Paleo Solution - 235

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Robb: Howdy folks, Robb Wolf here. It's been a while. Sorry. We had to do some recycled shows but we had a baby and all that type of stuff. But to welcome you all back to the PaleoSolution podcast. We have the gorgeous gentleman who quacks the gut Doctor Michael Ruscio. Doc, how are you doing?

Michael: I'm good. How are you my friend?

Robb: Good. We were just talking about people being set on fire here a moment ago with the drunken festivities. So hopefully the rest of our conversation was actually very entertaining before we started talking science and gut and all that so hopefully I'll maintain some element of that. Doc, you've been super busy. What have you been up to?

Michael: Well I did a webinar on the gut for about 2 ½ weeks ago and then I was at AHS and I did a whole presentation on the gut and the gut microbiota, AHS and that's why I kind of reached out to you because I thought especially the comparative analysis of different hunter-gatherers compared to agrarian societies compared to westernized societies. I thought that would be kind of cool to jump into that a little bit.

So that was with AHS and then about a week after that, I flew down to San Diego and I was interviewed for a documentary on digestion disorders and here we are today talking to the burtonesque Robb wolf.

Robb: Nice. Be honest, you were thinking a lot about poop and I just popped into your head.

Michael: It's always on my mind.

Robb: It's like if A then B kind of deal, poop then Robb Wolf. So awesome. I'm stoked. So we threw out to Twitterville that you were going to come on the show. We actually got some really phenomenal questions with that and I know you had a bit of an agenda that you had a few seemingly tangent chill topics that you wanted to hit and then jump right down into the pink pucker as it were.

Michael: Yeah. So I guess the couple of tangential things, so hydrochloric acid is a big deal. Everyone's trying to make sure they have adequate hydrochloric acid production. A lot of people tend to do better with hydrochloric acid supplementation. It can leave you at higher risk for infections, sebo, malabsorption, anemia, the first domino that falls in the digestive domino rally cascade so definitely HCL is a big deal.

I came across a paper recently of a group in Japan that was studying a condition known as oral lichen planus which is essentially an oral disease that often times manifest with malabsorption of vitamin B12. So they were administering vitamin B12 injections and as they were doing so, they tracked a few markers and to the markers they tracked were anti parietal cell antibodies and intrinsic factor antibodies.

And these are the antibodies that will elevate and can cause damage and destruction to the cells of your stomach that are responsible for manufacturing hydrochloric acid. And about 20-40% of patients with Hashimoto's for example will have elevations in these antibodies. Now here is the now and really cool thing. They found that after giving B12 injections, they actually saw the antibodies against the parietal cells and intrinsic factor go away.

So they were essentially able to turn off the autoimmune process in the stomach by administering vitamin B12 injections. Well that's important is because if you catch that early, you could potentially prevent someone from needing to supplement with hydrochloric acid for the rest of their life because you stopped the process that was damaging the hydrochloric acid producing cells in the stomach to begin with.

Robb: Interesting. And I just went in for some blood work. Everything's looking better and better as time goes on but one thing that we found that was kind of interesting was that my globulin was kind of low. And I have not been using supplemental like betaine hydrochloride like the AdaptaGest or now food super enzymes or something along that line. So I started using a little bit of apple cider vinegar with some of my larger meals but you know, I had never actually had my B12 checked.

Michael: Yeah. I mean you may be able to pick up a B12 imbalance. We'll just look at your mean corpuscular volume, your ARMC, the MCHC, some of those

markers that you'll see in the CBC with a differential. You can kind of maybe you pick up early signs of that there.

Robb: Gotcha.

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Michael: But B12 definitely a good idea also. But this is really interesting because this has the potential to really save people for needing hydrochloric acid supplementation for the rest of their life and we've been running these antibodies and administering the B12 injections at our clinic and I'm hoping that we'll find that this is something that we can replicate.

Because once the antibodies were shut off so to speak, these people only had to do a once a month injection which once a month injection with subcutaneous B12 is really – it's a tiny little pin prick that you can do and this retains fat in your stomach and that can really keep your stomach healthy then that can be really, really important. And interestingly, there were two studies published in the mid 80's looking at they had the gluten free diet would stop those antibodies and they did not find any effect on these antibodies specifically from a gluten free diet.

So I'm sure they're open to all the wonderful things that a gluten free diet does and one of the first things I recommend with all my patients but it doesn't seem that we can kind of use the blanket. And that's a recommendation we all like to make for managing autoimmunity being a gluten free diet as something that seems to be viable for stopping this in particular. So anyway we're not getting too far down that rabbit hole. I thought that was something interesting that people might want to be aware of.

Robb: Super interesting. And doc do you have a sense of maybe what the chicken and egg element is of this like you start getting some down regulation of the gut or some inflammation in the gut that influences B12 absorption and then kind of a feed forward mechanism or like a B12 deficiency then do you have any sense of what the precipitator is in this?

Part of my thought is that there may be multiple pads to get here which makes it devilishly hard to do like the classic evidence based medicine study to determine causation on this. In my head, I can think of a couple

different mechanisms that would get you to the same spot. What are your thoughts on that?

Michael: Well one of the things that it's hard to tie an exact path back to but I think may be possible is we do see that when an N gland is experiencing autoimmunity, sometimes there is a disruption of what's known as the gland blood barrier. So for example, the thyroid blood barrier – if the thyroid gland becomes overworked and starts becoming damaged and it can't repair faster than it gets damaged, you may see it breakdown in a barrier that separates the gland from the blood stream and then when that happens, it really increases immune surveillance of that gland and that can be one of the precipitating factors for autoimmunity.

So this is why a short term administration of some kind of like thyroid replacement hormone can – and hasn't shown to actually lower antibodies because if you give the hormone the glands making then the gland doesn't have to work essentially, work in the capacity of producing hormone so the gland can focus on internal repair. And as it repairs, it then repairs the barrier and when the barrier is healed, you don't have as much penetration of the blood gland barrier and less recognition or less surveillance of the minute system.

So we know by giving thyroid hormone you can get the thyroid gland a chance to rest, prepare itself and you may stop autoimmunity by restoring the barrier. I'm thinking that maybe an element of that that's present here but it's hard to say with just B12 specifically because there's more in regulating acid production than just of course B12. So that will be the one thing I maybe would speculate but I'm hard pressed or much beyond that. Do you have anything that comes in mind on your end?

Robb: Well just not specifically but a little bit of a parallel, these folks that we're working with at the clinic Liposcience, they've really pioneered the advanced testing, inter mark testing for lipoproteins. And they have developed a screen called the DRI, the Diabetes Risk Indicator and it's pretty snazzy. We're going to have Dr. Bill Cromwell on the show here in a couple of weeks to talk about that.

But part of what they are claiming and I'm still going through and trying to vet the science on this but what they're claiming is that based on your DRI score, they can give you a pretty good sense of where your beta cell

stresses for the pancreas. And based off of that, then instead of we have a little bit of a slap dash approach right now as far as well maybe if somebody's in an insulin resistant state, if we drop carbohydrate load then it takes stress off pancreas because the pancreas just works and works and works and then it just fails in the development of type 2 diabetes.

