've got my friend Kristi, and we are doing a deep dive into the concept of metabolic flexibility.

Which I'm sure you are all probably pretty familiar with and if you want more information on a metabolic flexibility including the Complete system of how to use it for primarily nutrition and recovery make sure to check out the flex diet Certification it'll be opening most It's likely for the last time this year.

I'm not sure if I'll have it open again towards the very end of this year, but next time it is going to be open is starting next Monday, June 17th, 2024. We'll be open for one week, but you can still get on the wait list for all the information. Go to flex diet. com. And I will send you all the information coming up this week.

I've got some super cool bonus items some fast action bonus items only be open for a little while once it opens. So go check out flex diet. com for all of the information. And for this round again, I'm not sure how long I'll keep doing this, but you'll actually get my private email when you sign up and register.

If you literally have any questions about the material or you get stuck on a client trying to apply it, you can literally email me and I will get back to you within 24 to 48 hours. If you don't understand the concept or there's something that didn't make sense, I will do whatever I can to personally help you out.

And those emails go directly to me. So check out flexdiet. com. It is based on the concept of. metabolic flexibility and flexible dieting. So you'll learn all about the concept. You'll learn the eight different interventions and the details involved with each one. Everything from protein, fats, carbohydrates, micronutrition, exercise, NEAT, sleep, fasting, and much more.

And then you'll have the five explicit action items for each one of the eight intervention areas. And this will tell you exactly what to do for yourself or for your clients. Of course, we explain the system of how to pick which one of those five is going to be best. So go to flexdiet. com And as I mentioned with Kristi here, she is a PhD student currently a PhD candidate in muscle physiology.

She is studying the effects of high intensity interval training, mitochondria, glucose, lactate, Metabolic flexibility and much more at Queens University. She's also a CrossFit level one certified. So we do a deep dive here. We do get pretty geeky about all the different aspects of metabolic flexibility.

And what I love about talking to students who are actively doing research in this area is that you get the latest and greatest of everything that's updated. Metabolic flexibility is probably one of just a handful of topics that I do stay up to date on, I think I've read, multiple read, almost every research study in that area.

But, when you work in a lab, there's new stuff coming out. You are, have access to some data. Other people in the lab are constantly publishing stuff. So you have access to a lot of data that may be submitted for publication, may not be public yet. And that's the one thing I do miss about working in the labs.

I don't miss Getting up at 4 30 in the morning to set up all the metabolic hearts and do all the testing, but I do miss the environment of having access to the latest information and literally being on the cutting edge a lot of times before even that literature is published. So enjoy this wide ranging study and talk about metabolic flexibility with Kristi.

Welcome to the podcast, Kristi. How are you?

[00:04:21] **Kristi Storoschuk:** I'm good. Thanks for having me, Mike. I'm excited to be here.

[00:04:24] **Dr Mike T Nelson:** Yeah, this'll be fun. I know you're doing some super cool research, on metabolic flexibility, and we'll get into some of the subtopics and other things you're looking at, but, How did you get interested in the topic of metabolic flexibility?

And we'll use that as a way to getting into what you're actually working on there, which is loosely tied to that.

[00:04:44] **Kristi Storoschuk:** Yeah, let's get into it. All right. Backstory. So, we'll start when I was a little 13 year old working in my parent's house. It's a health

food store

I remember like reading books because we sold books as well in our health food store.

So my grandparents own this store and I remember reading books and trying to memorize like what every vitamin did in the body. And I was just very interested in nutrition and health and like the natural health world. And I was exposed to that at a very young age. And so that was like my entire.

Upbringing for as long as I remember was being in that natural health world. I always thought I was going to be a naturopathic doctor. I did my undergrad and bought me thinking, Oh, I'm going to plant because of plant medicine. And I was really into, yeah, just natural supplements. Yeah.

[00:05:35] **Dr Mike T Nelson:** When you say plant medicine cures from plants, not like the crazy stuff where you see shit?

[00:05:40] **Kristi Storoschuk:** Both. Both? Okay, all right. When I was in my undergrad, I remember having a call with Wade Davis, who Oh, no way! You did a call with Wade Davis? Yeah, and he wasn't, he didn't have a running lab at the time, he was just Okay. He was just teaching at UBC but I I, I guess I was like, I don't know, 16 or 17, looking for grad school opportunities after trying to prepare for when I was going to graduate.

So yeah, I was probably closer to 17, 18. And yeah, I had a call with him and to this day. He seems like a character. Never met him.

[00:06:12] **Dr Mike T Nelson:** I only hear stories secondhand.

[00:06:15] **Kristi Storoschuk:** So no, plant medicine across the board, I was very interested in and I wanted to potentially study plant medicine, but also I just went into it thinking cause all the supplements that I was exposed to were like compounds from plants.

And I remember sitting in my plant physiology and plant metabolism. Click. Courses being like, they were talking about sulforaphane and like myrosinase and all these things and I was like, oh, I know that that's in Brussels sprouts or mustard seeds do this. And I would associate everything with food or supplements.

And meanwhile, people were probably like, what is this course? Girl, like why is she asking me these questions? So anyways, that was my entire like undergrad young adulthood experience and then my sister is a naturopathic

doctor Watched her go through the process of going through naturopathic college and it I love it for her.

She suits the profession very well, but I was turned off by watching her experience and then I, but I really wanted to get a doctorate. So that's what led me down the alternative, which was, well, not alternative. One alternative being research and getting my PhD. So, in my last year of my undergrad, I was reaching out to, different professors around in the U, in the States specifically because in Canada you have to do a master's before you go into a PhD.

And I know that's normal in the States too, but it's also normal to go straight into a PhD.

[00:07:37] **Dr Mike T Nelson:** Yeah. U. S. you can do both. You can do either one, which to me is a little weird, but yeah.

[00:07:42] **Kristi Storoschuk:** Yeah. And at the time I was really interested in the ketogenic diet. So I listened to Dom D'Agostino on Joe Rogan and I thought that the ketogenic diet was really interesting and I had.

I, my entire also young adulthood was trying different diets. I've been vegan, hardcore, paleo now being in this world I've done keto, hardcore, carnivore, etc. I've always loved like experimenting with diets and trying to find what works. Diet suits me best. But so the ketogenic diet though, I was really drawn to because I really liked biochemistry.

I really liked genetics and like molecular regulation of gene expression. And I thought that beta hydroxybutyrate was really cool because it had all these signaling roles in my body. And so I was reaching out to everyone Volek, Dr. D'Agostino, et cetera looking for a research position after my undergrad, and Dom happened to respond to me, he wasn't taking on new students at the time but He gave me the option of working for him, and he's I'm trying to build my company, are you interested in working in science communication?

And so, I took on that role, I was traveling the world at the time with my boyfriend, and so we were just living out of a bag, I had this opportunity to write for Dom's website, and I introduced Oh, I didn't know you

[00:08:57] **Dr Mike T Nelson:** did that.

[00:08:58] **Kristi Storoschuk:** Yeah, so that's what I did straight out of undergrad. I started working with Dom and eventually Dom's like the

[00:09:04] **Dr Mike T Nelson:** nicest person too.

Him and his wife are both just, not only are they both ridiculously smart and super humble and they're like, you talk to them and they're like, oh yeah, the one time we did this experiment or this or that, you're like, what the fuck, man,

[00:09:16] **Kristi Storoschuk:** I literally, I

could not express how grateful and lucky I am to have fallen under Dom's wing for so long.

He is quite literally the nicest person I think I've ever met Oh, he's so nice.

[00:09:29] **Dr Mike T Nelson:** And everything that comes out of his mouth is just so humble and you're like, dude, That's insane. Ah yeah.

[00:09:34] **Kristi Storoschuk:** I know. And yeah, he's just the kindest person, yeah, ever. And I'm very lucky that he saw something in me and let me work for him.

