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S1 00:00 Hi. This is Tim Lightfoot, the director of the Huffines Institute for Sports Medicine & Human Performance. I am so excited to let you know that the Huffines Institute now has apps for your smartphones and your tablets. We have apps for the Apple products and for Android products. You can go to iTunes or go to Google Play, either one, download those Huffines apps, and you can pull in our content every week. Now on to the podcast.

S2 00:28 [music] Welcome to the Sports Medicine Podcast, brought you by the Sydney and J.L. Huffines Institute for Sports Medicine & Human Performance in the Department of Health & Kinesiology at Texas A&M University. At the Huffines Institute, we're always working to facilitate, apply, and bring you the most up-to-date coverage of the wide world that is sports medicine and human performance, all in a language you can understand and share with your friends. And now, here's our host, the director of the Huffines Institute, Dr. Tim Lightfoot.

S1 00:58 Well, hello, and welcome to the weekly edition of the Huffines Institute for Sports Medicine & Human Performance Podcast. I'm your host, Tim Lightfoot, and I'm so glad that you took the time to download us and you're joining for this conversation. Every week, we work to bring you someone interesting in the world of sports medicine and human performance. And again, as I say every week, because it is true every week, that this is no exception. We have a great guest with us, we have Dr. Heather Vellers with us in the studio. Welcome to the podcast Heather.

S3 01:26 Thank you.

S1 01:27 We're so glad to have you, and we're going to tell the audience a little bit about you while we've got you here, and then we'll go on from there. So Dr. Vellers is currently a research fellow at the National Institutes of Environmental Health Sciences in Durham, North Carolina. That's a branch of the National Institutes of Health, so she is a federal employee. So you are listening to your federal tax dollars as we speak. Is that not correct, Heather?

S3 01:53 That's correct.

S1 01:53 That's correct. That's right. Actually, this is a bit of a return visit for Heather. Heather actually graduated from here at Texas A&M with her PhD in exercise science. She previously got a master's degree and a bachelor's degree in exercise physiology and clinical exercise physiology from the University of North Carolina Charlotte, and so Dr. Vellers' research is in a couple of different areas that we're going to talk about. We're going to talk about mitochondria with her. If you haven't heard about mitochondria, you should hang on because it's really important. We're also going to talk about overfeeding and what that actually does to whether or not you're active or not. So again, we're glad to have you back in the studio, Heather.

S3 02:33 Thank you.

S1 02:34 Let's just get started. First of all, let's start with the some of the stuff you did previously, and that-- you were very interested in the effect of overfeeding, on whether or not an animal was active or not. Tell us a little bit about that and why that's important.

S3 02:49 So there are human studies that show that when you overfeed, so when you have caloric intake that's above your normal weight maintenance needs, that humans typically become less active. And there's been various studies to show that, but no one's been able to show what that mechanism is linking overfeeding and reduced activity. So my study--

S1 03:12 So the first question that somebody is going to ask, and so we'll just deal with it right now.

S3 03:15 Okay.

S1 03:16 Are they less active because they weigh more?

S3 03:19 Actually, both in humans and in mice, there's a lack of to-- well, there's a lack of correlation between activity and body weight, so--

S1 03:29 So there's no relationship between body weight and activity?

S3 03:32 No relationship between body weight and activity. No one has been able to show that.

S1 03:36 Yeah, and that's [in?] some big human studies as well, so that's not surprising. Okay. So we overfeed them, humans, and they become less active. We don't know why?

S3 03:45 No, we don't know why.

S1 03:46 So how'd you attack that?

S3 03:48 So we had previous work in our lab to show that the primary sex hormones, being testosterone in males and estrogen in

females, has a regulatory effect on activity. Interestingly, overfeeding alters both testosterone and estrogen in males and females--

S1 04:06 In humans.

S3 04:07 In humans. So then we became interested in whether the sex hormones may be mediating the effect of overfeeding on reduced activity.

S1 04:17 Okay, so the idea was that if you overfed them then it's going to alter sex hormones and kind of change their physical activity as a result?

S3 04:26 Correct.

S1 04:27 Is that why they advertise so many low tea products on TV?

S3 04:31 Possibly [laughter].

S1 04:33 We don't know. Maybe. Okay. All right, so how did you do this?