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So what they feel like is that we'll have some really good very quantifiable end points where if we're seeing inflammation and stress in those beta cells, take that stress away similar to what you're talking about with say like thyroid replacement. And then part of the back mechanism on that which I had not really thought of which is decreasing the immune surveillance of that tissue and then it's able to recover and then we start thinking about addressing the fundamental causes of why this person is at the stage of throughout whether it's small intestinal bacterial overgrowth or just generally overeating or low vitamin D or whatever the myriad off issues are but it definitely makes me think of that.

Michael: Yeah. That's interesting. I'll be curious to see where you guys end up on that because I think it's great to have some kind of early stage screening that can prompt some kind of added measure so yeah that's cool.

Robb: Yeah. I'm very excited about that. These guys, again, they are really the outfit that pioneer the use of NMR in looking at lipoproteins and apparently you know, when you do an NMR on a complex item like plasma, you just get tons and tons of different signals and these guys at Liposcience have about 11 million samples that they've run and they still don't know what all these signals are.

Like they haven't gotten ahead and done controls to be able to identify okay this one goes with glycade. This one goes with valin. That one goes with LDL-P of this size and this one goes with LDL-P of this size. They're basically bat cataloging all that stuff and some of their correlation work that they've done again seems to tie in with some beta cell dysfunction and some other things.

So it's pretty interesting but it's also inexpensive and NMR's are pretty easy to get the diagnostics right. They're easy to run controls. They're easy to keep everything in calibration compared to a lot of other

methodologies. So it could be a really flick inexpensive way to get a lot of information not only on your lipoprotein status but also on a variety of inflammation and a variety of pre-diabetes information.

They have Alzheimer's predictive kind of measure in that which I'll let Doctor Cromwell talk about that stuff because he's much better versed on it than I am. I'm just now reading the papers. He's actually written all the papers. He might be able to comment on that better than I can.

Michael: Sure. Sure. Cool. Give me posted. I'd be curious even to start incorporation with those markers at the clinic as they become available.

Robb: They were super excited about it so and then again it kind of makes me think of what you're doing on the functional medicine side with the guy and these B12 supplementation injections to reverse that. I remember when you dug that paper up and shot it to me, it sounded like you had possibly wet on every flat surface on your home that day when you found it. You're like this is amazing. It totally is. It's very, very cool.

And in the functional medicine, maybe alternative medicine scene, the B12 injection has been very popular with some folks, kind of almost relegated to pseudoscience by other folks but it looks like this could vindicate a pretty big swat of potential benefit the people are getting from B12's injections.

Michael: And I agree. I do think sometimes B12 injections are overdone but yeah, like you said, I think this can really kind of substantiate or just show a very clinically relevant need for those and certain autoimmune population. It's a bit early to be you know, absolutely for sure I'd love to kind of report in on this maybe in six months once we have the crowd of patients that we're putting to this protocol now, once we have a chance to see if the numbers that were recorded in Japan, we can kind of replicate in our center but fingers crossed.

Robb: Nice. Super cool. Very cool.

Michael: So the only other tangential thing that I want to touch on, and this is a little less exciting and it involves diarrhea which is not quite as cool. In alternative medicine or even in conventional medicine providers that are practicing more alternatively, there's this kind of standard recommendation that if you have your gallbladder out, you should be

supplementing with bile salts or bile acids. And I certainly think that can be helpful for a number of people but there's something that I wanted to address in that regard.

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There's something known as bile acid diarrhea where bile can actually cause diarrhea in some people after a gallbladderectomy and the reason for this is because the liver is what produces the bile and the gallbladder is what temporarily stores it and what can happen in people that have a gallbladderectomy is instead of having the bile produced by the liver then stored in the gallbladder and released in the appropriate time and the appropriate amount, they have this constant leakage of bile from their liver into the intestines.

And how it kind of plays out is this, the short story is if you have too much bile, it can cause diarrhea. But here's how it actually works. In the small intestine you have the duodenum, jejunum, and then the ileum and so everything is released in the first section, the duodenum and the bile kind of coats down the whole small intestine, to the small intestine and gets absorbed in the ileum. But if you can't absorb enough of the bile and ileum, the next stop is then the large intestine, if too much bile gets in the large intestine, it can cause a diarrhea response.

So for people that don't have a gall bladder, they may actually be releasing too much bile and it may overwhelm the re-absorptive capacity of the ileum and that may cause too much bile again in the large intestines where it cause diarrhea response. And these people will respond to what's called the bile acid sequestrant. So there's a drug known as colitromin but there's also natural agents like propolis, boswellia may be helpful for that also, they kind of bind this excess bile and prevent an over – it's too much bile essentially.

Now, that also maybe a problem for people that have sebo and people that have Crohn's disease. And again it's definitely not going to be the majority but I throw this out there for people that are doing everything else and still not feeling well and still may be having a problem with loose stool or diarrhea, it's something they may want to try.

Because in Crohn's disease, Crohn's disease can affect the ileum. They can go all the way up to the ileum and if Crohn's disease is causing lesions in

the ileum, the ileum is responsible for reabsorbing bile. So it may be a problem in people that have Crohn's disease and then sebo actually, sebo is actually known to deconjugate bile or kind of break it apart so to speak and that actually makes it more difficult for ilea reabsorption. So this may be an issue with people with sebo also.

I would say probably more people with sebo would benefit from some bile then with less bile but there's a few tricky things with bile. It's not an incredibly straight forward piece because bile does act as a strong antibacterial but bile also seems to be somewhat of an upper GI. It seems that somewhat inhibit upper GI…

So and what you don't want sebo. So maybe I'm getting a little too deep. I guess I'll zoom out for a minute. If people have either sebo, Crohn's or have had their gallbladder removed and they've done all the other stuff you would do initially and are still having a problem, they may want to try a short course of a bile acid sequestrant like cholestyramine or preferably even a natural agent like guggul and see if that helps.

Robb: And planetary herbs has like a guggul boswellia combo that would be really good for that. Have you tinkered for that one?

Michael: I haven't used that one. There's a guggul policosanol policosanoid mixture that we've been using. But yeah, I think anyone of those would work if people want to try it.

Robb: So the same old deal unfortunately no one size fits all prescription on this stuff.

Michael: Yeah. No one size fits all prescription but I like to look at things clinically kind of in somewhat of a linear fashion where I start with the things that are most common and the easiest to do and then we work away through those things. And the end of the line are the things that are least common and potentially either more invasive or more expensive.

Robb: Right. Totally makes sense. Peel the onion instead of jumping into the most difficult stuff and then working backwards.

Michael: Right. Exactly. So with that you want to jump into some of the stuff on the bio?

Robb: Yeah. Let's do it.

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Michael: Cool. So this should be available on the AHS YouTube page. I don't think it's up just yet but it should be up soon and I'm just going to try to hit some of the higher points. There will definitely be more information on that presentation if people can watch it and also of course the visual aids because it's a video. But one of the things that we consistently see with micro biome is westernized societies have decreased diversity. And I think you've had – that's come up on your show numerous times before right?

Robb: Right.

Michael: So I think people get that so I won't really go into too much detail about that. But maybe one of the more interesting concepts is looking at this, how different populations that maybe pathogenic to some may actually function as a symbiotic organism in other populations and I think a population that illustrates that is the Hadza. The Hadza are arguably one of the last kind of untouched hunter-gatherer groups that are around right now.

