Paleo Solution - 299

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Robb Wolf: Hey Doc. What's new and exciting?

Dr. Ruscio: Oh boy, just working too much probably, but excited about this event we have coming up in London that I want to make people aware of.

Robb Wolf: So tell folks about that.

Dr. Ruscio: So Melissa Hartwig and I decided to team up and do a two-day event in London, January 16th and 17th. I'm really excited this event because I think it will provide a good continuum of information from diet all the way through Functional Medicine. On the first day, the first 60% of the first day, Melissa will be going through The Whole30 program and all her

tips and tricks for implementing the Paleo diet in a successful and sustainable way.

On the tail end of day one, I'll be talking about what you can do if you've done a diet program and haven't seen the results that you're looking to see and this would be enter Functional Medicine and what some of the first few Functional Medicine interventions and steps you should think about would be. This day one will be for predominantly a lay audience, but definitely, it would be useful for a clinician for review also.

Day two I'll be on expanding on the Functional Medicine concepts from day one and focusing on gut and thyroid. The whole second day will be available for continuing education credits. I think in London, they call it something slightly different, but continuing education credits will be available for the event for day 2 and day 2 will be super clinical and we'll be able to geek out on testing and treatment protocols and clinical algorithms and all that good stuff. So if you're able to stay awake through it, I think it will be a great event.

Robb Wolf: [Laughs] I was just going to say like pack some caveman coffee because you guys are going to be jetlagged. I suspect that January in the UK is probably not the most spectacular weather. I'm going to go out on a limb and say that.

Dr. Ruscio: Yeah, I've heard it's cold and I'm leaving on a plane for 10 hours, I'm afraid I might hulk out and just destroy the entire aircraft so…

Robb Wolf: I'm mildly claustrophobic and there's been a time or two where like my seating got messed up and I ended up in the middle row between two really large people and I seriously had to go to my happy place and just breathe. Like, okay, you can do this. You're not going to freak out.

Dr. Ruscio: Yeah. I've never done a 10-hour flight so it will be interesting to see how I fare with it, but I'm hoping for a small person next to me instead of the huge guy that you never want to sit next to you so…

Robb Wolf: Right, right, which is pretty you.

Dr. Ruscio: I guess which is myself.

Robb Wolf: Yeah. It's basically you. Yeah. Yeah.

Dr. Ruscio: Yeah so.

Robb Wolf: Well, Valium on an empty stomach with a couple of shots of vodka and that will make the whole thing better so.

Dr. Ruscio: Yeah. Just get drunk it would be fun no matter what you do.

Robb Wolf: Exactly.

Dr. Ruscio: Exactly.

Robb Wolf: Doc, you as always, you are Johnny on the spot with everything, poop related. What do we have cooking lately? Like you pinged me some information about celiac and hypothyroid people, folks with IBS have some increased microbial diversity. What's the story with that? Like everything that I'm reading like Human Gut Project and whatnot, were wanting an increase in diversity, which I'm just still trying to figure out like who's on first with all of this stuff, like I have no idea which end is up with the gut microbiome currently.

Dr. Ruscio: Right. Well, one of the main things I wanted to come on the show and talk about and of course will be expanding upon that in London, but it's a lot of information to sift through. I think one of the principles is if you're looking for information and you're excited about the microbiota and you're just wanting a new cure or new treatment or a new lab marker, you can easily find that. But of course, that can be a very erroneous way of thinking because you may miss contradictory evidence or evidence that's important that suggests what you're thinking is not right and I think a lot of that is occurring right now.

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There's so much excitement and we're just seeing all this research, but we're not really sure what to do with it and also to be fair, I think if you read the research papers or you speak with the researchers directly, they are much more tempered in their recommendations and in their conclusions. It's the translation from the scientist to the public that these things really get kind of skewed. So with regards to adversity, I mean, there's a thousand thoughts running through my head right now, but there had been some papers and these are in humans. These are human studies and there have been a number of them. I wouldn't make an announcement based upon one outlier of a finding. But there have been a number of papers that have shown increased diversity in celiac, in hypothyroid, in IBS and this is not just SIBOs. I mean, this increased diversity is increased microbial diversity in some cases and then there has also been bacterial overgrowth as in SIBO reported in many cases of celiac, hypothyroid and IBS.