S3 04:37 First of all, I had a study that was three-fold. The first part of the study was to simply overfeed mice with a high-fat, high-sugar diet that resembled a typical western diet that people have here in the US and--

S1 04:51 It was like, give them soda and high fat, wasn't it?

S3 04:53 Yes, they had a 20% fructose solution in their water, which is typically, on average, what humans are taking in on a day-to-day basis. Their percent fat in their diet was around the same, and when you give this type of diet to mice, they overeat and they become fat. So the purpose of doing that was to see, if you overfeed those mice, what happens to their activity. We measured activity through wheel running, and mice are like humans. They get on the wheel on a regular basis just like humans will go to the gym. So what we did was we overfed the mice for a series of nine weeks, and then we provided a running wheel for three days to see what would happen to their activity as a result of the high-fat, high-sugar diet.

S1 05:43 Okay, so you built us up now. What happened?

S3 05:46 Their activity decreased.

S1 05:47 By how much? A lot? A little?

S3 05:49 Around 50-70% in both males and females.

S1 05:53 Wow. So they really became couch potatoes when they ate that high-fat, high-sugar diet.

S3 05:58 They did. I actually had one, he-- a little mouse. He chewed his wheel off and he made a little bed out of it.

S1 06:04 There you go. So he really made sure-- it's like the treadmill in the bedroom thing where you put clothes on it so you can't exercise on it.

S3 06:10 Exactly.

S1 06:11 Yeah. So you talked about the sex hormones. Did that have any role in this when you tested that?

S3 06:16 So that was the next part, and we measured the sex hormone levels in the males and females. So again, the primary in females is estrogen, primary in males is testosterone, and so we measured those in each of the males and females, and we surprisingly found that neither was altered due to the diet.

S1 06:37 So diet didn't change-- even though diet changes it in humans, diet didn't change it in the mice?

S3 06:40 It did not change it in the mice.

S1 06:42 But their activity still went down, right?

S3 06:43 Activity still went down.

S1 06:44 So what did that tell us? What did that tell you about?

S3 06:47 It tells us that overfeeding is decreasing activity, but it's doing it independent of the sex hormones.

S1 06:54 So it's something else going on?

S3 06:55 Yes. Something else is happening.

S1 06:57 Okay. So did you leave it at that? Were you done and walked away and said, "Okay, I've proved my point and I'm done."

S3 07:03 No. I bought a few more ELISA Kits just to make sure [laughter] I was not having any error, just to make sure that the sex hormones were not altered. So that was tested multiple times, but--

S1 07:14 So you know everybody but two people out there listening are going, "What the heck is an ELISA Kit?"

S1 07:18 ELISA Kit. So what that does-- so we take blood from the mice, and from that we extract the serum, and that contains these sex hormones, and we just run a Kit just to determine what the concentration is approximately in the mice.

S1 07:35 So that's just the way that you measure sex hormones.

S3 07:38 Yes, the way we measure sex hormone levels.

S1 07:40 Excellent. All right, so you did a lot of that to make sure you weren't incorrect, right?

S3 07:45 Right.

S1 07:45 So you tested that a whole lot. And at that point, you just wipe your hands and go, "Okay, I've proven that we decrease activity and it doesn't do anything with sex hormones," and walk away?

S3 07:55 No, I had to do one more experiment.

S1 07:58 We always have to do one more experiment, don't we? Okay.

S3 07:59 Yes. So that experiment involved giving a high dose of estrogen to females and a higher dose of testosterone to males in the mice that were overfed. The purpose was to just make sure that if you give them a higher dose than is physiologically normal to these mice, does that alter their activity? As a confirmatory measure.

S1 08:28 Well, did it? We're waiting for the punch line here.

S3 08:30 It did not. It did not. It did not. So they had a higher dose of the sex hormones, and their activity was not altered as a result. So given we did not see their sex hormone concentrations change as a result of overfeeding, and then supplementing them with additional testosterone and estrogen didn't change it, that was our indication that overfeeding was not decreasing activity through the sex hormones.

S1 08:59 So you really did show through several replications that overfeeding decreased activity, but in any way possible, you didn't see that affecting sex hormones.

S3 09:08 Right.

S1 09:09 So how's it working?

S3 09:10 Well, there are a lot of effects that overfeeding has on the body, on metabolism. There's different hormones that are altered, and those things need to be investigated further.

