Paleo Solution - 367

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Robb: Hey, folks. Robb Wolf here, back again with another edition of the Paleo Solution podcast. Hot on the heels of the most recent Paleo f(x) Nerd fest wing ding extravaganza, got to hang out with one of my good friends, Dr. Michael Ruscio and got him liquored up pretty good. And he, in a moment of weakness, agreed to come back on the show. How are you doing?

Michael: I'm doing good, Robb. It doesn't take too much to get me liquored up. It's a pretty easy elbow twist. But, yeah, that was definitely good hanging out at Paleo f(x). It's good to be back.

Robb: And by easy, that's mainly just that the booze goes down you easily. It takes a fair amount to make an impact on a strap and a hunk a man such as yourself but it was really good to get to hang out. What were your highlights at Paleo f(x) this year?

Michael: I'm glad you asked it because I was thinking about that before you happen to call and one of the things that I was most happy to see was -- I sat on the gut panel last year and I sat on the gut panel this year. Last year, I almost felt kind of like the dick on the panel because I was kind of squashing some of these overzealous excitement that was budding regarding the microbiota and some of the microbiota tests and trying to feed the microbiota with prebiotics because we are thinking that the microbiota is like the ultimate driver of all disease.

This year, it was really nice to see the general consensus of the panel was shifting more into the direction that I had last year and a little bit more bridle, a little bit more conservative, a little bit more "this is interesting stuff but it's really pre-clinical and it's academic and we shouldn't be using this information to inform or guide healthcare decisions." So, that was one of the cooler things that I noticed from being on the panel.

Robb: Nice, nice. And I definitely touched on that in my explorations of quantified self. Like Nicky, as usual, she manages to kick me in the balls whether I'm coming or going, my bubbly wife, and she's like, "You know, the way you started your talk was kind of a buzz kill because you basically said you wanted it to be something big and over the top and then it's not going to be and so--" And she hates that stuff.

But I really did. I was kind of like, "Okay, quantified self. This is going to be amazing. It's the matrix. It's where we're going to learn all the stuff." And I've just tried over the last year to really find what is worth taking away and it's like

heart rate variability is cool, blood sugar monitoring is cool, doing some stuff that really gives you a deep insight into your insulin sensitivity, insulin resistance, tracking a few metabolic substrates like ketones and blood glucose.

But even all that stuff needs a really good -- I use the telescope analogy. Like you can grab all this big information with that front lens of the telescope but then if you don't have this orienting lens at the back it's just all bullshit. It doesn't tell you anything and it makes people more confused. And one of the most confusing areas for, I think, everybody right now is this gut microbiome story.

Clearly, it's a huge deal. And I think equally as clearly we just still don't really know what the heck is going on. Like I know for you, you have some clinically relevant protocols to help people move through the whole thing. But, I mean, correct me if I'm wrong but a lot of this is not quite -- It's coin toss and then it's like, "Well, if you respond this way then we know we're probably dealing with X. If you respond this way, we might be dealing with why or it might be Z and I don't know what Z is and we're going to have to keep working." Would you generally agree with that?

Michael: Yeah, I would generally agree with that. It's actually quite simple if you have the right approach in terms of how you analyze information. To be candid, the problem is you have people on the field of the microbiota speculating from information that is not clinical. They're looking at observation, they're looking at mechanisms. This is the allure of information. This is the major problem on the internet, "Oh, because we gave a rat this prebiotic and they increased their short-chain fatty acid and short-chain fatty acids are anti-inflammatory, we should all take prebiotics who have intestinal inflammation."

No. Because when we give prebiotics to some people with inflammation, they actually get worse. So, when you filter your information through a clinical or when you look at clinical information, it's very easy to see the writing on the wall. And this is why I've had a different opinion on a lot of the gut stuff than many others because I didn't get swept into a lot of that allure of information. It was the same thing I saw early in my career in exercise. I saw the same thing happening where people get sucked into this very sexy detailed academic information and they forget to just come back to the practical clinical information. And when you focus on clinical, you don't really get misled.

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So, what to do isn't that difficult if you focus on the right type of information. And, yes, I've kind of worked this into an algorithm and it's not really that difficult. The broad stroke is the healthier you are the more you can afford to feed your gut microbiota and the less healthy you are the more imbalances that you have in the gut the more you may need to focus on an approach that kind of

keeps the microbiota trimmed.

And it's so comical to me that some people have such a hard time understanding the concept that a diverse microbiota is not only arrived at when you eat a lot of fiber and prebiotics. It's a silly concept and it's flawed for so many reasons. I think your experience, Robb, from what you were telling me at Paleo f(x) is totally in alignment with exactly what I'm describing, which is you have a history of inflammatory bowel disease and I think you tried maybe like a year or so ago some of these feeding interventions like really some starch and whatever else.

