Paleo Solution - 254

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Robb: Howdy folks. Robb Wolf here, another edition of the Paleo Solution podcast. Six listeners can't be wrong. We have yet again the handsome, the devilishly intelligent Dr. Michael Ruscio. Doc, how are you doing?

Michael: Hey Robb, I can't believe you keep having me back but thanks for having me on.

Robb: I think this might be the fourth episode. So in Star Wars lingo, this is a new hope.

Michael: This is the one. This is where we make up for all the past wrong doing.

Robb: So what's new with you? I know you are busier than a one-legged man in an ass kicking contest. So what's going on?

Michael: I feel like a broken record I think every time we talk I mention that the new website is almost done but I really, really mean it this time. The new website I've code should be done hopefully by February 3rd, the coding and then hopefully it'll be up a couple of weeks after that. So I'm hoping maybe February the site will be up, we've been recording some podcast on so many different topics everything from Vitamin D to SIBO to thyroid disorders.

So the podcast hasn't launched yet at the time of this recording we're doing on January 13th but when the new site goes up the podcast will go up and definitely digging myself a plaque here. If you guys want to go over to try and ask questions, we're trying to make the podcast listener question driven.

So if you have questions, shoot me a note on my Facebook page or send a note over to the platform that will be on the website and we'll do our best to try to get back to you on that regard. Other than that, just reading a bunch and trying to keep what a few shards of a social life I have together and that's pretty much it.

Robb: Nice, nice. Well if you ever need a pinch guest host for that to read your questions, let me know. I would love to do it.

Michael: Oh you're definitely coming on. Absolutely.

Robb: Sweet, sweet. And we'll bring down property values anywhere I go. So I'm stoke with that.

Michael: I'm going to try to find the most socially awkward discussion and have you be the guy to spearhead that. i think you got the social grace for it.

Robb: Yeah, yeah. I will meddle through that. I guarantee it. So you've shot me a number of really interesting papers lately ranging from SIBO to vitamin D. Where do you want to jump in?

Michael: I guess let's start with Vitamin D maybe since we've talked about gut health before, so maybe let's swing over to vitamin D for a quick minute. Yeah, so I think me and you have a very similar kind of a philosophy in vitamin D which is not an extremist philosophy right. It's not get your levels to 80, 90.

And I think a lot of people in the paleo community are starting to lean toward that more conservative position. But I want to cover a couple of things that may help people understand why that position might be a good position to kind of be updating to. The most exciting about this just to kind of cut to the chase is there is a theory out there.

I can't say it's proven. It takes a lot to really lot to prove something but the theory definitely makes a lot of sense which is one of the reasons why some people may have low vitamin D or lower vitamin D even though they're supplementing or getting adequate sun exposure maybe because they have an infection that is modulating the vitamin D receptor in attempt to evade the immune system.

And what can actually happen there is these certain types of infections, intercellular infections specifically can compromise a new receptor and what ends up happening is vitamin D gets converted into its metabolite 1,25 vitamin D or calcitriol and what you'll see in lab work is someone will have a normal and maybe a lower range vitamin D. But the 1,25 vitamin D is actually high.

So if that person takes more vitamin D, they actually maybe throwing gas on the fire because at high 1,25 vitamin D again is kind of orchestrated by a certain pathogens to try to help suppress the immune system to evade eradication by the immune system.

Robb: So help folks and help me understand this a little bit. We know that vitamin D is a modulator of the immune system. It seems to have some pretty potent effects in avoiding a variety of different ailments including infection, including cancer. What is the 1,25 calcitriol? Is that actually an immunosuppressant relative to the non-metabolite form?

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Michael: Well and again this is where I don't think the mechanism is completely well understood. It's a bit more hypothesize as to exactly what happens. But it's actually the 1,25 form that goes into the nucleus via the vitamin D binding protein and then is able to dock to the vitamin D receptor. So what has the nuclear effect as I understand it is the 1,25 vitamin D.

And these bacteria seem to have the ability to produce ligands that essentially alter or down regulate the vitamin D receptor. And what ends up happening is or what may end up happening is 1,25 vitamin D goes high because the receptor is not working. And as the body continues, as more and more 25 vitamin D is shot at the 1,25 vitamin D in attempts to trip the receptor, that actually uses up the 25 vitamin D.

So that's 25 vitamin D go low, the 1,25 go high. And the 1,25 is going high because the cellular receptor is somewhat not responsive due to how the bacteria are modulating that. And then apparently that 1,25, that high 1,25 has a negative feedback or a reflexive inhibition to the immune system through how it modulates cytokines. So I know it's not a super neat answer but it's a little bit of a feedback group.