S1 09:22 So if you had to place a bet right now, someone said, "Heather you have to bet on what mechanism is," which one would you bet on? Audience, that pause is because scientists don't like to speculate.

S3 09:34 I haven't decided, but I-- my gut instinct is going more towards something with the mitochondria that may be involved.

S1 09:43 Excellent segue because that's what you're now doing research on is the mitochondria, right?

S3 09:48 It is.

S1 09:48 So tell everybody what mitochondria is, number one, if they don't know, and then why is it important?

S3 09:54 So the mitochondria is your primary-- your powerhouse. That's what regulates your cellular metabolism, so food that you eat. It's what regulates how that food is utilized for different types of-- your heart to function, your lung to function. When you go out and exercise, that's your primary energy source.

S1 10:16 So these mitochondria are in all your cells--

S3 10:18 Correct, yes.

S1 10:18 --in your body, and they help you make energy.

S3 10:20 Yes.

S1 10:20 All right. So are they different?

S3 10:23 They function different in different types that is dependent on the tissue that they are in.

S1 10:28 So they can be different?

S3 10:29 Yes.

S1 10:30 And so your current research right now, you're actually genetically sequencing--

S3 10:34 Correct.

S1 10:35 --these mitochondria. So tell us about that.

S3 10:39 Okay. All right. So mitochondria, they have a separate genome from our nuclear genome.

S1 10:46 So we need to really clarify that for people. That's not part of the human genome. That's a separate genome. In the past, biologists have thought that mitochondria and cells actually evolve separately, and then at some point then they joined together to help each other, right? So that's why the mitochondria has a different genome than a human has a genome, right?

S3 11:08 Right.

S1 11:09 Okay, cool. So you're sequencing the genomes.

S3 11:11 Yes, and so what we are looking at right now is determining how mitochondrial DNA sequences affect your susceptibility to different types of diseases and then how that sequence may be altered due to different types of exposures that you may have [crosstalk].

S1 11:31 So what kind of diseases you talking about?

S3 11:33 This could be a wide variety of diseases.

S1 11:36 So you're talking about the type of mitochondria that each of us have may predispose us different kinds of diseases?

S3 11:42 It can, and this is just dependent upon the sequence of the mitochondria.

S1 11:48 Cool. Tell us more about this, this sounds interesting. So this sounds like we need to all go find out what kind of mitochondria we have [laughter].

S3 11:54 So the different types of sequences, there's a term that we have called heteroplasmy, and this is when the DNA-- you have multiple copies of these DNA sequences within the mitochondria, and when those become different, that's when we call that a heteroplasmy. And there are certain thresholds of heteroplasmy in certain tissues, and once that's reached, that can cause or put you at risk for a disease.

S1 12:26 Any particular diseases that you're working on right now?

S3 12:29 I have a couple of different things that I'm looking on right now as some are just your genetic susceptibility. So looking at how your genetic background may be influencing different types of mitochondrial sequences, for example, in the heart, looking at how that may be regulating your heart rate and heart rate variability, and that's important when we look at your risk for cardiovascular disease.

S1 13:00 So if you find that there's these sequence differences make a difference, is there any way we can fix it?

S3 13:07 That we don't know yet. That's something that I'm really trying to work on. It will be more of a preventative-type thing.

S1 13:16 So it'd be one of those things that you--

S3 13:16 [crosstalk] how you alter that.

S1 13:17 Yeah. You can have your mitochondria sequenced and they say, "Oh, you're at risk for, say, heart disease." And you'd have to really think about you eating a preventative diet, being active, not smoking cigarettes, and doing other things that may put you-- susceptibility for cardiovascular disease.

S3 13:35 Right. Yeah. One thing we don't know is how-- we know some of these DNA-- the mitochondrial DNA sequences are different, but we don't know what that means. So what does that mean for the function of the mitochondria? What does that mean for disease susceptibility? This is a very new area that's starting to be studied.

S1 13:57 Yeah, and that's one of the reasons that we really wanted to have you on, because this is a really brand new area. You're working with someone named Dr. Steve Kleeberger, who we've had on the podcast in the past who's done this stuff. So it's really kind of cool, this is kind of the new and upcoming area. So what makes you excited about all this as you think about this stuff? What is it that excites you about it?

S3 14:17 It's applicable to so many different things. A lot of different diseases. I have, goodness, three or four projects going on right now that are all centered around mitochondrial DNA sequences, but other than that they're not really related, other than being disease states.