Their diet consists of about 70% plant foods, highly fibrous tubers, honey and berries and about 30% of meat in the form of birds and game animals. And what's interesting about their microbiota is they have an increase in what's known as treponema pallidum is the organism that causes syphilis. So while they did not sub classify what type of treponema specifically it was, they did see an increase in this treponema which can be considered opportunistic in industrialized countries.

But interestingly it's also this family of bacteria is also very efficient in breaking down xylan and cellulose which are really fibrous hard to digest parts of the plant. So they had a high level of what may be considered an opportunistic infection that we don't really see in westernized countries. Probably because they have this highly, highly fibrous diet. So it seemed to be working to their advantage.

The other interesting thing about the Hadza is they have no bifidobacterium and bifidobacterium in populations in westernized countries is maybe one of the most successfully used probiotics. It's been shown to be very helpful in IBS, in IBD, and I'm sure people have certainly heard of different extremes of supplemental bifidobacterium. But what's interesting in this very healthy hunter-gatherer group, they have no

bifidobacterium and the group of researchers that examined this population commented that there may need to be dairy and or livestock contact or consumption in order to colonize and maintain a colonization of bifidobacterium.

So a few really interesting things about that, this is really how the Hadza, they have no bifidobacterium which has shown to be one of the most protective bacteria in westernized populations and they have this elevation of treponema which some of which can be pathogenic but they seem to be functioning kind of in a symbiotic relationship there.

And I think that reminds me of something else that's really important to mention in this regard which is bacteria have the ability to evolve their genes much, much, much more quickly than humans do. And if the environment changes at a rate faster than the human genome can evolve, what humans seem to have done is they borrow from the genome of bacteria which can adapt quickly in order to survive. So it's really interesting if you kind of think of it in that context right?

Robb: Right.

Michael: So that's one point that I thought was kind of interesting. Another really interesting finding and again this is a really cool slide that people can view in the AHS presentation. They did a huge heat map study where they showed – you have essentially this board and whatever is the darkest colors on the board that's where you have the highest density of whatever bacteria corresponds to that quadroon of the board so to speak.

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And they plotted the Hadza against Burkina Faso and the other group of Africans Malawians so we had like Hadza hunter-gatherers next to rural kind of agrarian non-industrialized societies next to westernized societies. And it's amazing to see that the heath signatures of the different bacteria populations are significantly different from population to population. So we definitely see that the environment is a huge driving factor in terms of the microbiota.

But I guess the follow-up question built into that is what do you really do with that information and what lessons can we learn from that which I

think is a good segway to talk about the study that looked at the microbiota of the Italians versus the Africans. Have you heard about that study Robb?

Robb: I have not.

Michael: So it got a decent amount of press kind of in the little circle of people who are interested in the microbiota.

Robb: Geeks.

Michael: Yeah, geeks. They looked at the Italians next to the Africans. So the Italians were westernized and Africans Burkina Faso were rural agrarian cultures meaning they participated in farming but they are not industrialized. And one of the big findings here was that the non-industrialized Africans had higher levels of this fraction known as bacteria bacteroidetes and they had a higher amount of this bacteroidetes relative to fermicutes and there's this kind of bacteroidetes fermicutes ratio that purportedly you will see out of balance in obesity.

And so what the theory suggests is that those that have a higher bacteroidetes are healthier. And so we see that in this African population, they have a higher amount of bacteroidetes compared to the Italians. So again kind of the theory is westernized people don't have enough bacteroidetes. Non-westernized people have more bacteroidetes and they're healthier and that's what we see in the study.

One of the key bacteria that contributed to that elevate bacteroidetes is this prevotella and when someone people are with this data, they said well this may be because they have a higher grain consumption and they have a higher carbohydrate consumption because prevotella is especially important in breaking down potentially grains and carbohydrates. And so I think some people were starting to think maybe the "ideal diet" is one that's higher in carbohydrate and maybe we need to rethink grain consumption.

But that finding of the high prevotella has not been reinforced in all studies looking at the microbiota. In fact in a Russian sample that were also kind of somewhat agrarian they – and specifically they had a high carbohydrate intake of a lot of grains and potatoes, they did not find an

increase in prevotella so there's definitely some very major contradictions to that theory.

Also something that's really interesting is this "high carbohydrate adaptation" we don't see this reflected in paleo fecal samples. So we do have samples from the Paleolithic era where they were able to really analyze the microbiota and they actually found that bacteroidetes was a greatly, greatly reduced bacteroidetes of the paleo samples were about 20% whereas in the modern day Africans, they were almost 70%.

So what does that mean? We can't derive any definitive conclusions from that but it may mean that people in the paleo area were eating little to no grain and perhaps quite a bit less carbohydrate in the way of grains and potatoes and in highly, highly starchy foods.

Again, we don't know enough for sure but when we look at the paleo fecal samples and some of these carbohydrate adaption that we see in modern day non-westernized populations, we don't see these things kind a match up so to speak. So maybe right there, I'll let you jump in with any questions because I know I'm getting kind of deep and I don't want to lose anybody too much.

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Robb: No, I think that's great and usually the folks who follow the show are pretty geeked on this stuff. I guess when we step back for so long we were just kind of looking protein, carbs, fat, insulin, glucagon, maybe talking lepton and you know, high carb was good, high carb was bad. When we peeled a bunch of that stuff back, it seemed like kind of a commonality whether we were talking about the intuit or we were talking about the Katavans or Okinawans which just this kind of like largely whole unprocessed food and going to bed when the sun went down and getting up when the sun comes up and you know, vitamin D and all these other things. And I guess in a lot of ways this gut biome story is just another layer to that whole picture.

When you start thinking about how – and you're already eluded to this, the bacteria, very promiscuous as you eluded in sharing genetic material to a degree that it even makes a speciation of bacteria really kind of dodgy from a microbiological standpoint because they can just swap DNA

like crazy. So it gets really dodgy about what even really constitutes distinct species.

Michael: Sure.

Robb: And then also you have regionality, people needing to adapt to the particular region that they're in, different animals and plants and microbiota that's going to be indigenous to the soil, the water, the plants, the animals and then just some sort of lottery wheel spin of whether or not that microbiota constitutes health or pathology.

I mean it's funny. It reminds me of Zen and the Art of Motorcycle Maintenance when the guy you know, went into science to try to like clarify the world and figure out that each time you ask one question, you create two or three more. The guy went crazy off that, was actually increasing the entropy of the whole universe trying to figure it out. And so I mean what's your kind of takeaway on this stuff?

To me, there's probably not going to be a really cut and dried story as to what's helpful with regards to gut microbiota? There might be but it may be a little bit more regional. It may be regional plus the type of diet that you're eating. It might be regional plus the diet that you're eating plus epigenetic factors like somebody born of non-vaginal birth, non-breastfed, 8 courses of antibiotics by the age of 5. Their gut biome health, what will constitute health for those people may be very, very different than somebody who's raised on a farm around lots of animals, huge biological load. Their immune system is dealing with and each of those is probably going to have pluses and minuses when we play all this stuff out.