Robb: Got you. Got you. And then clinically that's where we could see people if they're supplementing aggressively with vitamin D like the Carlson's liquid drops or something like that and then we could potentially see these folks with some calcification issues particularly if they're K2 and vitamin A are possibly deficient or not keeping pace with the status in their system.

Michael: I think that's certainly possible. Although I haven't seen that the pattern on lab work where people come back with high 1,25 vitamin D and also have high blood calcium. Although I also have to admit I haven't been running. I've only been running the 1,25 fraction routinely for a few months now, so it's possible I just haven't come across that grouping of patients yet.

However, in some of the publish literature that same remark is echoed that you don't always see high calcium in the presence of high 1,25 vitamin D may be due to the fact that the effects of the 1,25 vitamin D are extra renal and potentially extra gastrointestinal.

So I think it's definitely possible but it doesn't change the recommendation that if someone is going to supplement with vitamin D they should always have a balance with K2, right? So at the end of the day, the recommendation's the same and it's a good lead into maybe outlining and I know you talked about this and others have. But just to quickly outline, we have great observational data showing that those that have healthy vitamin D levels are generally healthier. And those who have low vitamin D are not.

But the interventional studies don't match what the optional observational data are showing and so that tells you there's some kind of disparity there. So if people have let's say people with average vitamin D levels of 15 have a higher incidence of disease and people with average vitamin D level of 35 have less of that disease, well, if we go and we take the group of people that have the vitamin D level of 15 and we supplement them to 35, it doesn't translate always to that same health outcome. And there's actually, does that make sense?

Robb: Absolutely, yeah.

Michael: And there was a great systemic review published in the Lancet and that the PM ID number of people who wanted because I know they're going to ask is 24622671 and you can just punch that number into PubMed search box and this paper will come up. But it was a systemic review published in the Lancet. Again, the systemic review is essentially where you go and you look at multiple other studies and you try to summarize them to see what the data is showing.

I'll just quote because maybe this will help people and they can take it from the researcher's mouth instead of mine. The discrepancy between observational and interventional studies suggests that low 25 vitamin D is a marker of ill health. Inflammatory processes involved in disease occurrence and clinical course would reduce 25 vitamin D, which would explain why low vitamin D status is reported in a wide range of disorders.

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And they continued that results from interventional studies did not show an effect of vitamin D supplementation on disease occurrence including colorectal cancer. So again, this doesn't mean that there is not a one study showing that vitamin D works. There are studies that show vitamin D work. But when you look at multiple, multiple studies and try to get a big picture of summary, analysis of is there an over-all effect or not at least according to a very well-performed systemic review the results don't seemed to be great especially for non-bone related disorders.

And I think that discrepancy is there because the efficiency for vitamin D at least in some part may not be because of a true deficiency, maybe secondary to inflammatory process in the body.

Robb: Interesting. It's really reminiscent of the cholesterol-lipoprotein story where you know for ages we saw kind of discrepancies between high cholesterol, low cholesterol, some people who had heart attacks had cardiovascular disease had low cholesterol. And now that we're looking at the lipoproteins that carry cholesterol, we seemed to have a lot better picture that models the disease process.

So with the case of vitamin D, these folks look like they maybe deficient but in fact that low 25 vitamin D is actually just reflective of some sort of inflammatory process usually mediated by infection.

Michael: Exactly, exactly. And so i mean certainly I think for hopefully most people, if they are a little bit deficient in vitamin D, that may just because of the lack of time in the sun. Maybe a lack of dietary vitamin D and a small reasonable dose of sun or vitamin D ingestion will bring the levels up and everything will be fine.

But for people to have chronic health conditions and that are getting some sun and they're taking some vitamin D but their vitamin D still

doesn’t responding, pushing harder with their vitamin D supplementation maybe the exact opposite of what you want to do.

Robb: So from a clinical perspective, what are you doing? I know that this is going to have a huge range. I guess what are the types of infections that we’re seeing? Is it like spirochete like with lyme disease that are avoiding the immune system. What are the infectious processes that you're seeing with this at least potentially?

Michael: Well and that’s something that I'm still trying – so what I'm doing right now in the clinic is with patients that have this initial disparity between 25 vitamin D and 1,25 vitamin D in their initial blood work then I'm seeing how does it correlate with what certain infections, I'm tracking that data right now. And then what I'm really hoping to see is when we clear these infections that we’ll see the vitamin D bounce itself back out.

Now according to the published literature, these are specifically intercellular infections or infections that are also known to be cell wall deficient. So these are things like spirochetes, certain viruses, aspergillus, certain fungi. But lyme and the lyme co-infections definitely would be included in that like you eluded to.