And that eventually led to me, my grandparents happened to have a place in Florida that was near Tampa. So I would live there for months at a time, go into the lab, made face with the lab. Like now I feel like they're I feel like I've made my way into their little family that I now see very rarely, but at the time it was I incorporated myself as this pseudo student in their lab, and that was really fun.

Get, I got a lot of experience and just, it was my entry into academia. And I rode that wave for a very long time, for maybe too long, it was about three and a half to four years I was working with Tom and just working as in different science communication like positions. I worked for the Zero Fasting app, which is now Zero Longevity app, worked for them for a while.

And then I was like, okay, I got to go back to school. I've been, that was my initial, I really wanted to do my PhD and I've just been putting it off. And so I, I live in Ontario and Queens, I now go to Queens University. That was the closest school to me. They had an exercise physiology lab that looks at mitochondrial adaptations to exercise.

And being in the keto world, I was very, exposed to the importance of mitochondrial function and metabolic health and the role that mitochondria play in our metabolic health. And so I was interested in mitochondria and studying

mitochondria and this lab just fit the bill for me. And so I pitched to the supervisor that eventually took me on.

I wanted to look at ketone metabolism, see if there's any signaling in skeletal muscle related to any sort of mitochondrial biogenic responses. That. Has not come to fruition and will not come to fruition, but that was the initial like the idea of what I was going to study and the

[00:11:31] **Dr Mike T Nelson:** study never happened.

Correct?

[00:11:33] **Kristi Storoschuk:** Right. We never designed to study around ketones. But I was also just interested in fasting and I just thought I thought it was just a well accepted idea that Fasting is an energy crisis, so we need to adapt to that energy crisis, and one way that we adapt to energy deficits is By upregulating the signaling, signaling responses that are related and associated with the initiation of mitochondrial biogenesis.

So there was like a good rationale or theory that fasting could induce mitochondrial biogenesis and this would improve metabolic health because we know that We mitochondria are so important for our metabolic health and specifically in skeletal muscle because that's our most metabolically Active tissue and where we're going to be metabolizing most of our fuels So if we have good mitochondria big powerhouses where the energy powerhouse of our cell Then we should be able to metabolize the fuels that we eat efficiently and effectively.

And when that is deranged when we have poor mitochondrial function, low mitochondrial content that's associated with insulin resistance, metabolic syndrome and chronic disease. So my, And my next idea after ketones was let's design a fasting study and see if we get the activation of energy sensing pathways related to AMPK and PGC1 alpha.

So one energy, like an energy crisis is sensed through the protein kinase AMPK, which I'm sure you're very familiar with. But the act, like an increased activity of AMPK one of the downstream targets Is this protein called PGC one alpha and when and PGC one alpha is often referred to as the master regulator of mitochondrial biogenesis, which is currently challenged and not something we necessarily accept in our lab, but it is really important for mitochondrial biogenesis and it is.

definitely associated with an upregulation of mitochondrial content, mitochondrial proteins. There's just a lot more to the story and a lot more players and many novel regulators of mitochondrial biogenesis that are not, that will one day probably see the spotlight. But at the moment, PGC1 alpha gets most of the attention.

So if fasting was an energetic stress in skeletal muscle, then we should see an upregulation of AMPK and PGC1 alpha, and then. Hopefully, an upregulation of mitochondrial proteins, the synthesis of these proteins that eventually get incorporated and expand the mitochondrial reticulum, which is the definition of mitochondrial biogenesis.

But my supervisor, I was apparently ignorant to his past research because they had already done And who was your advisor, just so people know?

Dr. Brendan Gerd.

[00:14:19] **Dr Mike T Nelson:** Okay.

[00:14:21] **Kristi Storoschuk:** And so they'd already done fasting studies because So, Brendan, Dr. Gerd, he wanted to use fasting as a model for activating these pathways because in mice and in rodents, it's very clear that fasting upregulates these pathways associated with mitochondrial biogenesis and and so he wasn't even looking at fasting as an intervention the way I viewed fasting because I was thinking like, oh, fasting increases these pathways increases health.

He was just thinking like, how do we mimic what's happening in mice and to be able to study the pathways involved in mitochondrial biogenesis? Yeah, what's the stimulus we can push this little variable around with? Right, and then

study those proteins and figure out those pathways. And They, so they ran 48 hour fasting studies and showed that there was no increase in AMPK, no increase in PGC 1 alpha.

This is in

[00:15:17] **Dr Mike T Nelson:** humans, correct?

[00:15:17] **Kristi Storoschuk:** In humans, in skeletal muscle. And then they thought that, oh, maybe we missed the time point. Maybe the activation is earlier. So they did another fasting study where they did eight hours of fasting

which is short term for someone like, You and I that think about fasting eight hours doesn't seem like much also saw no activation, but then they increased energy expenditure above that by doing our arm ergo ergometry, ergo, er, yeah, ergometry, yeah

[00:15:47] **Speaker 3:** arm peddling yeah, arm

[00:15:48] **Kristi Storoschuk:** peddling to increase energy expenditure without activating pathways in the leg, because that's where the biopsy is.

The biopsy was taken from the vastus lateralis. So they're

[00:16:00] **Dr Mike T Nelson:** taking a chunk of tissue out of the leg muscles. They did not want to use exercise in the leg muscle because it would change a whole bunch of other stuff they're looking at. But they wanted to use some something that's going to drain energy. So that's why they had them do the upper body kind of pedaling thing.

[00:16:16] **Kristi Storoschuk:** Exactly.

[00:16:17] **Dr Mike T Nelson:** Got it.

[00:16:18] **Kristi Storoschuk:** So even increasing energy expenditure didn't result in any changes in any of those signaling pathways that I mentioned in the leg. So basically is that Dr. Gerd's conclusion is that, okay, in humans, these pathways are not activated the way they are in animals. mice and in rats that is very well cited.

So in the nutrition and the metabolism literature, you'll often see figures showing exercise and caloric restriction leading to the same pathways. So, leading to the activation of AMPK and being this way to induce energetic stress intracellularly. And that's what we want. So we mitochondria adapt to energetic stress because We've exceeded its capacity.

So once the way that we resynthesize ATP is by using ADP and AMP and our mitochondria add that phosphate group to it and make ATP but once our ability, our mitochondria's Capacity to re synthesize ATP beyond what's being used by the muscle cell that's when we adapt because now our, we have this energetic stress, we get the accumulation of AMP and ADP we get declines in ATP and then this activates those pathways But the thing is that exercise and so increasing energy expenditure.

Through something like exercise is not the same as reducing energy intake. So you wouldn't expect Fasting or caloric restriction to lead the activation of the same pathways that exercise do does even though like it You can look in really well established, really high impact journals, and it's often cited that caloric restriction and fasting activate AMPK, and this will lead to the upregulation of longevity genes and longevity proteins and

[00:18:15] **Dr Mike T Nelson:** things

[00:18:15] **Kristi Storoschuk:** that are associated with metabolic health.

[00:18:17] **Dr Mike T Nelson:** And I'm thinking on stuff, I'm probably guilty of saying that also. So side question is, so for AMPK, is I went to a seminar, it was experimental biology once, and the presenter was like, AMPK is the energy sensor of the cell. And this guy got up out of the audience and was arguing with them. And I'm like, well, that's weird.

And then later I was like, who was that guy? And it was Paul Greenhoff. I was like, oh, the guy who's actually studied a lot of this stuff. And I don't remember exactly what he was saying, but I think his argument was something like, It's more complicated than that. That's an oversimplification. And again, this is at a Uber, egghead meeting of all egghead meetings.

So you're saying that AMPK, is it true that it is still an energy sensor, but maybe it's not as sensitive to periods of low incoming energy, meaning like a period of fasting versus a period of. Increasing energy expenditure with something that's more of a caloric drain, like exercise, like those are, it's a three part question.