Michael: Right. It actually reminds me of because breastfeeding is such an important part of establishing a healthy micro biome, we've actually been ruling out a study in the clinic where we take an adult population or we're trying to roll people from the adult population that have gut issues and bring them back to breastfeeding. And we've actually had overwhelming response from male participants to actually undertake in the study so it's pretty crazy.

Robb: Shocker dude. Do you get to pick [Cross-talk] you use or…

Michael: No. By the way to clarify, I'm kidding about that post. Don't call my office.

Robb: You'll have both tons of people wanting to sign up and people hating us for making fun of boobs. But as I've discovered with cross-fit, controversy is good so I'll just embrace that.

Michael: And there goes all my credibility…

Robb: Or just by being on this podcast pretty will destroy that.

Michael: I agree. I think another thing maybe to add to those different layers of your painting that I agree, all of those, there's also the issue of it seems that while bacteria populations can be different in different guts, there seems to be dysfunctional redundancy meaning if you're missing one bacteria then another bacteria can step in and carry out the functional role of that bacteria.

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So it gets very hard to say your gut is healthy or not healthy because of these bacteria because the bacteria can shift to kind of – they can compensate for other bacteria that may not be there. So it's not as simple as this one bacteria does this one thing and if they're deficient in this one bacteria, they're going to have a problem with this function. Because bacteria can fill a diverse range of different functions so there's this interchangeability of what function a certain bacteria population will play depending on other bacteria are there and who needs to fill that roles?

So it adds to the complexity of figuring this all out and I agree with you where I don't think it's going to be something that we can say you're deficient in this, you need more of that. And that's how we're going to fix this whole thing in a very straight forward manner which is you know, when I get around to some of the other things that I wanted to mention, you'll see that there's a lot of – there's no answer to this issue. We don't know enough of this issue or the data is very contradictory.

And I know people like to have definitive kind of take homes but I really want to get people what the science is showing because I want to save people from maybe falling to some kind of gimmick that's derived just by looking at one small piece of the data rather than looking at all the data and being able to be honest and say well, we can't really make a recommendation in your confidence because there's not enough agreement of the data to be able to do that.

Robb: Even so like resistant starch and homeostatic soil organisms have been very, very popular in paleo land maybe the last year, year and a half and I would say gaining momentum, gaining awareness, I would say that I've benefited from the introduction of resistant starch and some green bananas and cold potatoes versus just sweet potatoes and what not. But it's also looking like there's a huge variety of fermentable carbohydrates that can be either beneficial on one individual or potentially problematic for other people because there are these a lot of these fermentable carbohydrates falls under that fog map stories.

So what for one person is going to be really just phenomenal for their gut health and overall health maybe really problematic for another person which gets – again, not to beat on the evidence based medicine scene too too much but how do you construct randomized control trials around this? Now we have another layer of this thing where we not only are trying to match for lifestyle and age and gender and as best we can like genetics, being able to do some genetic screening, now we need to screen for the gut.

And while we're screening that gut, the gut biome is incredibly labile and if an individual goes on around of antibiotics, if an individual increases or decreases carbohydrate load if they alter the carbohydrate type so that it's either more processed or less processed so that you end up getting more activity in the small intestine versus more activity in the large intestine. It can completely change the whole story.

And how do you again construct a nice tight beautiful randomized control trial to get that tight beautiful randomized control trial to get any information out of that at all. You just can't. So with a lot of these stories, I think that it's building a mosaic, a really big picture and then doing the best clinical medicine that you can to try to figure out okay, where is this person at? What are our perceived mechanisms that may be occurring here and then what can we do about that? Raging from like increasing resistant starch or other fermentable carbohydrates all the way to like a fecal transplant in other situations.

Michael: Right. And I think it's definitely a challenge in – and that's why I like to look at these things kind of in a somewhat linear fashion because a lot of these issues really disappear after you adhere to some of the basic dietarian lifestyle principles. And it's really a hugely important part of

trying to see through all the noise which is yeah, this person has all these symptoms that look like a bacteria imbalance and they also have symptoms and what maybe a fungal imbalance.

But if you put them on a healthy diet that's low inflammation and has a good balance of macronutrients, those things, they go away completely. And also if they're sleeping enough and don't have you know, could be amounts of stress then a lot of those things tend to rectify because the system if put in I think anyway somewhat proximal of a healthy environment will rootly get itself back to neutral or at least as close to as possible and that's where I think the collision can come in.

Should they not get all the way back to neutral, the collision come in, try to identify what are the few things that are hindering them from getting back to that balance and try to balance those things out or remove those factors and then let the body continue on its process of just getting itself back to neutral so to speak.

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Robb: And then to your point like swinging back to the B12 story again, the initial nutrition sleep food exercise intervention may be great for most people to probably where virtually everybody needs to start but that may not be the finish. You can still based off lab values and clinical outcomes you may need to dig deeper and figure out oh well, we've got some sort of auto immune response occurring with the parietal cells and we have some data that would indicate that improving B12 status may end up fixing that.

Michael: Exactly.

Robb: Interesting. Makes your job easy.

Michael: Super easy.

Robb: And you know, it reminds me a little bit and I forget if you mentioned this last time or I was talking with Chris Kresser at one of his book signings but some interesting data that indicates like H Pylori maybe beneficial if kids are exposed to that at the age of 5 but then becomes pathogenic if you're exposed to it later.

And so that's just another really interesting piece to this that depending on when one is populated with a particular bacteria, that may be a factor or that maybe just again correlative. There may be another piece to this that actually makes the H Pylori pathogenic later.

Michael: Exactly. I think Chris and I are both – have spoken about that as has Moises, the author about that book the Epidemical Maps which was a great book. I talk about that in the AHS presentation. I'm kind of glossing over that in our discussion today because I think it was time before last when I came on we spend maybe like 20 or 30 minutes on that specifically. So in interest of time, I've kind of lapped that part out but definitely that's a really important part of this whole conversation and maybe using that as a segway.

One thing I wanted to touch on slightly in that regard was modern dirt versus old dirt as I call it. I'm looking at US children compared to those of – sorry, looking at Hadza children compared to those of Bangladesh, both are in dirty environments and both have significantly higher diversity which is good compared to westernized countries.

However, the Hadza are healthy and the Bangladesh population has significant higher rates of diarrheal diseases and if you look at the dirt specifically the Bangladesh population dirt is kind of like a slum. It's what I call modern dirt and the Hadza dirt is more that you would see a hunter-gatherers society where you're just enclosed approximation with dirt and animals. So I think it is important.

I think this was mentioned in the Paleo Manifesto book also that the type of dirt does matter. Where modern slum conditions where there's just poor hygiene, that's not necessarily the best kind of dirt but dirt that you would see in a more ancestral setting is a little bit better and I know those terms are vague but I think people probably can connect with what I mean there.

Robb: Yeah. And this is again I think another layer to this whole agricultural transition story that humans went from a mode of being largely mobile and would deplete the resources in an area and then move on and then those resources would kind of replenish where when we started living in close proximity with each other and with other animals even this eboli

outbreak which I think people have kind of lost their minds about the whole thing.

But as we encroach into different biomes and alter the way that those biomes exist then you can take something that was historically not a pathogen in human populations because we never really came in contact with it or didn't come in contact with it with enough frequency for it to get penetrance into the human population. Now we see that stuff happening. So again, just biological tradeoffs.