And I've been doing some research to see if there's maybe like a panel that I could comprise of maybe common intercellular infections. But like many things, I think it’s a little bit too complex to just have one standard panel that you can run because there’s so many different pathogens. I think what ultimately is going to have to be done is the testing have to be individualized to what your suspicious of with that particular patient.

Like if someone has a lot of pulmonary involvement, aspergillus might, that would make a lot more sense because they're known to probably cause pulmonary complications. If someone has these paradoxical neurological complications, then lyme might make a little bit more sense to screen for because the symptoms fit.

So the short answer is they're intercellular infections but there's quite an assortment of them. But maybe the most common ones that people will see will be the lyme, lyme co-infections and then certain viruses.

Robb: Interesting. I was just doing some quick poking around, trying to see if there's any literature on syphilis and vitamin D status and just with the

cursory look at it, I didn't find anything. But I mean it's you know it's doing it well chatting with you. So that's something that I'd like to look at a little bit deeper syphilis being another spirochete organism so it would be interesting to see if there's anything there.

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Michael: Yeah and what I'm really curious to see is what kind of effect is – we see this imbalance with vitamin D, we find an infection, we treat the infection, what kind of effects I can have with the vitamin D because certainly it's been published. I want to see how this shapes out in the clinic a little bit more thoroughly. And there is a doctor that kind of pioneered a lot of these work his name is Marshall and he came up with something called the Marshall Protocol which is extreme vitamin D avoidance.

And I haven't looked into all the details of the Marshall Protocol. It seems a bit extreme to me from my first analysis. But maybe something between the Marshall Protocol, maybe something not quite as intense as the Marshall Protocol will be helpful for some of these people to have these conditions and I should also mention that there are agents that can be used to help restore vitamin D receptor competency.

Resveratrol seems to be a natural agent that has that capability. It's hard for me to say clinically because there's a few moving parts with the vitamin D which is the infection and its role in the vitamin D receptor and then also if they're co-administering anti-microbials to kill an infection also giving a vitamin D receptor modulator like resveratrol. It's hard to piece out what's having most of the effects right.

But resveratrol can help restore by vitamin D receptor competency and there's also a medication known as Olmesartan that can also modulate the vitamin D receptor competency. And the interesting thing about Olmesartan is it's been recorded that when people who take this drug they can have die off reactions like the reaction seem when killing an infection and it's theorized the reason for that is is because when you restore vitamin D receptor competency, the immune system that are functions and then can kill pathogens and now you have a die off reaction.

Robb: Interesting. Interesting. So now, you also – related to vitamin D, we're kind of shifting gears a little bit. You recently sent me a review paper on vitamin D status related to melanoma. Do you want to talk to folks a little bit about that?

Michael: You know I haven't had a chance to dig deep into that specific paper yet but there's a couple of tangental things I definitely would like to mention. Did you have a chance to dig into that paper yet?

Robb: Just again, probably a little bit beyond the abstract and not much beyond that. I mean it's a review paper. I did have a couple of dermatologist completely call up my hooha about it being a review paper and you know like well this is a randomized control trial and then I got into this kind of pissing match with them where I'm like you know review papers are what drive our CTs you assholes.

The evident space medicine crowd I mean clearly we need to function within evident space medicine but people seem to forget that this stuff starts from observation typically clinical notes. You get enough clinical notes together and people start thinking about maybe propose mechanisms and that usually where overview paper will come out. And these review papers will you know, add people in the related research areas will say well, we could construct a study that would look at this and it might answer these questions in this way.

And that's where you actually get to a randomized control trial or maybe even retrospective trial first. But this is part of a process. It's kind of maddening to me that these guys forget this. But yeah, anyway that was the story with that.

Michael: I share your frustration already and I haven't even been on the conversation firsthand and I'm already frustrated with that. But you know there's a couple of things maybe real quick here and I actually wanted to correct something I had said. I think the first or second time I was on your show, I made a comment about some hunter gatherers having vitamin D levels that were on the 90's to hundreds and that was actually - it's correct but it depends on the kind of units that you used.

And that the units that were referenced there, they were not the standard units that we used in the United States which are milligrams per milliliter. So I want to revise that. In the study I was referencing was of

the Maasai and the Hazbib and hoping I was pronouncing those right. And their vitamin D levels, so hunter gatherers, somewhat made of untouched, un-westernized people hunter gatherers modern day, their vitamin D levels came in about 46. So I think that bodes really well again for the conservative vitamin D camp.