[00:19:25] **Kristi Storoschuk:** Yeah. So AMPK is absolutely still the energy sensor in our cells. It's just that Without increasing energy expenditure. Our muscles are just really good at preserving energy. We're not going to break down glycogen without exercise and we do really well just upregulating fatty acid oxidation. So we're never putting a stress on our muscle cells.

By reducing energy intake. We're only doing it by increasing energy expenditure. And this is specific to skeletal muscle. So liver is probably experiencing something very different where we are breaking down. Glycogen and we might be upregulating these fats. I don't know. I've never looked into this, but I just wanted to disclaim that I'm looking at this from a very muscle centric perspective in that the resting state, reducing energy intake doesn't Is not an energy crisis for our skeletal muscle cells.

And why this is important is because of that link to metabolic health and metabolic flexibility. Very determined by skeletal muscle. So, skeletal muscle is like a huge contributor to our whole body metabolic flexibility. So, and if we wanna, you've talked, I'm sure at length about metabolic flexibility.

Oh, sure. But just briefly. Being the ability to respond to insulin. So, we want to, if we eat carbohydrates and we increase blood glucose and we release insulin, then we want to be able to switch to burning carbohydrates. We want to be able to respond to that insulin, let glucose in and start oxidizing carbohydrates.

However, when we're doing something like fasting. Or we're going from rest to exercise. We want to be able to increase lipid metabolism. So we want to be able to burn fatty acids efficiently and be able to make that switch. And that really defines metabolic flexibility at the level of the skeletal muscle.

But I don't know what my point was other than that's a quick question and

[00:21:26] **Dr Mike T Nelson:** you might think of it So the mitochondria in muscle under the condition of fasting It's not that much of a stress to them because they're very easily able to take That and make energy from it and we don't have this huge energy drain on the system So there's no reason for them to do this like super hardcore at a high rate or anything like that You Is that correct?

And the assumption there is that. These are metabolically flexible people, or that this is just healthy muscle tissue and this may not necessarily be true for unhealthy muscle tissue?

[00:22:06] **Kristi Storoschuk:** I don't know if I really understand your question, but to go with your first point, that yes being able to, if you're going into the fasting state burning lipids.

at rest is not inducing a metabolic stress.

[00:22:22] **Dr Mike T Nelson:** It's easy. It's ah, we don't need adaptation. This is, we do this all day. No worries.

[00:22:26] **Kristi Storoschuk:** Yeah. You can't exceed your mitochondrial capacity base. Like essentially, like our mitochondrial capacity is so much bigger than our metabolic demands at rest.

So So the only way to exceed that is to exercise and increase energy expenditure and I always borrow Tommy's Tommy Wood, his cognitive demand theory of cognitive decline, and I think of that in terms of the mitochondria, in that we need to exceed the demand in order to induce an adaptation and fasting or caloric restriction, just reducing energy intake at rest is not going to ever exceed our capacity.

So then we wouldn't expect an adaptive response. It's just, and like also the idea that burning more fat induces fat adaptation. I'm not convinced of, but I could change my mind, but I'm currently not convinced that burning fat and forcing our bodies to burn fat actually induces adaptation.

I'm more on the side that it's a response. It's an acute response, but it's not gone. It's not something that is an adaptation, if that makes sense.

[00:23:39] **Dr Mike T Nelson:** Yeah, so second question on that, so this would apply to healthy tissue from healthy people. We're not talking about Pathologies or metabolic inflexibility.

This is just individuals who are at a healthy state. Is that correct?

[00:23:55] **Kristi Storoschuk:** Yeah, and if unhealthy tissue, so overweight, obese, insulin resistant individuals, they still make the switch. It's just not as not as large as someone who is trained or healthy and insulin sensitive. So, if you give lipids, like intravenous lipids, to someone who's super insulin sensitive versus someone who might be insulin resistant we're gonna increase lipid metabolism.

To we're going to make that switch to burning fats. The trained individuals would, and then the insulin resistant people would not would still be able to upregulate fat oxidation. They just wouldn't do it to the same extent. And then they don't shut down carbohydrate oxidation the way a trained individual would in response to high fatty acid flux and similar vice versa.

If you infuse glucose and insulin Insulin sensitive person will rapidly increase carbohydrate oxidation and shut down lipid metabolism. However, that switch is just a lip is just less. in someone who's insulin resistant. And then like glycogen storage insulin sensitive people will respond to an increase in carbohydrate intake and insulin by storing more glycogen.

And that is impaired in someone who's insulin resistant as well. So that means that they're just not taking in that glucose and it's not being stored as glycogen.

And one of the very clear training responses of someone who's regularly exercising is their ability to store glycogen because our glycogen stores grow.

The the more trained that we are, we can store more glycogen.

[00:25:26] **Dr Mike T Nelson:** Cool. And the second point, my thought is if I have someone, let's say hypothetically, I want to train their body on the metabolic flexibility and to increase fatty acid use. Let me know what you think of this. I currently do have them do some fasted training.

The intensity will vary. I'm not entirely convinced that zone two is enough to do it. Maybe the higher volume it is, but I will over time push the exercise intensity in a fasted state. And then contrarily, I will also get their VO two max higher because we know if we look at something like a MFO, so maximal fat oxidation, that the higher, the bigger your engine is, you're just going to burn more fuel.

So if the percentage is the same, You're literally burning more fuel from fat because you've got a V12 engine compared to a three cylinder Yugo that you're, put your feet through the floor and you're trying to pedal around town like a Flintstone to get it going faster. Would you agree on those two interventions or is there something from a practical side you would do different or is that I'm living in the stone ages from 10 years ago and I should go read more PubMed shit?

No. I think that fasting at rest is a different intervention than fasted exercise. So I do think that there's a rationale for fasted exercise. I don't think the evidence is super strong, but I'm still Yeah, it's mixed. I agree. Yeah, I'm still like hopeful and have generated hypotheses around fasted exercise from my own research because there are hints within the literature that reducing carbohydrate availability before exercise, so exercising in a reduced carbohydrate state does change certain signaling responses to training, and there's not a lot of training studies per se, but there are acute studies that could rationalize or at least support a hypothesis towards why facet training might increase our adaptive response to exercise.

[00:27:29] **Kristi Storoschuk:** The training studies that have been done are. are also very promising. So one of my favorite studies is one six week training study in men where they gave them a hyper caloric diet. So 30 percent increase in calories for six weeks. And then they had them train. So both groups, we had a fasted and a fed group.

Both groups are training for six weeks and both groups were consuming the same amount of calories, same amount of food. caloric surplus, 30%, but one group was training before breakfast and the other group was training after breakfast, same breakfast with carbohydrate, high carbohydrate intake. And only the group that was training in the fasted state was protected and improved insulin sensitivity.

And they, so they improved insulin sensitivity in the face of a hypercaloric diet. the group that was consuming carbohydrates before their training sessions, they didn't see any improvement in insulin sensitivity. So suggesting that fasted exercise is doing something that and altering the training response in a positive way in terms of metabolic health.

And there's a couple other training studies, six weeks long as well, showing improved insulin sensitivity with fasted exercise, but not fed. So there's, I think. I think there's a rationale. I don't know if just a general healthy person would experience significant benefits from training in the fasted state, or if you have to be somewhat metabolically impaired to see those improvements just because I think like our world is obsessed with optimization.

[00:29:00] **Speaker 3:** Oh, yeah.

[00:29:00] **Kristi Storoschuk:** And it's often the people like we're often doing things that Are used in the literature in people who are already a bit impaired and see improvements and then we're putting it on like we're using those interventions in an already healthy state. And I just I don't know if the improvements we're going to healthy people are going to see those improvements the same way someone who needs improvements is going to see those improvements.