And from what you told me, that didn't really work too well for you. And then eventually you ended up doing a protocol, I think, yeah, it was passed along via a mutual friend, one of my protocols, that is nothing exotic. It's just more so an approach that focuses on reducing the shrubs or trimming the microbiota and you responded beautifully. So, you fall right into that typical kind of presentation.

Robb: Yeah. And it was, we still haven't fully unpacked all that but you had long suspected that I might have some fungal biofilm type stuff going on and so part of the protocol effectively addresses those issues. I got to tell you, it was literally like a light switch flipping kind of event. It was like, holy smokes, just like my poo was better, my blood sugar control was better. I mean, it was kind of crazy. It was not a subtle effect. But it was so powerful that for the first week of it, every single day when I got up, when I get ready to do my business, I had like triple fingers crossed. I'm like, "Please poop like a teenager. Please poop like a teenager."

And everything has been good. It's funny the only disruption I've had in maybe the two weeks now that I've been playing with this protocol, got super tired at Paleo f(x), not enough sleep, but even then things hung in there pretty good. But then we went to a Thai restaurant and I begged these guys to kind of dial the heat down. Like I can do a little heat but not a ton of heat. Basically, about 2:30 in the morning after eating lunch that day, I was sitting on a column of flame for about an hour, getting all that purged up.

I wasn't quite right the following day. That capsaicin was really not a good fit for that particular situation. But then I got to say the day after that I bounced right back. Everything was right back in line. And so definitely a more resilient system. But the protocol was not a feeding protocol. It was largely a weeding element and then also addressing, kind of introducing some antagonistic elements to potential yeast. So, very interesting and very, very different, and nothing that's being discussed by anybody that I've tracked down other than yourself. So, huge, huge hats off to you on that.

Michael: Thank you. Yeah. When you look at the clinical literature, you figure that out

pretty quickly. What I mean by that specifically is when you look at trials in different subgroup of patients, those with IBS, those with IBD, those with diabetes, those with heart disease, you start seeing a general trend where many people are going to do better on a bacterial trimming approach. That is so confused by all this observational data that we have showing that healthier populations have more robust microbiotas.

Well, I think in the large part, we're going to find that it's not the cause of their health. It's a byproduct of their health. This is probably why when people exercise they have improved microbiotas, when type 1 diabetics go on insulin they have improved microbiotas, when people with rheumatoid arthritis go on anti-inflammatory medications they have improved microbiotas. Even when people go on an elemental diet, which by definition starves the microbiota, they have increased diversity after that according to one study that we have.

So, I think that the main problem here is people who are -- and I'm using these terms loosely, but people who are healthier probably have healthier immune system calibration.

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And that allows a more robust microbiota to live in the gut. People who are less healthy have less attuned immune systems and they don't get along with the gut microbiota so they have a less diverse microbiota. And so, when you look at that, when you look at the end result of that, healthier people have more diverse microbiotas, less healthy people have less diverse microbiotas. Therefore, we should feed the microbiota.

That's a reductionistic -- That's everything that we criticize in this community. It's being reductionistic. It's just looking at things in a narrow tunnel. And we always criticize that in this field. We want to be holistic and multi-faceted and what have you, except that's been told it's been out the window, I think, because, "Prebiotics and fiber are natural, man, and they're cool." And so if we use those, even if we're using natural things in a reductionistic model, people tend to miss the fact that we're using it in a reductionistic way, which is why, I think, we're not really seeing the results that we'd like to see.

Robb: We want to see, yeah, yeah.

Michael: Some people do respond but there's definitely a strong subset of people. And probably the people who need the help the most are the ones who are the most harmed by that miss there.

Robb: That's a really, really good point. Let's maybe unpack a little bit of that diversity stuff. So, Jeff Leach, absolutely love Jeff. He's an amazing guy, great researcher.

He's heading up this human gut project. They're working with the Hadza. They're sequencing their gut microbiome, they're hunting with them, they're living with them. Jeff did an infield turkey baster fecal transplant, which we still haven't heard what the results are with that. He is alive. So, we know that there's at least that part of it.

And again, because the internet and social media seems incapable of understanding that we can be colleagues and friends but yet not necessarily agree on everything. I'm not attacking Jeff. I just have to throw that out there because god damn it, people seem incapable of any nuance on this thing. But Jeff has thrown out some blog posts, something to the effect of, "Hey, low carbers, your gut microbiome is just not that into you." We could talk about that.

Michael: Yeah. Let's talk about that.

Robb: The limitation that these folks on low carb have a much less diverse gut microbiota, the Hadza, have a much more diverse microbiota and so there might be some problems there. But, I mean, you really kind of unpack that just now which is just that the Hadza for any one of a number of reasons including that they were vaginally birthed, breastfed, have probably never seen an antibiotic, don't drink chlorinated water, et cetera, et cetera, et cetera, et cetera, probably are parasitically infected with interestingly seems to have an immune tuning effect in the gut.