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And then regarding some exposure for vitamin D, there's been some published evidence showing that there are certain health benefits that one can derive from the sun exposure that are not vitamin D mediated specifically. So meaning the people who got sunshine had health benefit and it didn't correlate with their vitamin D levels. And in the systemic review that I pulled this from, prostate cancer, non-Hodgkin lymphoma and also blood pressure seem to experience benefit from the sun exposure that was independent of vitamin D.

Robb: And you know what a potential mechanism there like in the alternative cancer treatment world, you have low-dose naltrexone which is an opioid modulator and as part of the whole vitamin D production pathway, we have the secosteroid which modulate the opioid receptors and different opioid compounds in the bodies.

So I mean there's definitely in my mind at least some pretty good mechanism of causation there. It doesn't seem too far-fetched that if we see some immune modulation via opioid with low-dose naltrexone that maybe we're doing something similar with vitamin D production.

Michael: Exactly, I completely agree and along those same lines I think it's really important to try to just replicate doing things in the most natural way as possible. We're vitamin D deficient because we don't get enough sun. The answer is not sun, a sun pill like vitamin D right. It's actually getting some vitamin D.

And maybe there is a good spot to just throw out there and I think I shot you a note about this maybe even a year ago now. The Endocrine Society put out a position paper regarding vitamin D and I went through the position paper and kind of dogged out what people could do in terms of sun exposure to try to get the minimum amount to maintain healthy vitamin D levels as outlined by the Endocrine Society.

So I should say these are not the Endocrine Society's recommendation specifically but I'm extrapolating from the Endocrine Society what they think the minimum value of vitamin D should be, what they feel X dose of sun exposure will produce in terms of changes in the blood levels of vitamin D. And from there, I've engineered recommendations for people getting sun exposure to Vitamin D levels where they should be.

So if you're wearing a bathing suit. So I broke it down to if you're wearing a bathing suit or if you're just exposing your arms and your legs, of course there's a lot of surface area difference there. So if you're wearing a bathing suit and you are vitamin D deficient, so your treatment exposure would be one minimal erythemal dose three days per week. So a minimal erythemal dose just means enough sun exposure to allow a light pinkening of the skin. So just enough to know you're in the sun.

So for everyone that's going to a little bit different maybe 20 minutes would be a good barometer but what you'd want to know is that later on that day or the next day there's a slight, slight pinkening of the skin you might have a slight tan line for example where the clothes stop and you can just tell you had sun exposure. Of course that's completely different from being red or being burnt right? Definitely don't want to get anywhere near that.

But if you're wearing bathing suit and you're deficient, treatment exposure would be 1 MED three days per week. And if you're wearing a bathing suit and you're a vitamin D sufficient, your main exposure would be 1 MED one day per week. And if you're just exposing your arms and your legs and you're deficient, your premium exposure would be 1 MED seven days a week, your main exposure would be 1 MED 2.5 days per week.

So hopefully that will help people kind of steer their exposure levels and how I would recommend maybe applying this is when you come out of the winter months and you're trying to formulate how much sun you should get, have your vitamin D levels tested to see if you're deficient or sufficient and then pick your needed dose from there. And then monitor yourself maybe every two months while you're kind of getting sun exposure.

I don't really think you have to worry about excess from sun exposure so I don't think anyone is going to see hyper vitamin D from the sun. And one of the pieces that's been published and I tend to agree with is if people had adequate exposure during the sun months, they should be able to store adequate vitamin D in their liver and fat tissue to ride it up through the winter which from a developmental perspective makes perfect sense. I mean how do our ancestors get through the winter if we didn't have a storage mechanism.

Robb: Right. Doc, this is going to be a probably an unanswerable and controversial topic but tanning booths. The cost benefit trade off of tanning booths like going in and getting a minute, two minute bolt of these things every once and a while to get that minimal erythemal dose. Thoughts, feelings, catastrophe I mean there was just another big position paper really linking tanning booth exposures with melanoma particularly malignant melanomas.

But when I looked at these papers, I have not yet seen any attempt to quantifying how long people are in the booths. Like typically it's just did they go or did they not go, the more frequently they went it seems to increase the likelihood of developing problems. It seems like the people who do go to these tanning booths, they're shooting for a jersey shore leather handbag kind of look. So which seems to maybe going beyond the minimal erythemal dose. What's your gut sense on that?

Michael: I'm really biased because I'm from Massachusetts so I was pretty much tanning from six months throughout my whole life so even as a baby I was pretty tan. Have you ever seen an episode of Family guy where Stewie starts tanning?

Robb: Yes. Yeah.