So it's just an important point to mention because again, so much of the microbiota narrative right now is permeated by increased diversity. We need to increase our diversity. We need more diversity. We need to increase diversity because we're looking at this African populations in many cases and we're seeing that they have increased diversity or there is a smaller amount of data looking at healthy Westerners compared to non-healthy Westerners and we see increased diversity in the healthy westerners compared to their sick cohorts or controls.

But we still really haven't mapped out if this change in diversity is causal or if it's associative. So when we look at -- or one of the ways we can kind of sort this out is by looking at clinical trials where we have people undergo interventions at increased microbial diversity so we can essentially give them prebiotics or we can give them fiber and there have been a number of published studies showing that for example, those that have IBS, if we give them prebiotics and/or fiber, they get worse.

There had been studies showing that in ulcerative colitis and Crohn disease, prebiotics actually may make one worse and there was one initial study that may have shown benefit with prebiotics in ulcerative colitis and Crohn's and then followup trials have shown that it's more likely that one maybe done detriment from prebiotics than done a benefit.

So when we start looking at all these things together, we see that boy, this increased diversity is sometimes found in diseases and then in these diseases, if we administer an intervention that increases diversity, these people get sick. So we're really kind of reinforcing the concept that I don't think we can say across the board we want to increase diversity or increase bacterial counts. Are you with me on that?

Robb Wolf: Yeah, yeah, totally.

Dr. Ruscio: Okay and there is one other that I think is important to mention and that is in… and maybe to zoom us way out. If we're looking at this from 30,000 feet, we want to look at the gut, look at this ecosystem and there is, generally speaking, two directions we can nudge it. We can nudge it in a pro-bacterial direction or we can nudge it in an antibacterial direction, right. That seemed like a kind of a fair assessment.

Robb Wolf: Yeah, totally, like a limiting fermentable carbohydrate, limiting substrates where you can get fewer critters versus feeding it with more.

Dr. Ruscio: Exactly and so if we look at most of the clinical interventions for the gut, I think we have better data for interventions that are more antibacterial. At least when people are sick, when people aren't feeling well, it seems to be a few examine all of the literature starting off with an intervention that's slightly more antibacterial and this maybe a little bit lower fiber especially if people have really flared bowels. It maybe lower fod map, people have SIBO or it maybe antimicrobials or even antibiotics if someone has SIBO or bacterial infection or what have you.

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This approached first when people are sick seems to work really well. Then as the gut health improves and someone stable and improving for several months, then considering an intervention that may feed bacteria is something that people can try, but it's not going to work well for everyone. There are some people and I'm sure some people listening have tried resistant starch. They've tried prebiotics and they felt worst from that. There are also people that may have tried resistant starch or prebiotics and felt a lot better.

Sometimes, I think what happens is people aggregate in camps on the internet that reinforce what worked for them, right, shocker. And then what happens is you see all this people aggregating in the camp that supports them and then a newcomer sees these different camps and as like, boy, it's look like it's either prebiotics and resistant starch and feed your gut bugs or it's low carb, it's low FODMAP.

It's you have too many bacteria and I think that really dose people a disservice because what it discounts is that at first, an antibacterial approach seems to work really well. Then we can kind of titrate up the carb intake and the probiotic intake whether that be supplemental or to the diet and help people to evaluate how much of a -- I guess we would say pro-bacterial lifestyle and diet they have to live or none.

That would mean, are you someone who's going to settle in at a higher carb, higher prebiotic intake or are you some of those who are going to do better on a lower carb and prebiotic intake. So I think that's kind of the big picture view that we pay on this and hopefully, when people look at or hear about the fact that we do see increased diversity in some of these conditions and then when we look the clinical trials in some of these conditions, treating bacteria can make some of these conditions worse.

I should also mention as I'm giving you a very unorganized line of thinking here, in people that are celiac or even I would speculate non-celiac gluten sensitive although this hasn't been published. This has only been

published in celiac. People that are celiac and are none or minimally responsive to a gluten-free diet, one of the first things that should be evaluated next is small intestinal bacterial overgrowth, which of course is then treated with an antibacterial approach.

The same thing happens with ulcerative colitis and Crohn's. Antibiotics and early antimicrobials have been shown to induce remission in those diseases and high fiber intakes at least initially have been shown to make those worse. But with ulcerative colitis and Crohn's, when someone is in remission, increasing dietary fiber intake maybe a healthy thing, which again, kind of paints this canvass of start antibacterial, get the person healthy, give them a little bit of time to I guess settle in or recalibrate or just ride the wave of improvement and then once things settle down, experiment with increasing stuff that feeds bacteria that would be dietary carbs, dietary prebiotics, supplemental prebiotics, dietary fiber supplemental fiber and even resistant starch. So that was a bit of a rant, but was anything useful factored in that?