But if there are. I'm still like one of those people who would be willing to change my lifestyle and do things like that because I am obsessed with optimization a bit. But yeah, so there, I think to your point, I think there is still a rationale for using fasted training. And I just didn't at the point where I'm just a little confused about how the fuels that we use during exercise.

How that actually relates to the adaptive response or if it's just the demands of exercise that are triggering the adaptive response. So a lot of research will suggest that okay, increasing fat oxidation during exercise, which would be increased in the fasted state is beneficial, but I'm like, but why?

Like, why what's the mechanism that's explaining why burning more fat would lead to a burn? Bigger adaptive response. I'm just I'm not at the point where I could conclusively give you an answer for that. But that is an often. I guess repeated message is that oh, if you burn more fat during exercise, you'll improve your fat metabolism.

And that's the rationale for a ketogenic diet. That's the rationale for a fasted exercise. That's a rationale for zone two training because you're basically burning lipids as your soul as your primary fuel. And I'm still just not convinced that necessarily is. Is leading to the adaptive response that we're hoping for but I want it to like I want to believe it is I just like I need more convincing evidence

[00:31:05] **Dr Mike T Nelson:** Yeah, I think it's my bias is I think that does so if you are Burning more fat in your training does my bias is that it would lead to an increase in fatty acid metabolism the big caveat I would say though is What intensity and what parameters do you need to do that?

And what was your baseline, right? Because I'm sure you've seen some of the data from like Helges 1999. I published a study in 2015. Gudecky did a study. We're just pulling randos off the street that are, recreational athletes that are healthy. Stuffing them in a lab, putting them at the same percentage of their VO two max.

And then how well they used fat, they were fasted. They were low to moderate intensity was like 19 to 93 percent difference. This is a massive range of some people were really good at using fat. Other people were not very good. However, if you look at the output, they were still able to hold that percentage of their VO two max.

It's just what fuel were they actually using? So I think without quantifying what end of the spectrum these people are on if you've got someone who's really good at using fat already under those conditions, yeah, doing 40 percent of their VO2 max for Zone 2 training is probably not going to do dick for them.

Right? But, if they're mostly burning carbohydrates to do 40 percent of a VO2 max, and you do it fasted, and maybe you bump the intensity a little more. Right. My bias is I do think that is a stimulus to increase the use of fats. But again, what parameters, how long do they need it? Just like with weight training, what's your intensity?

What's your volume? All that stuff is, I would say, very much up in the air.

[00:32:53] **Kristi Storoschuk:** I agree with you. It's definitely a stimulus to upregulate fat oxidation. Again, I just like I still don't know. Would you have any hypotheses around like a mechanism that would explain why increasing fat oxidation would increase?

Like adaptive responses that make you more, fat adapted or

[00:33:10] **Dr Mike T Nelson:** Yeah, my, my basic one would be, I think it's just the said principle, specific adaptation to impose demand, right? You're getting better at running more fats through the mitochondria. So you have an upregulation in that process. Again, what part of the process is it a flow issue?

Is it CPT1A enzyme? Is it a transporter? Is it the size of the mitochondria? I don't know what one of the pathways it is. If I were to purely guess and throw darts at a board. I actually think it's a CPT1A enzyme issue, is my guess. I think the mitochondria are a little bit over designed for what they're doing.

I think it's an issue getting the stuff into the mitochondria at a low level. Again, that's so far down in the nerd weeds I, I can't point you to a study to justify that either.

[00:34:01] **Kristi Storoschuk:** Yeah, cause I agree, and what you're saying makes sense, but then I always think of the HIT literature and being like, well, HIT is, inducing mitochondrial and fat adaptation despite using carbohydrates.

So yes, but

[00:34:15] **Dr Mike T Nelson:** that's because your upregulation is in the rest period to repay the debt that you accumulated from HIT, is my guess.

[00:34:23] **Kristi Storoschuk:** You don't think that like a 10 minute steady state HIT, or high intensity bout, or like a, yeah, like a single acute without the intervals, you don't think that would induce fat adaptation?

[00:34:38] **Dr Mike T Nelson:** I think it would, I think it would to some degree, now my bias, let me know what you think on this is that the stimulus is high enough for the body to be like, oh shit, we need a whole bunch of fuel, what are we going to turn over at a fast enough rate to create ATP, okay, it's mainly going to be carbohydrates, however, after that state, whether you believe it's oxygen debt or epoch or whatever theory you're with you took the system and you disturbed homeostasis so hard that it has to use energy after you stop exercise to get you back to baseline.

My bias is, I think that's primarily fatty acid use is upregulated to drive you back to baseline faster. I don't think it's during the actual exercise, but I think that stimulus overall is probably pretty potent to do that. Okay.

[00:35:31] **Kristi Storoschuk:** Interesting. So it's you're still on board with like that. It's the upregulation of fat oxidation.

That's inducing the

[00:35:38] **Dr Mike T Nelson:** adaptation. Think so. Right. Because I think the aerobic component. Is what's going to help drive you back to baseline from that huge disturbance to the homeostasis by doing a true HIIT type training.

[00:35:53] **Kristi Storoschuk:** Yeah, I don't know. I'm guessing here, right? That goes into literature

[00:35:57] **Dr Mike T Nelson:** looking at aerobic base, higher levels of aerobic fitness.

Generally, you can get back to baseline faster. Heart rate recovery, parasympathetic tone, all that kind of stuff.

[00:36:07] **Kristi Storoschuk:** Yeah, interesting. I'll have to give that some more thought. What are your thoughts? Interval training. I would have just thought that we're inducing the high intensity. The actual interval of the high intensity, not the rest interval is, yeah, like you said, such an energy demanding time and bout of exercise that we're now having to adapt our mitochondria to be able to support higher energy demands because The whole reason we adapt to exercise is to prevent energy crisis in the state of an increased energy demand.

So that's why we, that's why we need to exceed its capacity. And then we adapt by building our mitochondria and building our aerobic metabolism so that in the face of increased energy demand, we are, we're able to handle that. So that's why I think that the actual, the high intensity bout. Induces that because now we're exceeding the capacity of our mitochondria to re synthesize ATP and we're having we're telling ourselves to respond by building a bigger.

Oxidative engine so I do think it's the actual high intensity. Part that's inducing that because that's when we get an accumulation of and that's when we're going to activate. Because there's literature showing that if you don't. AMPK, so AMP and ADP increase in our cells in an intensity dependent manner.

So we would expect AMPK to be activated in an intensity dependent manner. And if AMPK is so important for the adaptive response that, that is related to our fat oxidative adaptations and our aerobic adaptations then I think there's a pretty strong argument for intensity being really important.

And. Not necessarily the increasing flux. If anything, I think that I don't disagree with you, by the way. I'm not I'm just I have a second part

[00:37:58] **Dr Mike T Nelson:** where I probably agree with you more but continue

[00:38:02] **Kristi Storoschuk:** I was just gonna mention quickly that if anything I think the flux part might be important on a whole body level and not necessarily just to skeletal muscle.

So our ability to increase lipolysis and adipose tissue, release those fatty acids and our things going on in our liver and just the whole body system might be improving. Metabolic flexibility might be improving from a whole body system. Perspective and not necessarily from the skeletal muscle centric view that I typically take.

But what were you going to say? Yeah, I would, I'd say I actually agree with all that. I think it's both. And I think the caveat is my assumption when you said high intensity intervals, my brain went to 10 second intervals. But I agree that if you're doing 20, 30, 40 second intervals at a very high let's say, give an example, get on the rower.

[00:38:53] **Dr Mike T Nelson:** Do a wind gate, do 30 seconds, like balls out as hard as you possibly can. Keep as high a wattage as you possibly can for 30 seconds. The thing that really, I've talked about this a ton in the podcast that just screwed me up for six years was seeing Aaron Davis in Austin years ago, stick one of the early moxie sensors on a quad.