[0:45:00]

It's easy to try to make it into a right-wrong kind of story. Clearly civilization has all kinds of perks and upsides but there are just some other potential – in biology it seems like there's never free lunch. It's always a matter of tradeoffs like you've given up a little bit here to get a little bit there. I was helping out with a book signing here in Reno and some folks had some questions about like vaccinations and I just made the point that we clearly in my opinion and this whole really – the anti-vaccine people will come out in the droves.

But when you look at things like polio and whooping cough and a number of things, small pox you know, I think that vaccines have really combined with better sanitation. Hygiene have really remarkably decreased the disease load that humans experienced and in a very favorable way but that might also have a certain amount of tradeoff with it. Like now we're vaccinating against things like chicken pox and rotavirus which are annoying but generally not fatal.

Michael: I think we talked about that several months ago about the same thing where yeah, if you're – maybe a little over zealous to vaccinating again something that the worse sequel of the disease is two days of diarrhea.

Robb: Right. And then how much benefit are you potentially getting from the tuning of the immune system to dealing with something like that. Now, I don't have a good answer. I was more asking a question and at this public deal, there was a guy who apparently I find out later is a mathematician. He just got up and stormed down to talk when I said this. I wasn't saying one way or the other that it was a known entity but maybe we should just contemplate that there are pluses and minuses with these things and

kind of factor that into how we're administering medicine and public health and what not.

Michael: I agree. And I kind of have a middle of the road view on vaccines and sounds like somewhat similar to what you do and it's funny to see I was having a conversation with a friend of mine who's a pharmacist and the guy almost lost it when I was trying to propose just like a contemplating view of the pros and the cons and it was almost like this gentleman couldn't even think. He couldn't even have a slot about it because it was such an emotionally charged issue.

And I'm not saying one view is right or wrong but if you can't even think about an issue or question then you've got some serious problems with that science so to speak because if it's drove in emotion, how do you really sniff out any inconsistencies or incongruence's in what you're doing.

Robb: Right. And again within dynamic biological systems there's virtually never – not an upside or a downside associated with something. I guess like bulleted ahead is always a downsize but if you really don't like your life then I guess [Cross-talk] I don't know.

Michael: Unless you're Sly Stallone.

Robb: Exactly. Yeah. So yeah, it's funny the emotionality on that and maybe at some point later maybe we'll get you and Kresser and multiple – because I think actually Chris is much more conservative on the anti kind of vaccine story so it would be interesting to get multiple people on and kind of hash out some of the pluses and minuses of all that stuff.

Michael: Yeah. I will certainly jump in on that. I won't volunteer right now because that would give me more stuff to do but I do have notes on that and I've kind of data basing over the years and it's not something I'm actively researching right now but in all the research that I do, I do come across things related to that periodically and I've kind of been storing those away and at some point in the future I'll probably do something with that. So this is where I say I'll gladly participate but I'll probably kind of do so because this can be one more thing I already overloaded

Robb: Maybe we'll just wait for you and when you're ready to do that then we'll crack that nut open.

Michael: Sure. Yeah. Whatever works the other way. I don't have much of a social life now anyway so…

Robb: Welcome to small business entrepreneurship. It's awesome.

Michael: Right. So I have a little bit of stuff on IBD from the presentation but I'm thinking I might gloss over that. There was one gentleman who asked a question any information or suggestions for the microbiota after colon removal. And yeah, it's really hard to just answer that question in a vacuum so to speak.

But I did speak on your show last time I was on about IBD and if you mentioned it on the AHS presentation, so for that gentleman who asked that question you may just want to look into those things. But the short story is there is a bi-directional relationship between the immune system and the microbiota. And so it's not just to say that you can manipulate the microbiota in so doing – it's just a one way street to the immune system. The immune system also monitors and prunes and regulates and shapes the microbiota.

[0:50:12]

So I go through in the presentation the probiotics that have been shown to be most successful for IBD that this person may want to look at and also some of the anti-inflammatory options that are available. So without going to in detail, that I think this person can get some good information from the presentation.

But the thing I want to leave more time for was for discussing obesity and this kind of ties in with someone asks a question what are the implications of a high fat diet on endotoxins in the gut and I think that question will partially be answered in this discussion of obesity. By the way Robb, how are we doing on time? I think we're about what? 40 minutes in right now?

Robb: Yeah but I mean you call it. There's no time limit on my side really. It's whatever you're willing to put into this so we're good either way.

Michael: Okay, cool. So with obesity, there's a few things I wanted to talk about, one is bacteria, the second is starchy and fatty acids and the third is inflammation. And I think it's the inflammation piece that answers the

question but we'll work our way up to that. With bacteria like we talked about a minute ago, there's what's known as firmicutes bacteria ratio and there's one lab with a stool test that reports this ratio.

And I comment the lab for trying to be progressive but the thinking that an altered firmicutes bacteria ratio can cause obesity, it's something that was derived from animal model studies and when you look at the human studies that were trying to replicate this, you do not see consistent reinforcement of this. In fact, you find several human contradictions.

So I really don't think we can put any stop into this firmicutes bacteria ratio because again, it looks very promising in the animal data but if you look at the human data, you will find maybe 60% of studies show that it's valid 40% of the studies are a blatant contradiction. So that tells me that it's not as simple as that and I don't think we can really put a ton of stock into that ratio.

But regarding the microbiota in healthy versus obese, we do see it. There's consistently differences in what the microbiota looked like. It's just the differences aren't consistent. Does that make sense?

Robb: Right.

Michael: Okay. So I hate not to give people highly definitive but the nature of this particular beast is there's a lot we don't know and there's a lot that I think we're not going to have clear back or white yes or no answers for. So for making these firmicutes bacteroidetes ratio, I don't think there's really much that go on right now.

But their short chain fatty acids which we've also talked about Robb in the last time we went on we went into some detail about low carbohydrate diets maybe causing a pruning as you call of the microbiota and a decrease structure of short chain fatty acids. So the other side of the short chain fatty acid piece is people with obesity tend to consistently have high levels or often times have high levels of short chain fatty acids.

Now it kind of mucks the waters up a little bit because on one side of the spectrum, short chain fatty acids are good, protective to the colon and just healthy for a general intestinal function. On the other side of the coin, people who are obese tend to have high levels of short chain fatty acids.

Now there may be two things that are at play for why this happens. Of course people that are obese may be eating more or they may just be eating a lot more carbohydrates which may be providing more of the substrate to produce short chain fatty acids or it may be that the people who are obese have a microbiota that is much more efficient in extracting calories from food because remember, about 10% of our total calories comes from bacterial fermentation of which short chain fatty acids are part of that.

[0:55:00]

So if you were more efficient at fermenting food stuff and extracting short chain fatty acids from otherwise indigestible food particles, that may be an advantage in an area where there is not a lot of food. So it may be one or the other of those. But to kind of make things even less clear, we have shown that those who have obesity tend to have higher levels of short chain fatty acids but paradoxically the Hadza like we were talking a little while ago, the hunter-gatherers, they have very high levels of propionate

So you know, you see all these other populations that have high short chain fatty acids are obese then you see a very healthy hunter-gather population to have high propionate. May that be because they're eating maybe less calories…

Robb: In the westernized population wouldn't it be more like butyrate versus the propionate?