Robb Wolf: No, no, incredibly useful and what it's kind of making me think of this is this new paper that came out in Cell, talking about the need for personalized nutrition and I just have only read the papers. Tim just stirred in one time, but it had some really interesting stuff in there, like certain folks like they pulled out two different patients. Both of them ate cookies and bananas and I think I talked about this at last week's podcast so I'm still noodling on this.

But both of them, they ate cookies and bananas separately and they tracked the blood sugar response and one person had high blood glucose response to banana. The other person had high blood glucose response to the cookie. What they found though is just massive variation from individual to individual based off of the type of food and they also looked at gut biome and they seem to get some correlations between some things that we would typically consider unhealthy gut microbiota being related to poor glycemic control. They did kind of a cool crossover process where they created an algorithm based off of all the inputs that they had in the 800 folks in the main study group.

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Then based off of that, they created a kind of an algorithmic yes-no flow chart. Are your gut microbiota like this or like that? They had like 800 or 850 different steps to flow people through this thing then they got 100 new folks and they assess them based off of several different parameters than they recommended a dietary approach that they thought would -- in one group should be good for them. They would get good glycemic

control. In another group it would be poor for them and it will give them poor glycemic control and again, this depended.

Some people ended up with a lot of carbs. Some of them ended up with relatively few, but they had really good predictive power based off this algorithm that was going in. But the thing that just struck me was that it was incredibly individualistic. One person would or would not react to various foods. They were looking at again the gut microbiota, but they were not looking say like at the immunogenic potential of food. So like are people immunogenically reactive to dairy or gluten or things like that, which is another layer of this thing. But one thing that did stick out to me is that, in general, folks that had what we would consider a poor gut microbiota, if they fed them in a way that they anticipated would produce a beneficial glycemic profile it then did improve their gut microbiota.

Dr. Ruscio: Got you, got you.

Robb Wolf: What to draw from that, I'm not entirely sure? This is I think some of the point that you've made. Where is the correlation, where is the causation in this? So it was modulating insulin in some way. Did that influence the changes in gut microbiota? Was it direct?

Dr. Ruscio: That's been shown.

Robb Wolf: Was it feeding them? Was it both? I suspect it's probably both. So you start getting this push-pull thing where you've got multiple vectors influencing all the stuff.

Dr. Ruscio: Right and to your point, and I think we may have discussed this on the last time I was on your podcast. All this is starting to just blur together in my head so forget if I'm repeating myself. But we have seen treatments in type 1 diabetics with insulin and their microbiota is normalized and become more like that of healthy controls after they're administered insulin. I know you posted a paper where when rheumatoid arthritis was treated and normalized the microbiota.

Robb Wolf: Right.

Dr. Ruscio: I know that we're looking for this world of bacteria in the gut to be the next key to disease just like we're looking for sequencing the human genome would be the next cure for all disease, but look what we came away with there. Right? We came away with a couple of few things that are mildly helpful at best, yeah.

But what I do think is very important and this is not to discount the huge medicinal benefit of improving your gut health, we want to first get rid of -- well first, we want to focus on one just general dietary principles and lifestyle and get them in a Paleo vicinity and get them in a decent lifestyle vicinity then re-evaluate. If people are still unhealthy, then boy, doing a thorough gut evaluation and treating any overgrowths or infections has a huge impact and that does have a large impact also on the microbiota because if you have pathogens or overgrowths, they tend to change the environment locally to be more hospitable for pathogens and less hospitable for healthy commensals.

So I really think and this is one of the principles that's woven throughout the book that will hopefully one day be published is that we can't custom manipulate the microbiota, at least not right now. We're light years away from that and it may not even be ever be possible because it's even more complicated than your genes and we still can do that with treating it in genes for the most part. But what we can do is provide a healthy environment and a healthy environment will foster healthy bacteria and you will derive any of the health benefits that you can from those healthy bacteria.

Robb Wolf: Right and one thing that I didn't take away from this paper, there's a lot of contention about some folks are doing well on sweet potatoes and white potatoes and some white rice and all that stuff. I'm assuming that those folks are probably expressing some fantastic insulin signaling around that whole process. I'm not one of those people.