And had someone get on the rower. And so we were seeing the nearest technology can look and see what is the local oxygenation level at a muscle. So they have it on the VMO. We're looking cool. Muscles like 85 percent saturated. Yeah, that's exactly what we think. So it has a person do 30 seconds all out.

And you're watching the oxygen level in the muscle drop to like 19 percent by the time they're done. And I'm like, Oh, this device, the piece of shit. What is this? I was told this is anaerobic exercise. This isn't using oxygen. And I'm like, what the hell is going on? And it took me years to figure it out.

And I think the part I figured out, which I think you'd probably agree with is that the device was correct. that I was probably told something that was an oversimplification, and that as you do high intensity stuff, but the duration starts getting longer, the percentage of that as aerobic power, if you want to use that term, is way higher than I ever suspected.

So I do think from the aerobic system, the use of fat, you're coordinating all these fuels, like little waterfalls, filling a pool down below it, that as those intervals get a little bit longer, now again, this would taper off at some point, because you just can't hold that level of power. I do think there is a lot more of a hit on the aerobic system, which could be fueled by fat, than what we realize.

[00:40:35] **Kristi Storoschuk:** Yeah, and there was a pun in there too with the hit on the aerobic system. Yeah hit on the aerobic system I'm, glad somebody

[00:40:43] **Dr Mike T Nelson:** caught that

[00:40:44] **Kristi Storoschuk:** no, I that's very true and I think I don't know if many people are even doing I guess tabata would be the closest thing to someone practically using something under

[00:40:56] **Dr Mike T Nelson:** They're not doing a hundred and seventy percent of their vo2 max.

They're like Not even close to that level. That's my pet peeve about Tabata stuff. It's not that it was a poor study. It was just, yeah, I've had people come here and stick them on the rower, do a true VO2 max 2k and be like, okay, that's a hundred percent legit or as close as we can get all of your power.

And then you put them at 170, and yeah it's another universe. But anyway, go ahead.

[00:41:21] **Kristi Storoschuk:** Yeah, no. I would never be able to push myself on my own, like I would be, the only way I would get there is if someone's screaming at me in the lab, the way I scream at my participants.

[00:41:33] **Dr Mike T Nelson:** Yeah. And even then, Tabata designed the study for high level athletes not to be able to complete all the rounds.

Like it was designed to fail with incomplete rest, but

[00:41:41] **Kristi Storoschuk:** yeah, no, I like using tabata like timers Like just go all out and I also think that like people get so caught up on protocols Oh,

[00:41:52] **Dr Mike T Nelson:** yeah

[00:41:53] **Kristi Storoschuk:** Some of them are so arbitrary in a lab like

[00:41:55] **Dr Mike T Nelson:** most of them are arbitrarily picked if you look at the history of them

[00:41:58] **Kristi Storoschuk:** Yeah, so I like I feel like people need to not be so, criticized, criticize things online when people use the terms like hit or Tabata or whatever honestly, let's get people moving.

And if they want to call it hit and they're going all out and working hard, like kudos to them.

[00:42:17] **Speaker 3:** Yeah,

[00:42:19] **Kristi Storoschuk:** social media can be a toxic place for the exercise world sometimes.

[00:42:23] **Dr Mike T Nelson:** Oh yeah. Am I the only thing that does still trigger me as people who are like, selling programs of Tabata preacher curls.

It's well, you're not even in the same universe for Christ's sake. Just, you just want to use Tabata cause it sounds sexy. Go away.

[00:42:38] **Kristi Storoschuk:** Yeah. I'll give you that one. But no,

[00:42:41] **Dr Mike T Nelson:** I do agree. It's Hey, if you're doing hit and you're making progress and it works for you, cool. What am I going to tell you?

Hey, keep doing it, man. If it works, great. If it doesn't work, okay. There might be some better ways to do it, possibly. Let's look at your output. Let's look at some other things. But, yeah, I agree. If you're happy with the results and you're making progress, then cool.

[00:42:58] **Kristi Storoschuk:** Yeah, no, for sure. Yeah.

[00:43:01] **Dr Mike T Nelson:** But I interrupted your long tangent there about metabolic flexibility and mitochondrial health.

[00:43:06] **Kristi Storoschuk:** Oh, gosh. I don't even know. I feel like you need to prompt me on a question. Ask me something.

[00:43:11] **Dr Mike T Nelson:** So, how would you define, for the listeners, how would you define mitochondrial biogenesis? This is a term that, obviously, we use a lot.

It's in the literature. But, again, like Tabata's, I feel like it's,

[00:43:24] **Speaker 3:** that and

[00:43:24] **Dr Mike T Nelson:** mitochondrial content, I feel like they're getting to the point, they're so sexy now, they're becoming extremely bastardized as terms.

[00:43:31] **Kristi Storoschuk:** Yeah, I mean we just described, we just define it in our lab as the expansion of the mitochondrial reticulum.

So the upregulation of both the, Nuclear encoded and mitochondrial encoded mitochondrial proteins that expand the mitochondrial reticulum. So it's not that, you might not necessarily be making a brand new mitochondria, but you're expanding the reticulum. So the whole energy system, the whole mitochondrial system increases, and that would be, the definition of mitochondrial biogenesis.

And then, So could that be

[00:44:06] **Dr Mike T Nelson:** A size increase and a number increase. Is that correct?

[00:44:10] **Kristi Storoschuk:** Yeah. And then the mitochondrial function also gets thrown around a lot. And I personally don't even have a definition for mitochondrial function. I don't even know what that means. And not from an an uneducated perspective.

It's that I, Can't find a good definition of mitochondrial function. So like the way we can measure mitochondrial function in our lab using respirometry. So we have the O2Ks, and I don't personally use the O2Ks. I tried to learn and, oh gosh, it seems like a very tedious process to So now is this

[00:44:41] **Dr Mike T Nelson:** isolating?

Mitochondria to get them to run in a lab and put them in their little closed environment and measure all the gas that goes in and out, like a metabolic cart, but for just your little mitochondria in a dish.

[00:44:53] **Kristi Storoschuk:** Yes, we don't actually isolate the mitochondria. We isolate just the fibers. So choosing apart the individual fibers and then adding different substrates, so carbohydrate or lipid substrates, and then seeing how much oxygen, the oxygen consumption.

[00:45:08] **Speaker 3:** Got it.

[00:45:09] **Kristi Storoschuk:** So you can measure the respiratory capacity of muscle using something like that, and that would be an indicator of mitochondrial function, but it's only measuring the function of mitochondria of the specific substrate that you give it. So, you can't say okay, so if The respiratory capacity using the O2 case goes up, giving it a lipid substrate, and you you conclude that mitochondrial function went up, that could be a potentially inaccurate conclusion, it's that the capacity to use whatever substrate you gave it, that went up, it's not necessarily mitochondrial function went up but I don't know enough of that literature other than, there's definitely controversy with the term mitochondrial function, and we've just landed on mitochondrial capacity as a, an overall term that encompasses mitochondria, increases mitochondrial content, and then increases in mitochondrial function.

But yeah, there, there's definitely different. Even using something like PGC1 alpha mRNA expression as an increase in mitochondrial biogenesis could be completely inaccurate. We're just assuming that this gene is now expressed and that means that what our mitochondria now have increased the you, the gold standard would be using something like this.

TM and measuring like actually counting or using the size of a mitochondria using those images. But so I don't know, I think it's a bit controversial and we do throw around these terms probably most likely inaccurately. But I'm still learning because I haven't even done my analysis yet.

I've got boxes and boxes of muscle tissue that I will be. I will be analyzing at some point when I'm done my studies, and I would probably know a lot more on these specific outcomes at that point, but right now, I'm just in data collection which I guess brings me to what I'm looking at so

[00:47:17] **Dr Mike T Nelson:** I'm actually I ask one quick question on the mitochondria?