We've seen some instances of like various pinworm administration actually helping inflammatory bowel disease in some people. There's a million different variables there but it's definitely being put forward that this is the gut that we want to emulate. And maybe if we were able to engineer the whole story from birth starting forward, that might be true. But if you're broken, it might not be true. Could you unpack more of that?

Michael: Yeah. I mean, that's a great lead in, Robb. That's absolutely true. I mean, there's one or two kind of broad philosophical notes we can paint and then kind of filter underneath that some of the details. But, yes, we can't take one aspect of an entirely different culture and force that one aspect into someone not in that culture and expect the benefit. That's like Science 101.

Now, I appreciate what Jeff is doing and that's an important part of the scientific process. We take what we learn there. We look at mechanism. We look at observation. We speculate as to what we might be able to do for westerners with that. We then start some animal experiments. And if those don't do nasty things to the animals we then move to human experiments. And we're going to find a lot of what we thought was right was wrong. It's just the nature of science.

But we'll come along with a few things that will be helpful. That's what will

eventually happen. Right now, to take any of that information and draw clinical conclusions or, meaning, you do stuff in your life differently is way premature. And there are just so many ways I can destroy that argument. Don't get me wrong. I don't say destroy that argument like I'm dogmatic on my views. My views had been shaped by very objective purview of the literature and being humble and thoughtful in my analysis. So, I am totally fine with updating and evolving my opinion on these things as the information evolves and changes.

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However, my opinion is based upon, again, a thoughtful analysis of all the data of both sides. A simple example of this would be looking at different ecosystems across the world. This is something I talk about in my coming print book that I think you used as a lining of your bird cage, Robb. But if we look at ecosystems across the globe, we can see a few things. Let me paint one component of this.

So, let's say in this ecosystem analogy that fiber, carbs and prebiotics are like rain because rain feeds vegetation and carbs, fiber and prebiotics feed your gut bacteria. Now, if we went to the rainforest we may notice there's beautiful vegetation and there's a lot of rain. And that may leave some of say, "Gosh, there's such beautiful vegetation here. There should be a lot of rain in every ecosystem."

So, what if we take a rainforest level rain and just force that into an arid environment like that of Southern California. You create mudslides. People will die because of the damage, and it would wreck havoc in that ecosystem. So, just because we see a level of rainfall that's healthy for one ecosystem does not mean it's going to be healthy for another ecosystem. Now, how that analogy translates to modern day is for a Hadza hunter-gatherer to eat high fiber and carbohydrate and prebiotic that feeds this very rich microbiota is helpful. It fits.

If we do that for you, Robb, as you learned, or myself or many people in Western societies, that's like taking a rainforest level rain and just forcing that into an arid climate like that of Southern California and that's going to cause a lot of problems. So, if we think about it, hopefully anyway, the ecosystem analogy is helpful to realize that just because a lot of rain is good for one ecosystem doesn't mean that a lot of rain is going to be helpful or healthy for every ecosystem. Are you kind of with me on that?

Robb: Yeah, yeah. It's a great analogy.

Michael: So, there are, of course, clinical examples of that. We know that people with IBS, irritable bowel syndrome, which typically looks like gas, bloating, abdominal pain, loose stools, diarrhea or the other way, constipation, we know that they tend to respond very favorably to a low FODMAP diet. And low FODMAP diets do

display a degree of reduction of the microbiota. But what's interesting is that that degree of reduction of the microbiota has also been correlated with improved symptoms, less inflammation and we have even done trials where we've taken people, for example, with Crohn's disease who were quiescent. Meaning, they were in remission. Meaning, they were fine.

And then we put them on a higher FODMAP diet. Let me back it up. So, essentially what they do is they put everyone in the study in Crohn's disease on a low FODMAP diet. These people are already in remission so they're feeling fine to begin with. They put everyone on a low FODMAP diet then they had half the participants continue low FODMAP. The other half started to take a prebiotic supplement. Now, 80% of the people who stayed low FODMAP, they essentially felt well and they continued to feel well.

For the people that went on prebiotics, 70% of them had a relapse. And that relapse looked like a near doubling of disease activity and increased inflammation. This is one example. But if the prebiotics, and the feeding your gut microbiota is so good for you, we wouldn't see things like this happening. Let's look at some other information. When we look at comparative trials that compare a traditional high carb low fat diet, like your traditional health advice, eat lots of healthy whole grains, lots of fruits and vegetables, your standard American heart disease association diets or your kind of high carb in a training diet or even the vegetarian diets.