Michael: It's not always consist because I've looked for that. I looked to see could that be the case? And there's not a consistent elevation. Some other obesity studies have shown elevations of butyrate. Some have shown propionate. And that was the first place my mind went to Robb but there doesn't seem to be a correlation although there's some evidence to suggest there's propionate to acetate ratio has something to do with lipogenesis or lipolysis.

And I can't remember which way the ratio skews to feel which pathway but it was just something that was mentioned in one or two of the research papers but it doesn't seem to be something that has been shown to be a major issue yet. But anyway, there is all the speculation about that but since zoom out, yeah, there doesn't seem to be okay,

obese have high butyrate healthy have high propionate. It just seems to be a mixed bag.

Some obese have high butyrate or propionate and then some healthy have both high butyrate or high propionate. So the point I'm trying to make is there's a contradiction to or it's not like a one size fits all kind of thing. The other thing that's interesting about the short chain fatty acid piece is that low carbohydrate diets that will decrease short chain fatty acids are really good for weight loss.

So how this all kind of ties in is the Hadza had high short chain fatty acids and they were healthy but then some obese also have high short chain fatty acids.

So there's some disagreement there but what we have seen in terms of outcomes is when westernized populations go on low carbohydrate diets even though their short chain fatty acids decrease, the low carb diets seem to be superior for weight los in a lot of cases anyway.

And so how the short chain fatty acids play into that, it's a little unclear but what it may be doing is that these people may have an increased ability to harvest calories from their food and so depriving them of foods that have increased ability to energy harvest may be contributing to the weight loss.

But short chain fatty acid are only less than 10% of calories so I don't think that mechanism would be hugely beneficial and here's another reason why I don't think that depriving the body of the substrates needed to make short chain fatty acids to decrease calorie absorption is a viable mechanism for weight loss is because in other studies where they give probiotics, they've actually shown weight loss.

So you could say well maybe a low carb diet is cutting of the supply of short chain fatty acids and therefore decreasing calorie uptake from the gut. But other studies that are giving probiotic that increase short chain fatty acids have shown a mild amount of weight loss. So it's all kind of messy, murky and confusing and I'll give a few recommendations in a moment in terms of what people can do based upon clinical trials with this information.

But I have a feeling people are probably a little confused right now because there's not really a strong conclusion we can draw from that but is there anything there you want to jump in on or clarify?

[1:00:00]

Robb: No. It actually makes my head spin. So we've had done pardy come to Reno several times talking at the clinic, working with our folks there and he had some great studies that we're kind of looking at again because of his work with Stefan Guiney and like the neuro regulation of appetite. Looking more at this from kind of the left in signaling in the brain, neuro regulation of appetite, specific circuitry in the brain that responds to different macronutrients, it was interesting because then kind of played out this picture that a very low fat diet could be quite beneficial in various regards with neuro regulation of appetite and neuro architecture.

A ketogenic diet could actually be very beneficial in fixing broken neuro architecture in people who do not have a normal neuro regulation of appetite, the normal satiety society signals have been broken through whatever mechanism and that's where like this original question was actually kind of interesting because it was asking about high fat diet and endotoxins and endotoxins are pro inflammatory. They alter liver signaling. They alter immune signaling. Immune signaling has effects on the brain.

So we got not only the protein carb fat piece. We also have the byproducts of bacterial break down and fermentation of protein carbs and fat. Then we have potential inflammatory or anti-inflammatory signaling that plays into all that stuff. So it gets really complex I guess if I were to try to overlay some sort of template for making sense of this, it's what are those things that can influence the neuro regulation of appetite particularly when we're talking about maybe morbidly obese or people that have just had a heck of a time losing significant amount of body weight.

Why is it that those folks don't have these satiety mechanisms in place so they feel full of the appropriate calorie intake and other people are fine with that. They can eat almost anything and they end up stopping at the appropriate point. So there's something broken in the neuro regulation of appetite, protein, carbs, fat influence that, endocrinology influences that.

Gut biome likely influences that. The inflammatory signaling and the immune system and how you can pick which again like you said like this kind of linear thing and peeling the onion.

We have a couple of different maybe algorithms that we can go down trying to fix this stuff and so if one route does not succeed then we have some other routes that we can go down and try to succeed. I'm thinking about also the higher protein intake dramatically alters gut bacteria and somewhat argue in an unfavorable manner depending on the situation so that's a whole other interesting piece.

But yet we know that protein is highly, highly satiating due to peptide YY and leptin ghrelin interactions and pulses to kind of release so man it gets complex rather quickly.

Michael: It does. And to the question that person was asking about LPS and endotoximia and how that affects obesity, it's interesting because if you look at what weight loss trials have been maybe the most successful, at least in the ones I've examined, I'm certainly open to maybe there being some contradictions to this but I think the general trend shows that if you can restrict carbohydrates that tend to be the best for weight loss, they've done studies where it's been low cal or it's been low cal with low carb for example and it seems like when you get that extra little bit of the low carb that the low carb tends to be a little bit better for weight loss and they go through a number of trials in the AHS presentation outlining that data.

But to keep this from getting incredibly dry, the general trying to merge is where if you can push down the carbs a little bit it's better for weight loss which really contradicts the LPS induces obesity because you're going to be ingesting more fat which can potentially fuel more LPS. Right? You with me on that one?

Robb: And I guess the only thing that's a little circuitous about that is that fats, particularly saturated fats have a better binding affinity for lipopolysaccharide to pull it into hepatic circulation and administer it to the body. But then at the same time, a lower carbohydrate diet reduces bacterial load and so you would argue that LPS production would be reduced.

[1:05:00]

And that may be part of the story where a high fat diet plus high calorie is so incredibly injurious because you're not pruning back the bacteria and that may be why it's so – another layer of why it's so pro inflammatory. You're not pruning back the bacteria on the one hand but you're also providing a fantastic shuttle to move that LPS through the gut and into the system in the other hand.

So these studies have said that fat increases translocation of the LPS you know, totally true. But then I have not seen any type of a waiting to look at well if the low carb diet reduces bacterial load dramatically then is the total amount of LPS produced.

Michael: Right. That's a really insightful point. I like that. Because yeah like you're saying you may decrease bacterial load on the gut by going on a low carb diet which may kind of counteract the effects of any elevated transmigration. The other thing I think – and that's actually a really great point Robb. You're' not just good looking. You're also fairly smart.

Robb: Thank you. Sleep deprived too but my provigil is motoring me through. So

Michael: But the other thing that it just know worthy just to mention, in a lot of the animal trials anyway where they come to these conclusions, look at the diet and it's corn oil. We've fed these rats corn oil and bacon chips or something. The diet is just ridiculous from which they derived some of these conclusions.

But there is one another thing I wanted to mention along these lines and it's definitely a little bit more theoretical at this point but we've discussed how early exposure to germs is good and how that can kind of tone your immune system. And I think the last time we discussed the study published where they looked at pregnant mothers who worked on farms and they found the more exposure the mothers had especially earlier in the pregnancy and the more animals they had, the more protective of a role had on their immune systems. We discussed that right?