Michael: So that was me as a baby. But I mean definitely from the East Coast I know exactly what you're talking about which is some people just go way too much and even in the middle of winter, they look they just got back from Hawaii. And I wonder if there's any kind of bias in the sampling where people who have are tanning have a tendency to kind of overdo it with sun exposure and I think that would be really interesting to see that broken out as one of the details of the analytics of the study.

So my gut tells me there may be a little bit of sample bias there. I would be inclined to think that if someone was responsible with tanning use that they could do it in a healthy way. I think the problem is it's a slippery slope where people very easily can over expose themselves in a tanning bed. So I think there's something that one definitely has to be careful about.

I'm kind of on the fence on it. I'm not anti, I'm not pro. I think you can make a case for either way depending on the person and as long as it was done responsibly and reasonably you might be able to get away without a lot of – it wouldn't have much of a downside. I'm sure any dermatologist just listening right now is rolling their eyes at me. But I think it could be possible to do it halfway safely.

Robb: I've tinkered with it a little bit where some of these the booths like standard – I'll ask the gals what's kind of the standard deal and they're like start off with five minutes and then ten minutes then 15 and some people are spending 20 minutes in these things. And I end up spending, you know my first five or six exposures one minute in there. And then the next five or six exposures two minutes and then I top off it five minutes which interestingly it ends up giving me a little bit of a base tan, I feel really, really good after that and I am seriously at the skinny ends of those response curve with all that stuff.

And I'm really not trying to get tan, trying to stay in there the minimum amount of time so that when I leave the tanning booth I'm like I actually feel really good like I get a little bit of that endorphin feeling and I particularly do this if I'm heading to some place sunny like Arizona or Hawaii or something like that. I'm going to have a limited period of time to ramp up to exposure in that new environment. But I mean clearly there's a cause benefit deal that is kind of unknowable in that story.

Michael: Yeah, I think the way you're describing it that seem is reasonable to me. I think trying to do it from a minimalistic perspective where you get sun exposure naturally during the sunny months and then maybe a little bit here and there kind of ride out the winter would be a reasonable way to do it especially for not doing like the ultra platinum package that's the bed that you come out when you're literally like you have smoke coming off of your ears.

But hopefully those numbers help people in terms of maybe figuring out how much exposure they can get from a really conservative perspective. And then on the other side of the coin, there had been some books written about how importance of exposure is and recommending really excessive sun exposure.

And so, I think there is a spectrum here but I definitely think being a little bit more conservative overall is fine and you get that. I just wanted to chime in one more thing on the lab values. So if people are going to run these labs, there's a little bit of, I don't want to say controversy, but I think with vitamin D I think the range is maybe a bit broad.

I think Quest goes from 30 to 100 and over 100 is considered high. And I think many in our agreement with physician as 100 may be, no that may be a little bit too high for me to upper cut off, and the same thing I think holds true potentially with the 1,25 fraction where the 1,25 fraction at Quest and LabCorp range from about 15 or about 75 and I think the high end might be a little bit too high.

So that's just something for people to be aware if they're trying to piece this altogether or bring this information to their doctor and try to work together to make decisions based upon the lab values. And then the other piece that I see anyway in the small same thing that I had been doing at the clinic, people that come back high, everyone that I've seen high has been 90 or above.

So I've seen 90 elevate through 160 on the 1,25 fraction. So at least from the standpoint that I have been doing, it seems like that people are going to have a disparity, the disparity tends to be pretty, pretty sharp. I do have by example where that people have had health complaints and conditions coming in but that's what I'm saying anyway.

Robb: Maybe if you take away through people if somebody's having problems with getting the 25 isomer of vitamin D ramped up, then definitely get the 1,25 checked so that you've got some compare and contrast on that if the 25 is low, 1,25 is high, then we start thinking about some sort of potential infection. We need to track down a functional medicine doctor to maybe go after that, and then maybe what a rough ceiling might be 45, 50, is kind of the conservative top and that people should be gunning for.

Michael: For both of those, yeah I think that would be a good top end.

Robb: Okay for both, great.

Michael: Yeah. And again for both, I don't think there is a specific ratio but just look at the disparity. If you're low normal vitamin D and high normal 1,25 vitamin D, you might want to be a little bit suspicious. If you're high normal vitamin D and high normal 1,25 vitamin D, then you're pretty even keel.

Robb: So doc, you've had some interesting new stuff on everybody's favorite topic are poo and gut bacteria, what's new on the SIBO front?

Michael: SIBO is quickly becoming my favorite condition to work with. I don't know if that's weird to have favorite condition, but there's something about it I really enjoy and I just find myself getting pulled deeper and deeper. Probably because I find it so commonly. It's really remarkable how commonly I'm finding SIBO even sometimes in cases where it wouldn't be the top of my list of conditions I would suspect.