When we look at comparative trials looking at standard dietary advice, which is very much in alignment with, I think, Leach's recommendation which is lots of fiber, lots of carbs, what have you, when you look at trials looking at those types of diets compared to Paleo or low carb, for cardio and metabolic conditions like heart disease markers and weight loss and waist circumference, we see all diets are helpful compared to no dietary intervention. But there is a clear edge for either Paleo or low carb diets when you examine these comparative trials.

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So, again, if feeding the microbiota was so important, why would we be seeing all this data showing that a Paleo or a lower carb type diet works better for things like cholesterol, insulin, triglycerides, weight loss, waist circumference when we know that these diets, as Leach correctly sites, tend to be slightly lower in fiber and prebiotics and slightly less feeding of the microbiota. So, there's all these examples of why this can be a bad idea.

One more example would be looking at Africans. They have the highest colonization density of Methanobrevibacter smithii which is a methanogenic archaea which is one of the organisms that causes methane SIBO. For them, it's actually a positive adaptation because this methanogen, this advanced bacteria--

Robb: It slows gastric emptying, right?

Michael: Right. It slows down gastric emptying which in a society that's not eating a lot of calories and is eating a lot of fiber in those calories and fiber is laxating and hard to digest, it actually slows down the transit so that they can extract more calories from their food. So, for them, it fits. Now, you do that in Mary Sue from California and she gets bloated, constipated and she gains weight. This is why we've seen a correlation between methane positive SIBO and weight gain and high cholesterol levels.

Give it to you straight for a second here. It's idiotic to make some of these speculations saying that the Africans had this happening so we should do this to ourselves. It would be prudent to learn what we can from them and try to use that information to glean a clinical recommendation. But making clinical recommendations right now from that is so seriously misguided that it's admittedly irritating to myself.

Robb: Yeah. I just posted some pictures of both adult and child Hadza and they had this super distended bellies indicative of this super high fermentation rate. We're kind of putting that forward is kind of a laudable state of affairs but it's interesting when you look at pre-agricultural horticultural societies that seem to get arguably potentially a more nutrient dense dietary intake, possibly less fibrous dietary intake. You don't see that process. And again, it's not to say that necessarily that is bad and the other one is good depending on the individual and depending on what specific state of affairs are.

Michael: Exactly. And you also make another really important point in your comment from earlier, Robb, which is they have a totally different environment from in utero all the way up to adulthood. And that's going to have a major impact on one's immune system. And that's going to have a very strong impact on their microbiota. Not to mention that when we look at hunter-gatherer diets worldwide, and I think we've talked about this in the podcast before, but when we look at hunter-gatherer diets worldwide, of course we have a latitudinal change in the macronutrient consumption of the diet. From the equator to 30 degrees latitude, we see a high carb low fat diet.

And by the way, this is according to some pretty compelling anthropology literature that did a worldwide assessment of hunter-gatherer and macronutrient ratios by Cordain. So, zero to 30 degrees near the equator we see a high carb low fat diet. When we go from the 30 degrees to 40 degrees, the Mediterranean region, we see a balance of fats and carbs, kind of like the Mediterranean diet. And then when we go to plus 40 degrees latitude, we see a lower carb diet.

The problem with some of Leach's -- Again, like you said before, it' not a criticism of Leach. But most of the data that we're seeing looking at modern day hunter-gatherers is coming from a very equatorial zone. And in my book, I actually overlay the latitudinal regions and then I put stars on the globe where the samples are coming from. And you can clearly see that all of the, or most of the data from these modern day hunter-gatherers is occurring in a very equatorial region which is skewing what the diet we think is ideal is because we're getting that high carb low fat sampling.

We don't have data from those of a northern European hunter-gatherer societies because there's much less available unadulterated hunter-gatherers in that latitudinal zone.

Robb: So, Reno is 39.5 to 96 degrees north latitude. So, I should be eating low carb.

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Michael: Well, actually, I should make the point. It's not where you're currently living. It's where your genes evolved from.

Robb: Right. Which the bulk of my genes were way further north in that so, yeah.

Michael: Exactly. And there's another component of this too which most of the modern day hunter, the modern day microbiota analyses that are via stool, they're not assessing the small intestine and that's a huge miss because the small intestine represents over 56% of your digestive tract, is responsible for over 90% of caloric absorption, and houses the largest entity of immune cells in the entire body which is why it's more prone to or more impactful for things like autoimmune conditions and leaky gut.

So, that's another massively important miss which is the overlooking of the small intestine. Robb, have we discussed the early hominid kind of face-off between Paranthropus boisei and Homo habilis and how that shaped the anatomy of our intestines?

Robb: You and I have but we have not talked about it on podcast, so let's dig into that a little bit.

Michael: Yeah. Because that, I think, it's an important insight to glean from our evolutionary history. Long ago, when we're looking at early hominid evolution and trying to track back what hominid we evolved from, there were two competing hominids and one of those would become us. Homo habilis became us and Paranthropus boisei became extinct. So, these are two hominids that are living in a similar region at the same point in time.