So for me to be successful, I need to limit my carbohydrate load in general. If I do a larger carbohydrate load, it's got to be post workout. I've got to have some other factors in the mix. I've been making good use of chromium and alpha-lipoic acid. I take that with the meals that I have, a significant amount of carbohydrate with and that's been working pretty well. Like I feel pretty good, my body composition is good.

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I haven't been doing a ton of blood glucose monitoring because I'm just lazy and don't really want to do that stuff. But at the end of the day, all the theory kind of goes out the window and I was kind of noodling on like a blog post something to the effect, the death of diets or something like that. This thing just so blew me out of the water, but there is still this reality that I think we can pretty safely make an argument that sleep, food, exercise, community that's consistent with kind of this evolutionary framework is probably going to be pretty helpful as a starting place. That's going to mean different things for different people.

Some people, it's going to be pretty low carb or maybe even ketogenic. For other people they'll be able to kick their heels up and go wild on plantains and bananas and white rice and all kinds of stuff and will totally kick ass on that. But I guess we just need to move to a spot where we're okay with that. It's hard for me trying to be in the spot of health education. I'm always searching for that heuristic, that simple rule of thumb that's going to help a lot of people, but I was kind of noodling on this. I'm like is there anything I can say that's going to help anyone?

Dr. Ruscio: Well, I think there is, but it's different. It's getting people to look inward and trust their own bodies. I think that's one of the biggest rules, is first, as you said get in a dietary vicinity and get in the lifestyle vicinity. I would add to that, then if things haven't gotten much better, remove any offending problems that oftentimes there is gut problems, infections, overgrowth, imbalances what have you. A lot of what I end up doing I think is it is that rule of thumb with patients which is let me just help guide you through the process of playing around a little bit with your diet and listening to your body and being okay with that.

People really just they need to be told what to do I think in a lot of cases and I think one of the things that's so freeing from my patients is that they come and think and they has to be ketogenic and then I tell them why don't you eat at least a 150 g of carbs everyday for the next month? And they almost kind of shit their pants when I first told them that, but they come back a month later and they've lost 5 pounds and their blood glucose has gone down ironically and so lessen their cholesterol and they feel a lot better. There's a need that person to give them the confidence that it's okay to listen to their own body and I think that might be that rule that we're after.

Robb Wolf: Right, right, just customization ultimately. Doc, where does subclinical hypothyroid come into this whole story? I know that we've seen a ton of different vectors from the gut and from the other hormonal axes, cortisol and insulin that can kind of alter thyroid regulation. One of the things that I guess I brought to the table when I got involved with the specially health risks assessment program, they were doing a really great job on the lipidology, but when I started reviewing some cases, like I saw a lot of people that just immediately and I'm not a Functional Medicine wiz, but because of figuring out some of my own stuff, t became recently adept with the thyroid kind of dysregulation patterns.

I was like man, I don't think this guy really has a cholesterol or lipoprotein issue. I think he's got a thyroid issue and we got in and tinkered with that

stuff. We could drop their lipoprotein count from 2000 to 1000 just by getting amount of some naturoid or something like that and addressing some adrenal issues. Like where does all that play into this and how do we make sense of that on blood work?

Dr. Ruscio: That's a great question and if you don't mind, I want to come back to that in just one second. There are two things before we left the gut. I just wanted to chime in really quick. We were talking a while back about Christopher Gardner who is the PhD researcher at Standford that did the A to Z weight loss trial and how his weight loss trial looked at the Atkins diet, the Zone diet, the Ornish diet and essentially looked at the spectrum from high-carb low-carb diets. He found this whole individuality of carbohydrate threshold and he termed it heterogeneity of insulin sensitivity. Meaning, some people are very insulin sensitive and they can have more carbs and some people are insulin resistance and they have to eat less carbs.

He is recently in the middle of a followup study on that and it's very interesting to see that and he just came on the podcast and that podcast should be released in a few weeks and we expanded on this. But they are essentially having people either eat a very low-carb diet or a very high-carb diet for a short period of time and then having them slowly kind of titrate back toward the center as a way to identify what dietary approach works best for you.

What's interesting is he's finding that people are gaining weight and losing weight on both a low carb and a high carb diet, which really reinforces this posit that one dietary approach isn't going to work for everybody. But I love what he is doing, which is essentially what I've been doing in a way in the clinic which is starting people at one endpoint on this dietary macronutrient spectrum and then having them slowly kind of come back to center to try to forgot where they feel best. So that was cool to see – this research is really at the forefront of this setting up his study in agreement with what I think a lot of us have been recommending for a while anyway.