And one of the things we're working on doing right now is putting together a study through the clinic where I wish I could, but I can't really go into detail as to exactly what we're going to be doing at the clinic. There's a question regarding a certain type of treatment for SIBO and no one really knows if the treatment in particular works or not. And we're collecting data right now with hopes of publishing that data that will give us an answer one way or the other. If this treatment works, keep doing it, this treatment doesn't work save your money.

So that's something that I'm really, really excited about and if anyone listening is involved in research, they're Ph.D. or they're getting their Masters in research and they would like to get involved, I've got 100 people we're working with right now but the real bottleneck for me being able to do this is having people to assist me with the right ops and the IRBs and so on and so forth.

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And so I have the patients and we have the protocols and we're tracking the numbers, but I definitely need assistance with the other hand of

everything. So if there are people out there that would like to try to work with us in that regard, that would be most, most appreciated.

Robb: Very cool, awesome.

Michael: And then to get into some of the nitty-gritty, have you heard of the whole autoimmune piece involving SIBO?

Robb: Maybe I heard a bit or two about that, yeah.

Michael: One of the forerunning theories for what causes the underlying dysfunction that allows SIBO to occur is that there can be this autoimmunity in the gut that damages the cells, called the interstitial cells of Cajal. And these cells are essentially the regulators of intestinal motility. And when motility is thrown off, that allows SIBO to propagate.

And usually how this happens is there's a bout of acute gastroenteritis, food poisoning, stomach flu, a bout of throwing up, diarrhea, what have you, things like Campylobacter, Shigella, some of these more classical infections that can cause this kind of presentation, that's self-resolve in a lot of case or is knocked out easily with a quick round of treatment.

But then, people have some symptoms that persist and these symptoms are IBS-type symptoms, so constipation, diarrhea, maybe an oscillation between the two, bloating, distention, abdominal pain, and what happens is when the immune system comes in to kill that initial infection, it triggers autoimmunity in the gut. Now we've talked about the infection to autoimmunity connection before, right? With thyroid?

So we know that premise has already been theoretically validated and this autoimmunity forms against these cells called the interstitial cells of Cajal, which are essentially these neuromuscular bundles in the gut that regulate motility of your entire gastrointestinal tract, from esophagus all the way to rectum. These cells are even contained in the pancreas, in the bladder, in the ileocecal valve, and when these cells don’t work the right way, food doesn’t move through at the appropriate pace gets somewhat slowed in the upper half of the GI, that slow movements allow bacteria to overgrow, and then the bacteria overgrow and you can end up with SIBO.

Robb: You know I'm guessing that a little bit of this is where we see like a low-carb protocol being helpful with this where we're providing less substrate

for the bacteria to potentially overgrow, sometimes maybe we're removing some of the offending foods like what do you feel are the most common foods that are causing this autoimmune response?

Michael: Well, we don't have any published data in terms of foods causing this autoimmune response, at least not from what I've seen. The researcher that has really been pioneering this work, his name is Mark Pimentel, he is a gastroenterologist at Cedar Sinai in LA, and they've done a really good job of mapping out the specific antibodies and the mechanism and it seems to be after this bout of acute gastroenteritis or food poisoning. So the initiating factor seems to be that.

But that being said, there's another issue that affects these cells and that's inflammation in the gut. Because one of the main questions that people are asking now that this information is surfacing is can those cells, can my ICC cells recover? Can I recover from this underlying cause? Can I recover? Can I go on with my life? And I think the answer to that is yes because there have been published human studies and published animal studies showing that these ICC cells are plastic. They have the ability to regenerate or rewire themselves to maintain function.

And how this comes back to your question is one of the things that affects the ability of these cells to regenerate is how much oxidative stress and/or inflammation is present. And so the more inflammation is present, let's say you have a low-level gluten allergy for example, if you truly do and you have an inflammatory response to gluten, then certainly it's possible to think that that information would inhibit the ability of these cells to recovery/thwarting the ability of your motility to recover/thwarting your ability to recovery from SIBO.

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Robb: So kind of a perfect storm kind of scenario where it's not potentially one causative factor. And then we have some of these foods are immunogenic by themselves but then also contain FODMAPs which may goose things towards some kind of a SIBO type profile also. So again maybe not getting too reductionist with this, there may be a little bit of a big picture story there.