So among those same lines, as part of one of these studies, they also looked at what was called CD14 cells and they found that the more exposure mom or child had to dirt, the more expression of the CD14 cells they're worth. Now why that's potentially important is another study looked at normal weight versus overweight children and they found that normal weight children had a higher expression of these CD14 cells. And

these CD14 cells are part of the DNA immune systems ways to detect the presence of LPS.

So what we may be seeing is it may not just be the LPS itself but it may be the immune system and how good the immune system is able to identify at LPS. So people that have a healthier immune system but more exposure to bacteria from a young age may be able to better or more appropriately tolerate exposure to LPS.

Robb: Yeah. So the CD14 cells produce lipopolysaccharide binding protein which will remove – I forget what the mechanism is. All of that stuff ultimately ends up back at the liver. But when the LPS is associated with the LPS binding protein, it's non-pathogenic, basically gets dissembled and I forget all the deep metabolism on that. But without the binding then the LPS is able to cause inflammation in the liver and in an interlocking cascade that is pro-inflammatory.

So it totally makes sense and it actually kind of reminded me of part of some talks I've given where I talk about metformin and one of the effects that metformin has. It does a lot of really interesting stuff. It improves insulin sensitivity both at the muscles and at the liver. It down regulates gluconeogenesis but it also decreases the tendency for LPS to be pro-inflammatory at the liver.

So you know, and so it's kind of a pharmacological kind of validation of the way that our systems should be working. We should be insulin sensitive. We should have the right immune system tuning and all that type of stuff or at least that's kind of the way I've spun that.

Michael: Yeah, I like that and I think that it kind of suggests I think what we're both driving at, which is you can get really mislead, if you get too zoomed in you can get really mislead and that's why it's nice to zoom out and just look at basic principles, look at outcome studies because even though this LPS potentially shows this obesogenic potential, when people go in low carb diets for example, they tend to do really well in a lot of cases.

[1:10:00]

So that's why I always say that interventional studies, outcome studies in my opinion always trump mechanistic studies in terms of guiding an intervention.

Robb: Yeah. I totally agree. And I mean really that's the root of a clinical base medicine. There's another thing for the more evidence based medicine crowd. If we held ourselves to a lot of the standards that folks write a whole nutritional science to, physicians would not be able to use off label drugs for any day. Part of this is having an understanding of basic mechanisms and then thinking about that and extrapolating from that, well if this is good for this and this situation over here kind of looks like modified or improved by the application of this drug like using arimidex which is usually an anti-aromatase drug that's used in cancer therapies.

But it's also used at anti-aging medicine to block the conversion of DHEA into estrogen and so it tends to cascade more into testosterone. So if folks really, really get wrapped around the axle of this evidence based medicine story then anything for which off labeled drugs are used, it should be off the table and that usually causes people to kind of pause a little bit if they can contemplate that at all.

Michael: Yeah. That's a good point. I don't know that bit about arimidex. That's pretty interesting.

Robb: Yeah and a high dose of zinc citrate ends up having about 30-40% of the effects that arimidex so you have some non-pharmaceutical interventions that you can go that drives that away I think Chrysin has some mild anti-aromatase activity too so you can tackle that in some different ways. But again, I really – to your point, these outcome based studies I think – are a course of action that we can hopefully get some progress with.

Michael: Yeah, I agree with most of what you said. I lost you there for a minute and then I came back in so unless you said anything slanderous…

Robb: Only to myself if I did.

Michael: Well there's maybe one question I wanted to address from another one of the Twitter questions and then I have a few things on the microbiota in terms of manipulations for obesity to maybe kind of bring everything home.

Robb: Cool.

Michael: So two good things from Twitter, one I want to make was something about protein, how that affects the microbiota and we certainly see that

carnivorous mammals have the least diversity. Herbivores have the highest level of diversity. Omnivores are kind of in between the two. But when we look at our microbiota, it most closely resembles omnivores so I think if we're looking at a comparative mammalian analysis we're pretty good where we are in terms of an omnivorous diet in terms of how it manipulates the microbiota.

And if someone had asked what are the difference between homolactic and heterolactic fermenters on the gut microbiota, it's actually a really interesting question to me right now because what this has to do with is essentially what this is driving I think is the issue of D-lactic acid doses or people from too much delectate and I think Kresser had mentioned this a little while back with people who have sebo may want to be careful with D-lactic forming probiotics.

I actually read I think all of the relevant papers on D-lactic acidosis over the past few months just to kind of weigh in on the issue to see if it's something I thought was a big deal or not. And I think it's going to be an issue probably for the minority of people but for that minority of people what may be significant.

[1:15:00]

Because in certain people where this originally comes from is those who've had a jejunectomy. They've had their jejunums removed and it alters the normal transit of carbohydrate down the GI track and if too much undigested carbohydrates gets into the large intestine, those bacteria can produce D-lactic.

Well I should say if too much of the carbohydrate gets through the intestinal track too quickly, it can over feed certain bacteria populations some of which produce D-lactic as part of their metabolism and D-lactic if the levels become too high can cause brain fogs, ataxia, slurred speech, just a whole slew of neurological symptoms.

But it's important to mention that typically happens in infants or those with pretty serve gastrointestinal conditions. So it's not something I think everyone has to be cognizant of but if you've done all the other stuff that the standard recommendations that we've kind of mentioned; paleo diet; focusing on your lifestyle, exercise, stress, sleep; maybe you've tried some probiotic, prebiotics, maybe some starch, resistant starch that is; tested

for and treated any kind of gut infection; done some work to get your hormones balanced out if they're of balance.

If you're going through those basics and you still notice you have a lot of brain fog, then this may be an issue. And what you may have is a relative increase in what would be called a potentially homo fermentative kind of bacteria population meaning there's homo and there's hetero. Homo will typically only produce D-lactic acid. Hetero will produce L'lactic acid and D-lactic acid. In some organisms actually will convert L'lactic acid into D-lactic acid.

The main thing to be cognizant of is that too much D-lactic can cause brain fog. This is actually one of the markers that I've added into our gastro intestinal workup through the clinic. You don't have to do this through a private casual lab. Lab corp quest do offer it as a 24 hour urinalysis that can be billed to insurance. And I think there's some science showing this may be an issue in terms of how prevalent an issue might be or populations other than infants or those who've had intestinal surgery, I'm not sure.

I definitely think it would be for some. I don't think it will be for everybody. but again, for those that have a lot of intestinal issues and have a lot of brain fog, maybe it's something to be cognizant of and we've been running this marker for the past probably month in the clinic. I'm trying to see if I can sniff out what kind of correlated issues has the brain fog specifically.

And what these people want to do is they can do a low carbohydrate diet and then also potentially some sort of herbal and antimicrobial can help beat down the excessive population. Also propylene glycol which is found in a lot of cosmetics products and it's also a preservative found in some food stuff can also help gunk up the pathway that's needed to detoxify D-lactic so people who are exposed to higher levels of propylene glycol may want to be cognizant of that and thiamine deficiency may also fuel that process.

Thiamine has been used as a treatment and then finally there's the issue of using non-D-lactic acid in probiotics which would be something like Prescript Assist or these soil based organisms and that certainly maybe beneficial because there's been I think two either case reports or clinical

trials or just small group number reports have used D-lactic acid free probiotic as a treatment and they have been successful. So that's just some options kind of with the whole homo versus hetero lactic fermenting bacteria.