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Robb Wolf: Interesting, interesting. Doc, one of my good friends strength coach Dan John ages ago, he was basically like any seminar clinic or anyone that you talked to who is more knowledgeable about something, make sure to make the conversation about your needs. So I'll steer it towards my needs and we've talked about this before. So I feel like I'm kind of in this spot where fermentable carbohydrate may not be the best thing for me, but this fermentable carbohydrates are often times lower glycemic load and I'm trying to figure out what the hack is there?

Like if I'm eating more or less ketogenic, good cognitive function, if I start putting some carbs in, I tend to get kind of hungry I guess kind of where I'm at now is like my breakfast is pretty protein and fat rich with a little bit of carbs, like 20 g of carbs. Then I try to get my training in around noon or 1 o'clock usually good with that. I might need a little bit of a snack to get me through doing jujitsu or what have you and then I throw in a decent whack of carbs based off of volume and intensity after that training. What do you feel like it's going on there and like we still -- Doc is trying to pin me down to get some gut testing through his clinic so that we can go deep into the poop on this, but from your vast clinical experience, what do you think is going on there?

Dr. Ruscio: Well, it's sounds to me like you're someone that's going to do better on the lower threshold and knowing your medical history, that make sense right with everything that you've been talked about earlier in the show. So it make sense to me that a high level of fermentable carbs is not something you're going to do great on, which I think it's important for people to hear that because it somewhat contradicts the eat fiber and prebiotics until you're blue in the face so you can have the most adverse microbiotic possible narrative that's circuiting right now about the microbiodata. It's not factual.

It's a little bit of a biased opinion and it's important that people know that because I want to try to prevent people from causing problems that look kind of like the problems that you noticed when you started to go to that higher probiotic carb intake. I mean it's good for people to experiment I suppose, but I just didn't want them to be blindly following that thinking that the only way to improve the health of your microbiota is by feeding bugs because there is this other side of the coin where trimming back bugs or an antibacterial approach maybe the best for some.

So what I think it's probably happening is you're probably feeding a little bit of an imbalance when you have. I'm thinking it's probably simple as that you're probably feeding a little bit of an imbalance with fermentable substrates and that's probably causing some problems. We can probably get a descent handle on that with some testing and you may need to go through a short course of antimicrobials or antibiotics or maybe even incorporate fasting. Fasting can sometimes be very helpful with the kind of quell inflammation in the gut.

But I think what's probably happening there just speculating and I don't know a ton about your history. I've caught bits and pieces there so I'm

kind of taking a shot at this with one eye closed, but there is probably some sort of an imbalance that we know is associated with Crohn's, which again can be via the dactin1 and card9**.** It can be a propensity toward fungal overgrowth. It maybe a mild bacterial infection or a mild case of SIBO.

But when you're eating these higher carbs, you're provocating that underlying problem. I think that's probably where some of these problems are coming from. So then, how do we fix that or how do we maybe increase your tolerance? First, we try to get a handle on exactly what it is and then certain probiotics or probiotic funguses like saccharomyces boulardii can be healthy for crowding out bad or overgrowth of fungus or bacteria.

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Depending on if there's a light fungal overgrowth that keeps coming back, you may need a biofilm agent co-administered. It's not the first thing that I do, but for conditions that had been present for a long time that don't respond favorably to the initial round of antimicrobials, then biofilms are a consideration after that. So it maybe a fungal issue that's protected by a little bit of a biofilm, which is why it's maybe never fully gone away.

And then the other thing that maybe helpful that we talked about last time and I sent you a paper on this recently was using some herbal anti-inflammatories maybe helpful. Many of the drugs that work well for these symptoms of Crohn's and ulcerative colitis are TNF alpha-inhibitors and there is also a family of herbal medicines that are very powerful TNF alpha-inhibitors, but also don't have the side effects that the drugs have which makes them a really favorable choice in my opinion. I just sent you a paper on how powerful the TNF alpha-inhibitors have been shown to be in fixing leaky gut.

So if there's a degree of that present, for example, if we saw a mild elevation of Crohn's antibodies, we know that administering some sort of anti-inflammatory TNF alpha-inhibitor may help with leaky gut and immune disregulation that's associated with that. So I mean there is definitely a few things at our disposal. It would just be figuring out what we have to use for you and kind of how to sequence them, but those are some of the big players right there.