Michael: Right, yes. To come back to – and i didn't answer kind of the second half of your question which is some of the other foods. So, definitely the

FODMAP foods are kind of like a standard of care in my opinion for treatment of SIBO and usually the way that's brought into the whole piece is of course FODMAP foods feed and encourage bacterial overgrowth and so you want to be careful with those specially in the initial phases.

Now usually the way this is applied is first you engage in what is known as your killing phase. You're using some kind of agent to kill SIBO, whether it's an antibiotic or an herbal antimicrobial preparation. Once you're out of the killing phase and you've objectified that with lab work that the SIBO is gone, then you revert to more of the maintenance or preventative phase. And that's where the low FODMAPs come in.

And usually the best way to apply this is once someone has been stable for at least two to four months, then you can start to wean them off of maintenance therapies and more preventative therapies and the FODMAP will be one of those where some of you go into a FODMAP reintroduction and what I have seen is some people will, just like with the food allergy reintroduction, they'll have a couple foods that they really don't do well with, and a lot of other high FODMAP foods that they'll be fine with.

And so it's just about helping them identify what their triggers are. And then also along with the FODMAP reintroduction you can start to curtail some of your other strategies like the prokinetic. Prokinetics are agents that help to aid in motility. And like when someone's been stable for a number of months you can curtail them off of these prokinetic agents.

Robb: Interesting, wow. So doc, what's else? We've covered everything from vitamin D from skin to poo, what else do we need to cover today?

Michael: Well I think I just want to emphasize this because some of the questions I got from my Facebook page which were people being scared about SIBO being autoimmune and wondering if they have this lifelong condition they're going to have to manage, I got the sense that people were asking questions from a very concerned and fearful place.

And I think some of that comes from unfortunately there's a niche of people treating autoimmunity that make autoimmunity not to be this terminal diagnosis and you'll never be able to eat one piece of gluten for the rest of your life or you'll have damage for six months to a year, and

you're not going to be able to do this and you have to take all your supplements for that.

And it's not like you're getting anything away from people who are truly very gluten sensitive. But I think what happens is some of these people have gotten a little bit overzealous with the treatment and management of autoimmune conditions, unnecessarily overzealous, and it creates a lot of fear in people. And I don't think that fear is well validated for SIBO because I think it is recoverable.

And yes some people are going to have a harder role than others but I do think it's recoverable and these cells do have the ability to regenerate and recover. If you're out there and you have SIBO and you're struggling with some of this and you're going through a fearful place, I don't think that you need to go there, get yourself to a good clinician that they can help you through this and you'll be able to recovery and do really well. I just want to make sure to echo that one more time.

Robb: I totally agree with that. It's been interesting. I think one of the kind of distinguishing features of this kind of Paleo ancestral health template particularly when you stick a good functional medicine perspective into the treatment side, is that we've had such great success with autoimmunity. You know sometimes it involves some work, there can be some significant life changes. My wife's mother died at the age of 52 due to rheumatoid arthritis complications.

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And I met my wife three months after her mom died, and I'm haunted by what could've happened if I met Nicky nine months earlier because we have so many success stories, so much power with this, and Terry Walls just got invited to speak and be present at the huge multiple sclerosis kind of convention that the multiple sclerosis society is putting on. She's historically been pretty shunned by that group but she asked them point blank, she's like why now? Why do you want to talk to me now?

I think the search traffic that they'd been getting when they ask people where they've heard about or what they've been talking about, what multiple sclerosis treatment options, nutritional approaches, this kind of autoimmune Paleo thing is discussed 15 times more frequently than like the Swank diet or the kind of vegan approach combined. And people

seem to be reporting some really remarkable success with it. So you know it's this little bit of decentralized market-based approach.

I would love to be proven wrong on a ton of this stuff if somebody could show me some protocol, some approach that works better because spin on a dime, shift gears, adopt something else and run with it, but this kind of comprehensive approach to addressing the gut, addressing photoperiod which includes vitamin D status, looking at exercise, looking at non-immunogenic foods, there just seems to be something very powerful in this evolutionary template.

Michael: Actually, that's such great news. I love it when there's this grassroots sort of movement just steers decisions that are being made at the top based upon consumer demand, that's awesome. That's a big hatchet to everyone who's listening and everyone in the community because they're driving that just through their own ability to pursue good information and make good choices. I mean that's awesome.

Robb: Yeah, it's definitely the way it should be playing out. The Atlantic actually yesterday had a piece on intestinal permeability and rheumatoid arthritis I believe. So it's hitting some mainstream, it's finally getting some stride and hopefully I become superfluous in this discussion and the people with the actual credentials to talk about it should talk about it. We need to tackle this in an integrated fashion and got to start with the gut first most likely if we're going to have any success. But it's exciting.