Homo habilis was an omnivore. He was kind of jack of all trades in terms of eating. His competitor was Paranthropus boisei who was a little different. Paranthropus boisei was kind of like a gorilla. He had this massively powerful jaw, these really strong teeth, and he ate more like a cow. He ate stuff that was just available on the ground, all these grasses and roots and things that the omnivore couldn't digest because he didn't have the jaw structure but also didn't have the digestive track.

Now, why that's relevant is because Paranthropus boisei had more of a large intestine dependent digestive tract, lots of bacteria, a longer gut to break down all this fiber that he was eating. Whereas, Homo habilis was a scavenger and so he was supplementing with things like meat from carcasses that he scavenged. His intestinal tract looked different because he was supplementing his diet with some of these foods that were, what's turned higher quality foods, meaning easier to absorb and not requiring a lot of bacterial fermentation.

His large intestine actually shortened and became, we think, what looked more like our intestines which is having a short large intestine which is where we have all this bacteria, all these gut bugs that are purportedly so healthy for you and are fed by fiber. That section shortened and he became more reliant on the large intestine which is more typical of an omnivorous digestive tract.

Now, when the Himalayan Mountains formed, it actually caused a change in the climate in Africa causing Africa to become generally more arid. And so what ended up happening is Paranthropus boisei, the gorilla-like cow digester that lived in the ground, died because he had one food supply and as it became more arid that food supply essentially dried up.

Homo habilis lived on because of its omnivorous lifestyle. Why that's important is because when we keep trying to make the argument that we need to keep feeding our bacteria and eating lots of fiber like the Hadza, our digestive tracts didn't necessarily evolve eating a super high fiber super high prebiotic diet. Yes, there was definitely a fiber intake there. I'm not trying to make the argument that we shouldn't be eating any fiber.

But we don't want to become overzealous or obsessive with trying to go to this almost vegetarian like level of fiber and prebiotic intake because we can make a pretty safe argument that that's not the anatomy of our intestinal tract. We're definitely more omnivorous. If you look again at the hunter-gatherer data worldwide, many of us do not thrive on a diet that is super high in fiber and prebiotics, more cow-like, which is dependent on the large or a long large intestine but rather more so the small intestine. That's another reason why the small intestine is so important but it's often left out of this dialogue.

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Robb: And so much of that is because we're really not sequencing the bacteria that are in the small intestine when we're looking at this stuff.

Michael: Correct. There may be, and there likely is a relationship between the large intestine and the small intestine but we're still not even sure how to ideally set the parameters for testing of the large intestine. So, we're just so far from being prescriptive and clinical in this, yeah. So, I mean, those are just some of the more salient things that come to mind regarding this topic.

Again, it's not to say -- I am not trying to paint the picture of everyone needs to be low carb and everyone needs to avoid fiber. But I'm just trying to pose the other side of this conversation so that people who do better on maybe a lower carb or a little bit lower FODMAP of a diet don't feel pressure to keep trying to force fiber and prebiotics down their throat even though they're clearly not responding well to that.

Robb: Got you. Got you. That's great advice. Doc, I know you also wanted to talk about some newly kind of discovered or emerging information on gut microbiota, gut health, and thyroid status. I want to be respectful of your time but do you want to jump into that a bit?

Michael: Yeah, yeah. Let's definitely transition over to that. I guess, maybe the 3,000 foot view takeaway on this, before we get into the details, is if you're struggling with a thyroid condition or a thyroid like symptoms, the problem could actually be coming from your gut. And there's a pretty compelling amount of data that supports this.

There is one study that was published recently. They looked at about 1800 patients. And they were trying to determine what the highest corollary or predictor of someone having small intestinal bacterial overgrowth was. They looked at things like intestinal surgery, amino suppressive drug use, acid lowering medication use, and amongst all the factors they found that being untreated hypothyroid was the second strongest predictor, and being levothyroxine therapy was the strongest predictor.

So, this showed us that problems with the thyroid, according to this analysis, were some of the most predictive indicators that you would have small intestinal bacterial overgrowth. But it's not because of being hypothyroid. Because the people that were on thyroid medication actually had a higher risk than those who were untreated hypothyroid. So why use this? Why do we use this association? It may be because there's something that is happening in thyroid disease independent of the thyroid hormone levels that also is associated with small intestinal bacterial overgrowth. I'm speculating here, but this may be one of the reasons why. Intestinal bacteria have an affinity for selenium. And so

there's been some research that is pondering since intestinal bacteria essentially feed on selenium.

An overgrowth may exacerbate deficiency. And selenium deficiency is definitely a cause, an environmental factor that contributes to thyroid autoimmunity. So, it may be that for some reason those who form SIBO, or not for some reason, those who form SIBO become more deficient in selenium and that opens the door for thyroid autoimmunity. That may be the reason why we're seeing this association.