[00:47:19] **Kristi Storoschuk:** Yeah.

[00:47:19] **Dr Mike T Nelson:** Correct me if I'm wrong, but what I understand is that the mitochondria themselves are actually I don't want to say over design, but have a huge buffering capacity. That, at any one point in time, if we stick those mitochondria back in the muscle cell, the argument is, the capacity Of the mitochondria just intrinsically itself is maybe never a rate limiter, but if you take them out, you put them in these little dishes, like you said, you give them just a shit ton of fuel.

They do fine. Like they actually do pretty good from a healthy individual is. So my assumption is that in physiology, you can upgrade them. You can make them bigger. You can increase quality. But when they're running in terms of a fatigue or a limiter of exercise, do they need this massive buffer zone to operate?

We can't get muscle glycogen below 30, 40%. There's some buffer zone that's fixed. Or is it getting the substrate into the mitochondria to give them enough fuel that's a rate limiter? Which is why I said CPT1A, and my bias is I think that's a rate limiter more so than mitochondria itself.

[00:48:28] **Kristi Storoschuk:** Right. Yeah. Yeah. Yeah. A limiting factor of fat oxidation. I think most people land on carnitine and CBT one being the mechanism that explains why we can't Keep burning fat at high intensities Because we just we that acetyl the acyl Co a pool is like being stolen or carnitine It's being used and then carnitine can't get back out.

I can't remember the whole pathway, but yeah I see the

[00:49:00] **Dr Mike T Nelson:** way basically just get co opted I think somewhere else and ceto Co a can go down multiple different, you know The old Krebs song like all roads lead to ceto Co a well that type of thing

[00:49:10] **Kristi Storoschuk:** Yeah. Oh, yeah, that's it. The acyl CoA groups can't basically re, be reused to make acylcarnitine and then yeah.

So that being the very limiting step in fat oxidation and why we see that as we increase intensity, we increase, we reduce, we, well, we initially increase fat

oxidation, but then it reaches a certain point and then at a certain intensity, we start burning more carbohydrates and fat oxidation comes down.

And then, yeah, being explained by that carnitine theory which I think isn't necessarily a theory. I think it's actually, there is some good evidence to suggest that's actually occurring. Yeah.

[00:49:47] **Dr Mike T Nelson:** Yeah, and you were going to talk about, I think you have a study looking at lactate that is related to all this, if people aren't incredibly confused enough as it is, because we're going down the nerd chute here, but for people who are interested, it's like a

[00:50:03] **Speaker 3:** Yeah,

[00:50:04] **Dr Mike T Nelson:** when you get into the weeds of stuff like people assume I think from The outside looking in that oh science must have all the answers and like One of my favorite quotes is the tim noakes thing.

He's like, all the stuff i'm going to teach you now 50 of this is wrong. He's I just don't know what 50 like we've fast forwarded enough time He's like i'm going to tell you a bunch of shit that's wrong, but I don't know what's wrong right now you know and it's just You know, ever going on process and, from hanging around conferences and I've seen two of the top protein researchers in the world, have arguments about the exact same papers that I know both of them read.

One of them was the author on the paper. For a half hour. I'm like, these are like two really smart people who know their field, who read the paper. One of them did the actual paper themselves and they're looking at the same data. This is not even over multiple studies and they still don't agree.

[00:50:58] **Speaker 6:** It's like

[00:50:58] **Dr Mike T Nelson:** a lot of times people think it's super black and white, but it's always these random shades of gray and stuff changing all the time.

[00:51:04] **Kristi Storoschuk:** Totally. And that was the most humbling part of that. entering academia and starting my PhD because I thought things were figured out and I thought what I knew, like everyone thought, and then I got,

[00:51:17] **Speaker 6:** oh

[00:51:18] **Kristi Storoschuk:** no, there's actually alternative views and a lot of what I know isn't actually true and I know nothing now so I'm just gonna stop talking.

[00:51:27] **Dr Mike T Nelson:** Yeah, that's the running joke I have with my buddy Cal Deets is like, it's yeah, the longer we know stuff, the more we know stuff, but then the more we know, we don't know stuff. And then. In 10 years, we'll just know fucking nothing.

[00:51:41] **Kristi Storoschuk:** Yeah, I know. That's why I get a bit like caught up or not or

bugged by this trying to find the exact protocol and the exact intention and everything.

It's no, we know exercise is good. Do it, do some intense exercise, do a lot of it. More is usually better because most people don't exercise enough. And also just move your bodies. Don't eat a diet that doesn't cause you to gain weight and eat enough protein and that's probably all you need to know.

[00:52:14] **Dr Mike T Nelson:** Yeah, you're pretty darn close. Again, same thing. My pet peeve is people arguing about what the optimal protocol is. And it's like, all right, what did you do so far? Like we're arguing about, okay. HITT well, I want to increase my VO2 max. I want to use it. What's the best protocol.

Okay, cool. Then we could have some discussions about that or whatever. But when you ask the person okay, what have you done so far? So I can iterate off what you have seen as a person. They're like, well, I haven't done anything yet. I'm like, well, shit, I don't know where to start. I can give you a general program and they're like, oh, but is this optimal

[00:52:46] **Speaker 3:** for you?

I

[00:52:47] **Dr Mike T Nelson:** have no freaking idea and we're never going to figure it out until you start somewhere With something that is established. That's a good solid beginning But without that like you said there's no way to get to this mythical Optimal like i've joked with andy many times before andy galpin that I used to put in my slides like optimal, and then I would put like pots of gold and rainbows and unicorns and stuff, because I'm like, you can't come to a lab and test optimal.

Like I understand what people say when they mean it, but you could give me 2, 000, 000 and ask me what's the optimal protocol to increase VO2 max, and I can't give you an answer. You give me 10, 000, 000, I'd still not give you an answer.

[00:53:25] **Speaker 3:** We could

[00:53:25] **Dr Mike T Nelson:** tell you, hey, for this population, here's protocol A and here's protocol B, Cool over this eight week period for these people protocol B was better and you can iterate that process for eternity We can get closer to optimal but you can never get the air quotes optimal answer So again, that's my little soapbox.

[00:53:46] **Kristi Storoschuk:** Yeah What's

a cool though? Is that? In some of the literature increasing intensity reduces variability in responses. A lot of times, yes. The non responders.

Yes.

Which, I'm, like, I'm a fan of high intensity exercise. And so, I think that's pretty cool that you can eliminate those responses.

Non responders by just increasing intensity, and it's probably going to work for most people then. Whereas there's more variability in responses and both health outcomes and then physiological like fitness outcomes. If you're only working at a moderate intensity.

[00:54:23] **Dr Mike T Nelson:** And we see some of that literature for volume now with hypertrophy, which I know is contentious, but some of the non responders when they did more volume, they became shocker a responder.

So

[00:54:32] **Kristi Storoschuk:** yeah, totally. Anyways, yeah, I'll just briefly, mentioned my study,

[00:54:37] **Speaker 6:** yeah,

[00:54:37] **Kristi Storoschuk:** I'm that I'm in the midst of and that I spend most of my days so I'm all, I'm studying fasted exercise. So I used the Burke

protocols, the five day ketogenic protocols, but I subbed in fasting instead of ketogenic.

ketogenic diets which is a very different physiological state than being in a, in ketosis. My participants might be in, or actually are often in ketosis in the morning because they're just young, metabolically flexible individuals that 14 hour fast induces ketosis in them. So it's a five

[00:55:09] **Dr Mike T Nelson:** day, 14 hour fast, is that right?

[00:55:11] **Kristi Storoschuk:** Yeah, they're just there. It's we're using time restricted eating. They eat within a 10 hour eating window every day and then they show up in the fasted state. They either get carbohydrates or they don't in a beverage. I'm blinded to the intervention obviously. So I don't know what group they're in, but they're either drinking carbs or drinking a placebo and then they get on the bike and do hit.