Robb: It's interesting. For myself, like that was – the first time that I went low carb, I've got to say like my whole life prior to that, literally as long as I could imagine, I felt like my head was stuffed with cotton and it was like the whole world was existing about 3 feet away from me. It was all percolating into a fog and then about 27 or 28 years old went very low carb and got into ketosis and it was just magic like maybe occasionally prior to that in my life, I had these moments of clarity.

[1:20:00]

Like if I was backpacking interestingly when I think back about it, it was actually in these almost like fasting kind of scenarios but the fasted scenarios were tough for me because I had these wicked blood sugar swings all throughout childhood and early adulthood. So this ketogenic state which is magic for me like I had amazing cognition. I had zero brain fog and whenever I would get in and start…

Michael: So was that the first time Robb that you – were you also going gluten free for the first time when you did this?

Robb: You know, all of that happened at the same time, yeah.

Michael: I don't want to cut you off but I do want to make one clarification which is there's a lot of causes of brain fog so I wouldn't say that this issue, the D-lactic acid would be the first issue that someone with brain fog would want to address. Brain fog is multi-factorial and just by getting food allergens out of your diet, you can have a huge impact on your brain fog. Sorry to cut you off but I just want to…

Robb: It's a great point but what I found is any reintroduction of carbohydrates were of any type of density were really problematic for me, even doing like a cyclic low carb diet was really hard. I hated the cyclic element of it because whenever I reintroduced carbs, it was just a problem and I've been looking at like iron overload and some different things like that, kind of a pro-inflammatory piece but I actually put in for the first time maybe

about four months ago, 4 or 5 months ago, Prescript Assist. And I've got to say that plus the resistant starch you know.

It's been a wiggling pathway getting healthier and healthier but I've tinkered with really dramatically increasing my carbohydrate intake while doing – I've bounced between Prescript Assist and then I'm now doing a big bottle of Primal Defense which was I think one of the first homeostatic soil organism products out there. And I feel pretty good and my digestion is pretty good and that brain fog is where or comparatively nonexistent.

Still, one of the frustrating things for me relative to being ketogenic is when I was in ketosis like literally I could go all day long and not eat if I needed to and I had zero cognitive fade. I was super solid on that, doing stuff like Brazilian jujitsu and what not was kind of a bare but I've talked about that ad nauseam on the program. But it's interesting. You actually linked a lot of pieces there because I had not really looked at het hetero lactate versus homo lactate kind of story.

Michael: Yeah. I'm probably going to write an article on that. I'll probably have a lot of articles and stuff coming out early next year. There's a lot of stuff on my plate right now so I've got a lot of really what I think is good information in text form I just haven't had a chance to kind of get it all cleaned up and try to get it out to people but I think that will be a really interesting article because there's definitely a few interesting little tid bits about that and yeah, I think for some people. And you would probably Robb an example of someone who've had more severe issues. You had pretty severe gluten intolerance.

Robb: Yes, severe gluten intolerance. You know, I never tested positive for celiac disease but my mom had celiac, definitely get wicked GI issues if I get even incidental gluten exposure. I think I had a little bit of like a dermatitis herpetiformis thing if I get serial gluten exposure. So had a lot of the pieces of that puzzle there and that was actually a question that I was going to ask you if you see this D-lactate issue you had mention it's usually prevalent in folks that have had significant gastrointestinal surgery but I was curious about its prevalence in celiac patients.

Michael: That I don't know. I had not seen that reported in any of the papers I had read. I don't believe anyway in terms of a certain percentage.

Robb: Right.

Michael: I think there was a slight – had mentioned on the imbalances created in celiac may set the stage for increased D-lactate because I believe there's a higher instance of small intestinal bacterial overgrowth in both inflammatory bowel disease and in celiac. But also the gluten free diet may fall underneath the most well recognized treatment which is a low carbohydrate diet for D-lactate acidosis.

Robb: Right.

Michael: Something else along the lines of brain fog that I just noticed personally lately I noticed if I do too much espresso which is kind of my drink, I get a little bit foggy in the head and I think I have a low level intolerance to the espresso being and which really sucks.

Robb: It does.

[1:25:00]

Michael: I can get away with small doses but too much, I kind of get a little bit spun out and I recently have been supplementing with a little bit of N-Acetyl-Cysteine which amongst other things can help to repair the blood brain barrier and I have to say for the past several days I've been doing much more espresso than I should and my cognitive function has been spot on. And likely that's kind of the mechanism there where it's probably still causing a little bit of inflammatory action in my intestines but it's not able to make its way through the blood brain barrier because the blood brain barrier potentially a little less permeable due to the N-Acetyl-Cysteine kind of healing that up.

Robb: Interesting. Very, very interesting.

Michael: Alright my friend well…

Robb: Do you want to do a little more? You want to wrap here and do another show in a couple of months?

Michael: Yeah I'm just going to say yeah. Maybe we should cut this as I'm sure people right now are probably getting really – hearing my voice. I may come on sometime in the near future and tie on some recommendations for manipulating the micro biome and obesity because I've done a pretty extensive review of probiotic, prebiotic, fibers and some other things that manipulate the microbiota and how they've been evaluated…clinical trials

and what kind of success people can expect to obtain from this intervention so...

Robb: Awesome. I dig it. I'm looking forward to it. Doc, where do folks find you on the inter webs?

Michael: I'm still trucking over at drruscio.com and hopefully we'll have this site revamped and up with some additional resources for people maybe the next month or two. It's been an uphill battle the whole way for sure but hopefully that will be done soon. And I did receive quite a number of people who left a message who wanted to apply for the podcast host. Thank you everyone for sending those over and I've kind of tabled that for a little while until we've gotten our website's finish. So website's the first thing. The podcast will be in the heels of that. So as soon as the website gets up and going then I'll probably be reaching out to a few people there and…

Robb: Okay, we're back. We almost made it through the show and then we had some breakdowns so we're back.

Michael: Where I'd lose you?

Robb: You were talking about tabling…

Michael: Right. Okay so yeah, I just tabled the podcast for a little while. We've got all the people's applications who applied so thank you to all those people and we've tabled it a little while until we got the website up and rolling. So as soon as that's going, we'll get that going and the other thing I just want to mention real quick was I'm doing a half day seminar for doctors healthcare professionals, nutritionist, it is illegible for a continuing education credits in October 4th in San Francisco on the gut.

So it will be kind of a way to consolidate all this gut information into a clinical approach in terms of how to identify what condition, the present, how to test that to objective, how to treat it, how to follow-up and trouble shoot, so I'm pretty excited about that and I think that's pretty much it.

Robb: Awesome. Let me know when that's ready to roll and we'll push that around so that folks will know about it.

Michael: Yeah. I'll shoot you a link. There's a web page up for it. I'll shoot you a link. It's October 4th so it's coming up and yeah, hopefully we'll maybe connect again in a couple of months and then finish some of these GI notes.

Robb: Very cool. Well doc, it's always great having you on. I'm stoked you could do it again.

Michael: Yeah, thanks for having me Robb.

Robb: Alright doc, we'll talk to you soon.

Michael: Bye. Take care.

[1:29:07] End of Audio