Robb Wolf: Got you, got you. Okay. And so what are you doing with the initial testing? I know that folks are going through things like uBiome and some other outfits trying to get a handle on what's going on with their gut. Like

what's the process with that, what's working, what gaps are left in the current battery of things that most folks are tinkering with?

Dr. Ruscio: Yes, great, great question and real quick before I jump into that one. I just want to make you be aware of another study. I don't think we mentioned this last time, but I think this is important for people to be aware of. A group of about 50 women were administered a flax derived prebiotic and their microbiotas were tracked as well as their weight and inflammatory markers. Now, the women didn't lose any weight, but they did see an improvement in inflammation and the inflammation improvement was independent of any changes in the microbiota.

So this is a great study that shows that the microbiota isn't the gatekeeper to every health improvement you have or don't have. I think it's important for you to realize that because it's been I think a little bit falsely propped up. Not to say that the gut is not usually important, I hope people understand that. If anybody understands the importance of the gut, it's me. It's just sometimes we add in this intermediary piece. We give prebiotics that causes this effect that we you want to happen in the microbiota and that microbiota change affects health. I don't think that's how it happens. I think this is a great study kind of reinforcing that, but to your question which also expands on this topic. The microbiome tests and you mentioned the names a moment ago.

In my opinion, they have zero clinical utility at this point in time. It's not to say that they are not great for research purposes and I think we should support these companies and these labs because as we gather more data, we may come to a place where we have actionable information. The only information we have right now that even hints on clinically relevant is using microbiota assays for screening for colorectal cancer and for inflammatory bowel disease.

These are just preliminary findings that are in my opinion not even really accessible to someone reading this because the researchers created their own groupings and these groupings skew in such a way that associated with colorectal cancer for example. This is quite a bit beyond I think it's even available to anyone commercially yet, but at best, we have a screening tool that may prompt treatment for inflammatory bowel disease or colorectal cancer, but it doesn’t mean it tells you anything about how to treat. Does that make sense?

Robb Wolf: Right, right.

Dr. Ruscio: So it just tells you hey, this disease maybe present, proceed to establish treatment for the said disease. It does not give you anything new in terms of treatment and that is after an exhaustive study of the high level science on this stuff. If I wanted to cherry pick lower level scientific data like a cell line study or one obscure observational study or one outlier study, I could do that. But you have to be disciplined in referencing with science. You have to only use what's truthful. You have to look at the entire body of evidence and then report what the entire body of evidence shows and not just report on outliers or just use references to footnote your opinion, right.

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So I think if anyone looks at the body of evidence at large, they will see that and gush, just go read some of the systemic reviews with meta analyses and you will see the researchers concluding this. You'll never see a researcher say we can treat the microbiota in this way to produce this effect. At best that you use language, by administering this compound, it maybe a favorable treatment for this in the future. At best, you hear that it maybe and while I'm really on a rant here, I'm sorry to just be going on my own there.

Robb Wolf: You're wild.

Dr. Ruscio: But there was another and sometimes, you hear bullshit, I'm sorry, from some of these researchers with how they conclude their studies and I maybe I'm being a bit harsh there because I understand there are publication pressures on these researchers on this kind of like publisher perish. Like I get that there are pressures there, but there was a study done in obese subjects and obese and insulin resistant subjects that were give fecal microbial transplant therapy. The researchers concluded that FMT maybe a successful and exciting treatment for insulin resistance in diabetes. Yet when you go and you actually look at the graph because when I saw that and I heard it, I was like wow, this is the first time I've ever heard this. This is substantial. I'd better really look into this.

When you pull up the graph of their data, the most important and relevant markers of glucose didn't change. Fasting blood glucose, insulin didn't change. There was one obscure glucose kinetic that changed just marginally and the people didn't lose any weight or any body fat. So to see one obscured glucose kinetic shift slightly and they conclude from that that we should give you poop enema from someone else as a treatment for diabetes when you can see at 20 point drop from using Berberine or over a 33 point drop by using certain prebiotics, that is infuriating in my opinion.

That's where you have to be careful because there's a lot of excitement here and there's a lot of overreaching and over speculating and it just creates all this confusion that people are presented with. I feel like the half the time I'm talking about the microbiota, I'm just trying to unwind all the BS that's circulating out there and it's starting to become the less fun for me because I just want to be able to talk about the cool clinical stuff. But there is just so much misinformation circulating. So I guess before I answer your question and irritating anybody else on the call, anything you wanted me to expand on in that Robb?