So the protocol is one minute on, one minute off, eight rounds. So it's a 16 minute workout, but it's really hard. They're definitely having, it's a hundred percent of work rate max. So eight minutes of a hundred percent of your work rate max is very difficult. Yeah. That's

[00:55:46] **Speaker 3:** brutal.

[00:55:47] **Dr Mike T Nelson:** Yeah.

[00:55:48] **Kristi Storoschuk:** So they don't like it.

But some of them actually like the muscle biopsies more than the hit. I've gotten that comment.

Which is actually hilarious. But yeah, so I'm, but my primary outcome is lactate threshold. So based on based on the. Keto literature that we can like basically induce this fat adaptive state where even once carbs are restored, carbohydrate intake is restored. If you follow five days of a ketogenic diet on top of your training with an, with athletes Even once carbohydrates are restored, there's still an increase in fat oxidation compared to those on a high carb diet.

So, perhaps there is something about increasing fat oxidation during that bout of exercise that is resulting in that fat adaptive response, which is what we talked about earlier. But I am looking at fasted exercise and whether it has to do with just that pre exercise carbohydrate restriction, or if it has to do with the fact that

They were on a ketogenic diet, and so if my results are different, then it could be explained by the fact that on a ketogenic diet and exercise, their muscle glycogen is probably lower because it's a five day protocol.

In the Burke literature, Louise Burke if you're familiar with that five day protocol. And so they're like, it's a very different physiological state with low muscle glycogen and low liver glycogen, whereas in my participants, they're just going to come in with low liver glycogen, normal muscle glycogen.

So, another thing that we didn't touch on when we were talking about fasted exercise is that as we increase intensity, the difference in fuel utilization between a fasted and a fed person is eliminated. So as if we're doing hit, I like everyone's going to be at the same rates of carbohydrate oxidation and the same rates of fat oxidation regardless of nutritional state.

And. That will eliminate, so again, if I see differences in my participants, and once I analyze the tissue if I see differences, then it's going to be independent of that fuel oxidation during that bout of exercise, which I think is unique because most of the fasted exercise literature uses moderate intensity exercise, oxidation during that bout of exercise.

I know I'm, like, This might be complicated. I'm one. I'm talking fast and to like spilling out just a lot of information at once. But I'm just really. Intrigued and interested in that idea that using different fuels during that bed of exercise and how that results in different adaptations.

But if I do so more of that again, it's just that if I do see differences, it's going to be independent of. Fuel oxidation during the fuel. the preference of fuel during that bout of exercise each day. Because we would expect that they're using the same fuels. But my primary outcome is lactate threshold.

So, I'm really interested in lactate just because it's such a cool. One, it's a molecule that is just gaining a lot of attention right now, so we need to, in, basically, the literature needs to pick up and we need to figure out what this molecule is doing and what it means so I would love to contribute to that literature, and two, it's an, it's a window into the bioenergetics of our skeletal muscles, so, yeah, you could use a met cart, and But I think lactic is way cooler to measure than like actual, just looking at the ratio of the CO2, V02 because well, I it's not an indicator of carbohydrate oxidation, but it is an indicator that your.

Production of lactate has exceeded your whole body capacity to oxidize lactate. So a lactate threshold test and the power output associated with a certain blood lactate level tells us a lot about the health of your skeletal muscle and Both and the respiratory capacity of your skeletal muscle. So I think it's just a really cool thing that we can measure.

That's also just easier than a met cart that to look at the bioenergetic or to provide a window into the bioenergetics of your skeletal muscle. And so the higher your lactate threshold, The more trained you are and we would expect that you have a higher fat oxidative capacity because you're The increase in blood lactate should tell us that you're burning more carbohydrates as lactate is a byproduct of glycolysis So

[01:00:20] **Dr Mike T Nelson:** yeah, that's the George Brooks study.

He did several years ago That was pretty cool where he compared athletes to people I think with metabolic syndrome Looking at lactate as a surrogate for carbohydrate use .

[01:00:32] **Kristi Storoschuk:** Yeah, exactly. And so, again, it doesn't tell us that muscle lactate production is going up or if it has to do with outflow, right?

So the influx or influx of lactate, so it, like glucose lactate is just if we're looking at blood glucose or blood lactate, that's just the, that's just like a snapshot of the net of inflow and outflow. So it doesn't, but those things should. Increase in parallel, so our ability to oxidize lactate should increase as an adaptation that parallels increases in.

Adaptations that would produce less lactate, so the greater our fat oxidative capacity, then, the less lactate we should be producing for a given power output. So if you're tracking your lactate threshold in, if you're doing some sort of training protocol and using lactate threshold as some as a metric of your progress then you would expect to see lactate threshold.

Increasing as you become more trained and that should indicate that your fat oxidative capacity, respiratory capacity, mitochondrial function are, and capacity are increasing as a result of the training. And you could use lactate as a measure of that. And then, and your lactate threshold is essentially like zone two.

So, your zone two would be the upper limits of the moderate intensity domain. So, The in classical like exercise, metabolism, exercise, physiology, literature,

there's three exercise domains. Really? There's the modern intensity domain. There's heavy and they're severe. And lactate threshold is the demarcates the moderate from the heavy.

And so your zone two would be the upper limits of moderate intensity and zones, came from more of the endurance world. But zone two would actually fall within the moderate intensity domain of exercise intensities. And that would fall just below your lactate threshold. And we, in our lab, define that as two millimolars.

Again, arbitrary, like it depends on There's I think like 30 different definitions of lactate threshold.

[01:02:43] **Speaker 3:** Yeah.

[01:02:44] **Kristi Storoschuk:** We pick one. We're going with it. It's two millimolar. So the power output associated with two millimolar of blood lactate if your power output goes up for that same amount of lactate, that is, that should represent increased adaptations in blood.

Mitochondrial oxidative capacity in your skeletal muscle, and so that's why I'm taking biopsies, and I'm going to be looking at mitochondrial outcomes, but I'm also going to be looking at lactate dehydrogenase, different enzymes, different transporters and hopefully paint a picture that explains any changes in lactate metabolism or just any changes in blood lactate that I Report in my participants just after 5 days of training and whether fasting beforehand or carbohydrate intake before your bouts of exercise 5 days consecutively if that changes our ability to if that changes any adaptations and whether that can be reflected in a lactic threshold test.

[01:03:46] **Dr Mike T Nelson:** Last couple of questions. Yeah. As we wrap up how much carbohydrate do you give them beforehand? And what is how soon before exercise? Yeah, 1.

[01:03:55] **Kristi Storoschuk:** 3 grams of maltodextrin per kilogram. Oh, so

[01:04:00] **Dr Mike T Nelson:** that's a pretty good dose. So 200, so 220 pound mammal, 100 kgs would be 130 grams. Is that right?

[01:04:07] **Kristi Storoschuk:** Yeah, 1. 3 per kilogram.

[01:04:10] **Dr Mike T Nelson:** Yeah. 1. 3 grams per kg, right?

[01:04:13] **Kristi Storoschuk:** Yeah. Yeah.

[01:04:13] **Dr Mike T Nelson:** Yeah.

[01:04:14] **Kristi Storoschuk:** So yeah, it's a lot.

[01:04:16] **Dr Mike T Nelson:** Yeah. That's good.

[01:04:18] **Kristi Storoschuk:** And it's 30 minutes before exercise. So trying to get that increase in insulin, trying to shut down hepatic glucose a bit. So I didn't want it too close to exercise, but I didn't go too far away that we're now like synthesizing glycogen and storing it.

So you're using

[01:04:37] **Dr Mike T Nelson:** it to primarily move insulin.