S1 14:36 Yeah. So that's what makes you excited about that?

S3 14:38 Mm-hmm.

S1 14:39 We always ask our guest, I guess to extend this, we always ask our guests how they got to where they are. So you kind of-- I told the audience a little bit about your background. Is this something that when you were like five years old you said, "Jeez, I'm going to study mitochondria one day." The teacher asked you in school, you said, "I'm going to be a mitochondria physiologist." How did you get there?

S3 15:00 Never thought that. It was a long road. A lot of different-- just things evolved. Going back to undergrad, I started off. I fell in

love with exercise science, loved working with older adults. Then I thought I wanted to go into cardiac rehab. I worked in a cardiac rehab facility for two years and found out that I needed a challenge and couldn't take the day-to-day same thing. And then I looked into getting a PhD, and I ended up at Texas A&M. And through going to a seminar that Dr. Kleeburger came to about a year and a half ago now, hearing some of his work with the mitochondria, I became interested in that. And again, it's just a lot of opportunities that have opened up and just how things have evolved.

S1 15:57 Just one of those things. You got to stay open for all those opportunities.

S3 15:59 Exactly.

S1 16:00 Cool. So is that would you would tell people who said, "Hey, I may be interested in doing what Dr. Vellers is doing down the line." What would be your word of advice for them?

S3 16:11 Take advantage of opportunities that come your way, and don't be scared to take risk.

S1 16:17 Yeah. Don't be scared to work hard, too, right?

S3 16:20 Right.

S1 16:20 Yeah. Because none of this stuff is easy.

S3 16:22 No.

S1 16:23 You know, we talk and we say, "Oh, we went in the lab and we sequenced the genome of the mitochondria."

S3 16:28 It takes so long [laughter].

S1 16:30 How long does it take to do that?

S3 16:31 I've been working on it since October and we're not even half way done [laughter].

S1 16:42 So if you take a sample, let's say you get-- I'm thinking about mitochondria, so it depends on what tissue you're pulling from. So you have to have a tissue sample. So let's say you had a piece of muscle tissue from a person-- or a blood sample. A blood sample from a person. How long would it take you to take that and wind up having mitochondrial gene sequencing?

S3 17:03 I would say it would probably take you at least six months.

S1 17:09 If you've got all the tools.

S3 17:12 If you have all the tools and then you have to also have a lot of money to do it. A big part of this is not only the manual labor that goes into doing many experiments but the bioinformatics portion of it. How do you analyze the information? I mean, you're talking about downloading a file that will take 10 or 15 minutes because the data is that massive, and how do you analyze that data? So that takes a very long time. Very complex.

S1 17:45 So this is something that you just didn't go into your garage and whip up and you've been doing on the side?

S3 17:51 No [laughter].

S1 17:53 This is something that has to be done in a big laboratory, right?

S3 17:56 Multiple laboratories, yes.

S1 17:58 Yeah, and I think that's the problem with sciences now. Sometimes people don't think about how hard it is to do at times, and it is difficult to do. Well, it's cool that you're doing that.

S3 18:08 It's fun.

S1 18:09 Yeah, it's fun. All right. So we're running out of time and so, as our regular listeners know, we usually give our guests an opportunity to give us a take-home message of the week. So if people were to listen to this podcast, when they listen to this podcast, what is the one thought you'd like for them to remember?

S3 18:30 To take advantage of opportunities that you are passionate about because that's what's led me to where I am now, and I would not go back and change that.

S1 18:42 Cool. Great take-home message. Thank you for being with us today.

S3 18:45 Thank you for having me.

S1 18:46 You're welcome. And thank you all for taking the time to download and listen to us today. And again, regular listeners know that this is the time of the podcast when we always put the podcast question of the week in and today with the podcast question is our Executive Producer, Kenneth McIntyre.

S4 19:02 What is the origin of the mitochondria in human cells?

S1 19:06 Great podcast question, Kenneth. Be the first person to send us an email to hufffines@tamu.edu with the correct answer

and you'll win one of those nifty podcast t-shirts we have. Again, send that email to huffines@tamu.edu. Don't think you're too late because we often give more than one t-shirt. [music] So send in your answers. And also, we hope that all of you take the time to join us next week again. We have another interesting person in the world of sports medicine and human performance and until then, we hope that you're active and healthy.

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