Robb: Interesting. And, yeah, it's funny because when you mention the hypothyroid was number two and then the individuals experiencing thyroid application were number one, that was super counterintuitive to me. That's really interesting. This is one of those situations where even supplementing selenium could potentially get someone in deep water if the kind of sequencing isn't correct. Is that right?

Michael: I think we can make a decent case for using selenium but if it's just selenium alone, I don't think there's any data that shows that selenium could feed small intestinal bacterial overgrowth. We may see that at some point but I think if that were the case you would see in the -- And this is where -- Okay, let me take a big step back and just give people like a bullshit detector tip here.

If someone is making that argument and then they say, "You should never take selenium because it could feed your SIBO," that is -- All that does is just introduce stupid shit into the space. Now, I'm really being pointed in that but I say that because I get so frustrated with patients that get these erroneous, totally off the mark takeaways and they come into my office living like a nut ball.

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Because of all these little things that the person speaking or writing a blog was trying to put forth some interesting information but they didn't realize that people hang on like every word that you say. So, you have to be very discerning. So, I don't think that people taking selenium is going to exacerbate SIBO because I always filter things through what happens in clinical trials and I have seen none of the clinical trials using selenium there being reported a high incident of adverse events that are digestive in nature.

So, there was never reporting of an exacerbation of bloating or nausea or what have you. I mean, I may be proven wrong in that but I would think that in the selenium trials that many clinical trials have been done, there's going to be a lot of people who have SIBO in those trials and if selenium made SIBO worse we'd see a flaring of digestive symptoms.

Robb: Got you, got you.

Michael: But regarding selenium, there's a couple important things that I wanted to touch on. I mean, there's a few other gut-thyroid things I want to come back to but since we're on the selenium right now let's jump into this. Selenium can, may decrease thyroid autoimmunity. So, one of the most common recommendations in the natural health space for thyroid autoimmunity is supplementation with selenium. Have you heard that, Robb?

Robb: I have, yeah.

Michael: And that's all fine and good. But let's take a look at what the evidence really shows for selenium rather than just saying, "Hey, I'm really into natural health. A vitamin should be the cure for every ail because I'm really into natural health so let me just look at the data that supports selenium being good and ignore all the contradictory information."

We want to have an objective sampling of literature here. So, when we look at the high level data, probably the best study that has been done here was a systematic review with meta-analysis via the Cochrane database. The Cochrane database essentially just filters for bias. And then a systematic review with meta-analysis just looks at all the available clinical trials and tries to summarize them. It's a beautiful way to get an aggregate opinion of what the literature shows.

Now, this Cochrane database meta-analysis essentially showed there's no consistent benefit with selenium supplementation for thyroid autoimmunity. So, there was not shown to be any significant benefit when all the studies were weighted using selenium. Now, that will make some natural health providers like lose their shit. Hang on a second, let's just paint the caveat here. But I would also say that if me saying that makes you feel mad, I would reexamine how dogmatic--

Robb: How objective you are, yeah.

Michael: Yeah, exactly. How objective you are. If you read the study carefully you see that when you break this down the most benefit from selenium was shown in three-month trials. There was less benefit when it was used for six months and when you go to trials that use selenium for over six months the benefit essentially drops off.

So, what this showed us is that selenium has its most utility in a short term repletion window. Why that is relevant is because some people who become diagnosed with Hashimoto's think they had to take a clinical dose, like 200 milligrams, of selenium every day for the rest of their life. And that's simply not true. You should use selenium for three to six months and then essentially stop.

And if you haven't seen a benefit in your thyroid antibodies, you may be one of the people that do not see an improvement in your thyroid autoimmunity from selenium supplementation.

Robb: Got you. Wow. That's good stuff. Well, Doc, again, I want to be respectful of your time. Do you have any -- Because this stuff is complex, shall we say super complex, but what do you recommend that folks do at this junction, like in my talk for PaleoFX I made this point to use blogs and social media and whatnot up to a point with some very strong caveats. In a completely self-serving fashion I recommended something like Wired to Eat, 30-day reset, seven-day carb test, get some granularity. For maybe 80%, 90% of people, that's going to be most of what they need. But then if and when folks run aground then I'm recommending that they need to seek out a practitioner such as yourself. What are your thoughts around that? What's the triage process least harm least intervention? How should folks be tackling, unpacking this whole health issue?

Michael: I think that's really well said, Robb, which is use each resources that we have. They're amazing and great.

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But use them cautiously. And I couldn't agree with that more. And perhaps I have a slightly biased view of people who have been using this information and not able to become successful at using it and so they come see me. But I will say it is disheartening the amount of useless testing people purchase or unhelpful supplements that people purchase or unnecessarily restrictive diets that people do when they don't follow the recommendation that you just made which is the using the information on the internet prudently.