[01:04:40] **Kristi Storoschuk:** Yeah. Because I want to know. What happens when we exercise in a state of elevated glucose and insulin. So I didn't want things to fall back down to baseline and then us exercise. I want it to be like in that state. And just have very different physiological metabolic states before exercise to be able to compare those.

[01:04:58] **Dr Mike T Nelson:** So is your, so my guess would be. The group that does get the carbohydrate will see better performance, a higher power output on the 2 millimolar lactate test. Was that your hypothesis also?

[01:05:15] **Kristi Storoschuk:** So the lactate threshold is conductive. Or maybe that's not the outcome of it. So well, one, the performance isn't different, assuming that I've had both carbohydrate and fasted participants.

Okay.

[01:05:30] **Dr Mike T Nelson:** So your null hypothesis is no difference.

[01:05:34] **Kristi Storoschuk:** No, my, my hypothesis is that, well, we have to pick a hypothesis and it's that fast and training will lead to greater adaptations. But. the lactate threshold test pre and post is done under the same conditions in both groups. So they're both just fasted.

[01:05:51] **Dr Mike T Nelson:** Oh, okay. So that's like a pre and post test. That's not, I gotcha. I gotcha. Gotcha. Okay. Cool. The intervention, the five days of hit, that's where the nutritional intervention comes in. And I'm also, all my participants are wearing CGM. So I'm also looking at glucose glycemic variability and whether fasted versus fed exercise has any influence on our 24 hour glucose level day to day.

[01:06:13] **Speaker 3:** Nice. Very cool. Yeah. Yeah, but anyways,

[01:06:17] **Kristi Storoschuk:** the lactate threshold is under the same conditions.

[01:06:19] **Dr Mike T Nelson:** Got it.

[01:06:19] **Kristi Storoschuk:** Pre and post. Yeah.

[01:06:21] **Dr Mike T Nelson:** Cool. And when do you expect this study to be out?

[01:06:25] **Kristi Storoschuk:** Oh gosh. Well, I'm about halfway, and it started in September, so we're worlds away.

[01:06:32] **Speaker 3:** So halfway on data collection, correct?

[01:06:34] **Kristi Storoschuk:** Yeah. And then I have to do all the wet lab analysis, so that's gonna be fun.

Yeah.

[01:06:39] **Dr Mike T Nelson:** Okay. Cool. Cool. Well, yeah, definitely keep us updated on that. I appreciate all the work you're doing. It's super cool. And it's one thing to sit around like I do and come up with crazy ass hypothesis. It's an entire another world to go into the lab and actually do them on living human subjects. I don't think most people who've never done human subjects studies have any idea of the shitload of work that goes into it that most people.

Even in a final publication, we'll just never see, so so much for doing all that stuff.

[01:07:12] **Kristi Storoschuk:** Well, you've been through the ringer as well. Oh, yeah. Thank you as well, but yes No, I definitely have a new appreciation

for just human research and also just like a PhD student. I've never you're everything.

You're, like, admin you're scheduling things, you're cleaning, you're checking inventory, you're ordering, you're also showing up for your participants, you're analyzing there's

just

A million rolls

that I'm like, oh my gosh how am I going to get all of this done?

[01:07:43] **Dr Mike T Nelson:** Yeah, and some shit goes wrong, right?

Like somebody doesn't show up, the metabolic heart of my kid barfs a few times, or the bike all of a sudden quits, or blew a fuse, or it's just Oh my god.

[01:07:53] **Kristi Storoschuk:** Yeah, we've had pedals fall off our bike and I'm like, Oh, okay, we're in the middle of a test, get on the other bike.

[01:08:00] **Dr Mike T Nelson:** I remember running out once to go get a fuse and trying to get back in time before the subject actually showed up because we blew a fuse on the only bike we had.

We had another backup and that one broke the day before and no one told me. And

[01:08:13] **Kristi Storoschuk:** yeah, I know it's a mess. Yeah. And then working with other

people too, who are also using the same equipment and it's just, Oh, that was my nightmare.

[01:08:20] **Dr Mike T Nelson:** Scheduling. That's life. That's actually one of the reasons why I put fast at exercise because I knew I could get to the lab early enough, turn on the metabolic hearts and get them calibrated before anyone fucked with them.

[01:08:33] **Kristi Storoschuk:** Totally, that's hilarious. Cool.

[01:08:35] **Dr Mike T Nelson:** Well, where can I know you got to take off? Where can people find more about you? And I know you got some cool stuff on Instagram. You're doing some stuff with CrossFit and all sorts of other great stuff.

[01:08:45] **Kristi Storoschuk:** Yeah, I guess I'm the most active on Instagram. These days I'm a bit quieter.

Like I said I'm scared to talk these days because I don't know what I know. But but yeah, Instagram Kristi Stores Check. If you wanna, you can use this the title of this podcast to spell my name on Instagram. But that's where you'll find me. You'll find me mostly, but I'm on Twitter and LinkedIn, et cetera, but you can just search my name.

You'll find me. I think I'm like the only Kristi Storeshook in the world. So.

[01:09:15] **Dr Mike T Nelson:** Perfect. Awesome. Well, thank you so much for all your work. I really appreciate it. And thank you for coming on here and enlighten us about the land of mitochondria and everything else. And we're super excited to hear what you find with your studies.

I know you've got some other super secret studies that hopefully will be out a little bit sooner too. We'll talk about those once they're public knowledge.

[01:09:36] **Kristi Storoschuk:** Yeah, we'll definitely have to talk about those. Although, I'm sure you'll get the head leaders on to talk about those outcomes, too.

[01:09:42] **Dr Mike T Nelson:** Yeah, hint it's with dr. Tommy and Dr. Andy Kelpin. So, yeah.

[01:09:46] **Kristi Storoschuk:** Yeah. No, thanks for having me on. This was super fun. I appreciate all the work you do as well. And it was a real honor and privilege to be able to speak with you today. So, thank you, Mike.

[01:09:56] **Speaker 3:** Thank you!

[01:09:57] **Dr Mike T Nelson:** Thank you so much for listening to the podcast. Really appreciate it. Huge. Thanks to Christie for coming on and spending all of her time discussing the metabolic flexibility and the intricacies and everything

that goes into that super excited for her research to be completed. I'm excited to see how it all turns out.

Make sure to follow her on Instagram is probably one of the better places. And if you want to learn more about the concept of metabolic flexibility, combined with flexible dieting. Make sure to check out the Flex Diet certification. It opens this coming Monday, which is June 17th, 2024. It'll be open for one week till June 24th, 2024.

Go to flexdiet. com for all of the information. You can still get on to the wait list there. I'll have a bunch of fast action bonus items. And all the information will be sent to you over the next week or two. And if you have any questions about the certification, send me a note via the newsletter. I will get back to you.

This is the certification primarily looking at nutrition and recovery aspects, as I mentioned, based on the concept of metabolic flexibility. We also have a ton of expert interviews in there. Everyone from Dr. Stu Phillips, Dr. Jose Antonio. Dr. Hunter Waldman, Dr. Dan Pardee, Dr. Eric Helms, and even Dr.

Steven Guillen talking about the neuroregulation of appetite, and many more. So check it out. Go to Flexidiet. com, sign up to the waitlist, and you will get all the information sent to you. If you have any questions, you can contact me by just hitting reply. Once you're on the newsletter and I will get back to you as always Thank you so much for listening to the podcast.

We really appreciate it Again, huge. Thanks to Kristi for all the information there Make sure to check out all her great stuff on instagram and everywhere else for publications And i'll talk to all of you next week

Personally, I don't care for puppets much. I don't find them believable. I don't believe you!

[01:12:09] **Nancy:** This podcast is for informational purposes only. The podcast is not intended as a substitute for professional medical advice, diagnosis, or treatment. You should not use the information on the podcast for diagnosing or treating a health problem or disease or prescribing any medication or other treatment.

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