Robb Wolf: No, no, no. I mean, it's funny like it's still the same rinse, lather repeat. It's like high carbs versus low carbs. It's just we have yet another layer to the onion that we're talking about with the microbiota. Like maybe it influences things, maybe it doesn't, maybe it's causative, maybe it's associative. There is probably situations where all of that is true and again, to get newsworthy pieces, you really need a piffy kind of one line definitive kind of statement and most of this stuff just doesn't lend itself to that, but it often times get shoehorned and do exactly that type of presentation.

Dr. Ruscio: Yeah. It reminds me of -- I was on Facebook the other day. I saw something pop up in my feed, something along the lines of cure asthma in children or like the cure for asthma in children. I'm like this is interesting because I just went through an exhaustive review of all early life interventions for the microbiota that may impact asthma so let me see what this person has to say. It was an animal model where they gave a probiotic that you can't give to humans because it gets killed by air and that had a beneficial effect on asthma in this group.

So the headline was cure asthma, there was a brief mention of the study and then recommendations of probiotics that were completely different. I gave this person a little bit of a crap about it and I said don't you think that's a little bit irresponsible especially when you look at and I cut and pasted like seven clinical trials using probiotics in children for asthma and there's minimal results. Don't you think what your writing is a little bit irresponsible when the clinical evidence suggest that right now, we don't have a viable microbiota treatment for asthma? The response was well, I get that, but I need to make sure that's it's a catchy headline.

Robb Wolf: Great.

Dr. Ruscio: It's going to turn the space into Walmart where everyone needs to have a bigger neon side and shout louder and louder to get anyone's attention if this is how we conduct ourselves. But let me get off my soap box and

come back to the one of the reasons why I think there is this confusion, this disparity with some of the microbiota testing and with health. It's because with the microbiota testing, we don't get a look at the small intestine and the small intestine is responsible for 90% on caloric absorption, 55% of the entire gastrointestinal tract and the largest entity of immune cells in the entire body as your gut in your Peyer's patches. So I think part of the reason why there is so much confusion is because most of your intestine is not accounted for on microbiota testing.

(0:40:53)

Robb Wolf: And I believe the most biologically significant portion of that.

Dr. Ruscio: Definitely, yeah, yeah.

Robb Wolf: And so I mean what are folks doing with that? Like I've seen like some lactulose manulose testing where they look at gas release at different stages that are going through the small intestine. Are you using things like that or like how do we untangle that problem?

Dr. Ruscio: We're using the lactulose hydrogen methane breath test and we just recently had approval for one of our clinical trials. We're starting a clinical trial in prevention of SIBO relapse. We just had the IRB approved. We're waiting on a grant and I'm hoping we'll be enrolling patients January or February next year.

We're asking and answering a question that's very important for the SIBO treatment community because it's regarding a treatment that is often used, but we don't really know if it works. We're hoping that it does, but no one's really have done the study to see if it does. So we're using, of course that test quite extensively in this study because we're tracking patients both their symptoms and their land markers once they've cleared SIBO and seeing if this treatment will have a positive effect in preventing relapse.

Then we're about to engage in another study or actually, we just enrolled our first patient last week Wednesday because we weren't in the office because of Thanksgiving. We're studying a new compound that is supposed to be better for methane gas SIBO, which can be the harder of the two to clear. Speculating a little bit, but I think that the methane type of SIBO may have the most metabolic implications because methane slows down transit and that may allow you to absorb more calories from your food.

And we see in Sub-Saharan Africa for example the highest colonization density of methane organisms like methane or Methanobrevibacter

smithii and we think it's an evolutionary adaptation because they don't eat a lot of calories and they eat a highly fibrous hard to digest diet. So by slowing down transit, you allow your body more time to break down the food and thus, absorb more calories from the food and also these highly fibrous foods can have a very laxating effect.

So this bacteria may help slow things down so that they don't pass through too quickly so that you can absorb more calories from them. That might be why when we seen an overgrowth of this bacteria in westerners, it causes constipation and there is some preliminary evidence showing that it may correlate with weight gain, high cholesterol and high blood sugar levels.

Robb Wolf: Interesting. What would be driving say like looser stools, food comes in looking somewhat the same -- as what comes out looking the same as it went it.

Dr. Ruscio: Is it a personal problem Robb?