I totally agree with that. Yeah, I would again agree with that. Use the information that you get on the internet to do some self experimentation and if after a little bit of experimentation you're not really making noticeable gains then I would turn things over to a clinician. My book, I think, will be a pretty darn good self help plan for improving the health of one's gut because it's not dogmatic. I factor in the whole spectrum of people.

Meaning, if you're moderate to severe IBS or IBD or you just got a really sensitive gut, then you'll end up in one part of the algorithm with a plan. If you're a crossfitter who is in pretty robust health and just has a little bit of constipation, then you're going to end up in a different part of the algorithm. And so, I think, the print book which will hopefully be out late this year but I'm learning that my estimates there are just pretty much meaningless at this point. So, soon. I'm trying to get it out as soon as possible. I think that will be a great resource for people.

And, Robb, I do have a couple other places people can go for some help but I have about 15 more minutes. There was one thing about thyroid autoimmunity I wanted to address just because I think this could be hugely helpful for people and it's important information to get out there.

Robb: Yeah. Please do. I'm here as long as you want to be here. I just want to be respectful of your time.

Michael: Okay. Yeah. Thank you, thank you. So, I guess, just two final things to touch on really quick. The first is a couple other gut-thyroid connecting points. We have shown that those with SIBO also have an increased incidents of H. pylori and after treating SIBO, I'm sorry, after treating the H. pylori SIBO can become worse. So, there's definitely this connection between SIBO and H. pylori where they have to be probably addressed together.

How that connects back to thyroid is that there has been some data, one Italian study most notably that has shown that the treatment of H. pylori has shown the ability to improve thyroid immunity. So, there's definitely this relationship between some of these gut infections and thyroid immunity. Also, people with H. pylori actually tend to respond better, some respond better to a liquid form of T4 medication because it's more easy to absorb.

And so some other reason why people with gut imbalances may struggle to find their ideal dose of Levothyroxine or Synthroid or Armour whatever it is, is because they have impaired absorption. And the problem is not the thyroid per se. The problem is they're not consistently absorbing the dose because of malabsoprtion in the gut. So, that's just a couple important things there for people to be aware of.

And when people are not responding well to whatever thyroid intervention that's oftentimes the autoimmunity that is blamed. I think we've become a bit overzealous with the autoimmunity piece. It's certainly important, it's certainly something we want to be aware of, but I've been saying for a few years now that when I see thyroid peroxidase antibodies or TPO antibodies, which is the most common test used to quantify Hashimoto's or thyroid autoimmunity, when I see those antibodies between 100 and 300, I consider that a clinical win.

Now, above, usually it's 35, depending on the lab, above 35 is considered positive. So, I've been saying for a while, between 100 to 300 seems to be a clinical win. There was recently a study published that showed that if people have TPO antibodies below 500 they are at minimal risk for future progression of thyroid disease.

Now, why that's important is because depending on what you read or the provider that you see, you may think you have a problem with thyroid

autoimmunity when you actually don't. We need to update the way that we interpret and manage the conversation around thyroid autoimmunity. So, again, if above 35, it is considered positive. Here is a way that you could look at this.

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You first learn that you have Hashimoto's. You test your antibodies. You may see that your antibodies are 800, 1100, 1400. Then you change your diet, perhaps you go gluten free or you generally avoid gluten which may be is a better way of describing this and not having everyone avoid gluten in overzealous nature unless they notice they have a really strong negative reaction.

You take some probiotics, you take some selenium, some vitamin D, you take some steps to improve your gut health, and months later you're feeling a lot better, you've lost some weight, you're sleeping better. And you retest your antibodies and now your antibodies are at 325. Here's where we have a real important kind of divergence in the road.

The appropriate way to manage that conversation or that finding is we've hit a clinical win, your thyroid antibodies are still positive, yes, but this is in the acceptable range and you're at minimal risk and you're feeling great, your antibodies have improved vastly, we're done, we'll keep doing what we're doing, live your life, don't worry about this smoldering autoimmunity. Don't live in fear. We don't need to go crazy with other interventions for your thyroid immunity because we've kind of arrived at the healthy endpoint. We're good.

The other way that this can go is people can get roped into doing other -- "Now, we need to do heavy metal detox," or "Now, we need to go deeper into methylation," or "Now, we need to test your home for mold." People can get pulled into this never ending black hole of interventions trying to wrestle those antibodies down even further not realizing that when they've gotten to that level that they're already kind of where we would like them to be.

And so I just mention that because I've seen a few people that have or a number of people that have been told that they had to go gluten free harder or they can't -- "Now, we have to cut out wine," or whatever it is, going to these really extreme endpoints of intervention trying to drive those antibodies below 35. And I really don't think that the data supports that. I just mention that for people who maybe are trying to manage their thyroid autoimmunity to know when you're done and when you have to keep working.

Robb: That's a great point, the diminishing returns versus perfection. It's like calculating the final decimal point, a pi. It's never going to happen.

Michael: Exactly. Exactly. So, that was it. I just wanted get those couple of things out there

because I think those are really important for people to be aware of. We can update our opinion there. And maybe one other thing, depending on how averse someone is in this argument, there's another study floating around there where the authors concluded that TPO antibodies above 100 were correlated with poor psychological and cognitive outcomes.

But here's the problem. This is why I start to read the studies. When you read the study they defined positive as TPO antibodies above 100. So what they are really saying is that people who had Hashimoto's had a worse outcome for cognition and psychological wellbeing. But when you look at the details of the study, those that had the poor mood and cognition, the average antibody level was 1122.

Robb: Holy smokes.

Michael: Right. So, it just reinforces my argument that if you're in the low hundreds, you're probably okay. If you're much above 500 or above 500 then we want to continue to do some work.

Robb: Wow. Well, Doc, thank you so much for doing the heavy lifting on all this stuff. The world has gotten so detailed and so complex with all this stuff. That was a little bit of the takeaway of my quantified self piece, just that we are awash in data. I've been quite sick in the past. When folks are highly motivated to move some stuff forward then that's important, it's laudable. But, man, it's super easy to just get -- It's like an undertow to a big wave.

Like you just get sucked out to sea and you're dealing with all this stuff that really maybe minutia instead of really focusing on those big picture pieces of sleep, exercise, circadian rhythm, community, happiness, and always keeping an eye open for being able to tweak variables a little bit more but definitely keeping an eye on that return on investment. Like what are you giving up to try to gain some incremental increase in performance or what have you?

Michael: Yeah. It's huge. It's really the double-edged sword of the information age, which is you can really kind of mislead yourself. And one of the things that I often say, more so when speaking to clinicians, but if you introduce non-useful information into the clinical process you make the clinical process harder for yourself.

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And unfortunately, I think, really unfortunately, that gut care has probably gotten worse in the past three years because of this bull list of non-clinically relevant information that has been introduced since the clinical process. And so when you introduce unhelpful information into what's already kind of challenging, which is being a good clinician, you just make your job more

difficult. And it's sad that that happens but it's definitely happening.

Robb: Right, right. Well, Doc, thank you again. Remind folks where they can track you down on the interewebs?

Michael: Sure. So, I'm at drruscio.com, which is D-R-R-U-S-C-I-O dot com. And a couple new things that have been happening there. One, let me just first say that my clinic and I am accepting patients, which I'm really happy to say, I've been working very hard to make sure that we keep our doors open because I never want to see the day come when we're not accepting patients. So, that I'm really happy about.

The other is I've recently released a clinical training newsletter. So, it's a monthly newsletter that's written to take all the stuff that we just talked about and really be clinical with it and give people the clinical actionables. And so, that's a monthly edition. There's one case study, three to five research studies that are reviewed, all clinically relevant research studies that really impact the way that we practice, and then one monthly practitioner question and one practice tip.

That's been phenomenal because I've had a lot of practitioners asked, "I like what you're saying. I like this cost effective conservative practical model of functional medicine. Where can I learn more about that?" And I said I don't really know. I mean Kresser has got a good program and there's a couple others, but I want to offer something there. That's been out for a number of months and we've gotten excellent feedback. That's at drruscio.com/review.

And if you want to plug in and follow for the print book for when that comes out, if you go to drruscio.com/gutbook, you can download a free 25-page ebook that I wrote and also plug in to be notified for when the print book is going to become available.

Robb: Awesome. Well, Doc, I'm sure we'll circle back a time or two before the book is released and definitely when the book is released. Great seeing you. Are you doing any other speaking gigs coming up here, AHS or Low Carb USA and anything like that? Or was Paleo f(x) your big shindig?

Michael: No. Actually, I'll be at AHS, yes, and then I'm doing the -- I forget the exact name of the conference but, I think, it's something along the lines of the International Congress on the Microbiome in Brisbane, Australia in June. I think that will be a really cool event to speak at a microbiota conference but, hopefully, be kind of like the voice of reason. Because they said, "We really wanted to bring you on board to kind of give us a clinical perspective on all this really interesting microbiota information but we want to kind of have someone who could maybe ground the conversation and bring it back to clinical perspective a little bit."

I guess, it's a pretty big convention that has a nice blend of conventional and alternative providers. So, I'm pretty excited about that. I've never been to Australia so that should be a terrible flight but a cool place to see.

Robb: Good time to be there, yeah, yeah. Awesome. Well, Doc, thank you again for taking the time to come on the show. Definitely looking forward to the release of the book.

Michael: Thanks for having me back, Robb.

Robb: All right, man. We'll talk to you soon.

Michael: Talk to you soon, buddy. Bye.

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