Robb Wolf: This might be a personal problem, yes, yes. I know a friend who has described something like this. Like if there is any type of IBS spectrum and I know this is the interesting thing. It can be either constipation or more loose stools or flipping back and forth from both, but my thing has always been it seems like maybe an overly rapid transit time and not breaking stuff down enough and I've filled a lot with like now food, super enzymes and things like that, but what's the driver with that?

Dr. Ruscio: Well, that completely fits with the presentation of IBD.

Robb Wolf: Okay.

Dr. Ruscio: People with IBD tend to have a faster transit and if we push that needle all the way into full blown IBD relapse, you have diarrhea, chronic diarrhea, 7 to 8 diarrheas a day, and you can even have bloody stools, right. So definitely, that fits your medical history. So the more I learn about you, we're kind of piecing these things together. This is a fun part of -- you know what I do all day is that you're trying to piece all these things together. So it maybe an issue of not having enough acids or enzymes. I generally I'm coming to question that as being a major driver.

(0:45:15)

I think some support can be helpful in the short term, but you being younger guy or not 60, 70, 80. You're not so old that I think you're gastric ells are really just kind of poofing out on this, right? And then also knowing your medical history, I'd be more inclined to think this is an

immune reaction of some sort where the body is just -- it's known as peristaltic rush where the body is trying to push something that doesn't irritant out more quickly.

I'd be more inclined to think that there is an immune angle on this especially because assuming you've already beta tested the enzyme and acids and it sounds like maybe you got a little bit of help from it, but it didn't fully fixed the problem, right.

Robb Wolf: A little bump and then you know I notice things like if I'm on vacation, things improve at ton. If I'm out on the sun, things improve a ton. So the stress level is a huge vector with that. When stress goes up, digestion starts to fail, yeah, yeah for sure.

Dr. Ruscio: And that's also been reported in IBS and IBD and most gastrointestinal conditions seem to negatively respond to stress and there is a microbiota angle to that. It has been shown for example that college students when they're under exam stress, see a decrease in lactobacillus and I believe also in Bifidobacterium species. The solution to that is really just the stress, which reinforces as part of my posit which is creating a healthy environment to foster healthy bacteria, right. So the stress input that creates an unhealthy environment and immediately, we see lactobacillus and Bifidobacterium populations to window.

So yeah, I think an immune piece would be where I think one of the good first steps would be for that. Addressing that could be with anti-inflammatories. They could be with probiotics. They could be cleaning out of something that shouldn't be there. All those would really help with the immune piece, but I see more of that. I see more of the loose stools being caused by immunoreactions that I do necessarily being a deficiency of hydrochloric acid.

Robb Wolf: Got you, okay, okay. Well Doc, we could go on and on and on, but I actually I have a jujitsu appointment here before too long. I need to go get rid of some of my stress. Remind folks where they can track you down online and then also remind them about the conference that you will be at in the UK in mid-January.

Dr. Ruscio: So I'm still plugging away over it, drruscio.com and I'll send you the link for the event in January. It will be London january16th and 17th. It will be Melissa Hartwig and myself and sponsored through Refined Health. So if you search Refined Health and then Michael Ruscio, you should see my page come up or if you search Refined Health Melissa Hartwig, you should see her page come up and excited about that event. I think it

would be really fun and next time I come on Robb, we can maybe jump into some of the stuff on subclinical hypothyroid because that's another interesting and important piece that I think it's important to kind of outline, but definitely a lot of hot wind for me to blow on that once so…

Robb Wolf: It's awesome, man, the more hot wind from you, the better. It comes out of the right end on you. It comes out of the wrong end on me. It's fantastic.

Dr. Ruscio: Hopefully, we can fix that.

Robb Wolf: Yeah. I will finally sack up and contact the clinic and figure out how to get rolling on this thing. Some of the stuff it's funny man like you almost go a little bit epileptic over the whole thing like I just should be able to figure this out on my own, but clearly, I cannot so…

Dr. Ruscio: It's hard when you're looking at it yourself and there is an old saying, he who asks himself or he who treats himself has a fool as a patient or something like that, that makes a lot of sense.

Robb Wolf: Right, right. Yeah, absolutely. Well Doc, thank you again, fascinating as always and will talk to you soon.

Dr. Ruscio: So thanks Robb.

Robb Wolf: Take care. Bye-bye.

Dr. Ruscio: Bye.

(0:49:23) **End of Audio**