Michael: And it kind of reminds me of the conversation that I was having with a friend of mine. He's a naturopathic medical student and we were talking about ways to try to get this study together that I'm wanting to publish in the clinic. And I thought he said it really eloquently where he said something along the lines of there's going to be a libertarian sort of movement in research where people like me, I'm not a huge academian, I'm not associate with a major hospital.

We're going to start publishing information because we're asking questions that a lot of these guys at the centers aren’t asking but my gosh, someone needs to be asking these questions and publishing these data. Because if they're not going to do it, then someone else has to step up and do it. I think what we're starting to see happen also in this

community and that to me is really exciting because now we'll have more of a hopefully strong scientific foot to stand on.

Robb: Right. So it moves from anecdotal spot – from anecdote to clinical medicine and then m maybe we catch a strong tail wind and get a good RCT on this material.

Michael: Yeah, and I think is we have more and more people flooding in to this space, the community is going to grow and there's going to be more resources available. Years and years and years ago there may have been only a small handful of Ph.D.s that were really into this sort of thing but now, gosh there's probably thousands and thousands and thousands who are really into this movement.

They are going to want to collaborate with different alternative clinicians and get together an start publishing and yeah, now we kind of take some of the power out of the monopoly so to speak, the research monopoly, so now we're able to distribute it back to the people.

Robb: Which ironically there's a book, I don't want to wax too political on this because this podcast needs to be about carbs, protein, fat and abs otherwise people are going to lose interest if you start talking about like actual solution related to politics and economics then people freak out. But there's this book called Healthy Competition put out by the Cato Institute and they detail a way of even doing theoretically what the FDA does on drug discovery and doing it in a largely market-based format and it would be very inexpensive.

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We would have a lot more effective drugs available, it would cost much less, and this democratization of research and discovery is what you're talking about here. It's very cool.

Michael: Yeah. This is very cool and very exciting and I am just excited to see how things continue to progress over the next number of years.

Robb: Yeah, me too. Well doc, what else? We're getting long in the tooth. Anything else you want to wrap up with folks?

Michael: Two quick things and hopefully they will be quick. One is definitely super quick. I had a few people ask on my Facebook page about the ileocecal

valve and how that's involved in SIBO. Of course the ileocecal valve is just the valve between the small intestine and the large intestine. And of course anatomically it's important because the bacteria of the large intestine can reflux into the small intestine and that's what can cause SIBO. It's one of the things that can cause SIBO.

And so I know that there's a lot of speculative information about ileocecal therapy and that's not something that I've really seen a lot of solid science behind although I respect any provider that's doing that sort of work. I haven't seen a lot of evidence showing that those interventions actually do work. They might but what I can say is that the ileocecal valve has one of the highest densities of those interstitial cells of Cajal of the entire gastrointestinal tract.

So if you fix motility reason will suggest you will fix your ileocecal valve instead of having to do other more exotic things that are supposed to be targeted directly at the ileocecal valve, if you're able to fix your motility then you should see the ileocecal valve function regained or restore itself.

Robb: Okay. How about some things like squatty potty, you know just addressing the anatomical position of dropping the dews, what are your thoughts on that?

Michael: I'm sure it couldn't hurt. I haven't read anything about that in the context of SIBO but certainly doesn't cause anything to do that and I just really don't think it hurting so yeah, I would say why not.

Robb: Cool.

Michael: And then the final thing is something really interesting also actually, either truly interesting or my life is just really boring and it's just interesting to me. But there is a SIBO symposium held at Cedar Sinai late 2014 and they discussed some new information to overseeing about potential connection between SIBO and weight gain.

And what's been published or what's starting to be found is that when people have both hydrogen and methane positive SIBO, that correlates with obesity and correlates with weight gain and I emphasize that because often times we see study showing this correlates with obesity but it doesn't necessarily correlate with weight gain.

There's a big jump between base population and the population of people that are 5, 10, 15 pounds that they want to lose. So this association seems to be valid for causing a marginal weight gain or significant weight gain. And again what the data has shown is that there is a significant jump in BMI when people have both types of SIBO rather than just one.

And what the thinking currently is, is that, well I guess a quick primer, with SIBO you could have hydrogen positive or methane positive, and this just means that you have bacteria that produce hydrogen gas or methane gas. And the way this has been hypothesized in terms of the mechanism at play is that when bacteria secrete hydrogen, that hydrogen gas that accumulates is essentially poisonous to that bacterial colony and regulates it from overgrowing.

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However, when there is methane producing organisms also present, the methane organisms eat the hydrogen gas in order to make methane. So now the hydrogen gas that should've accumulated to counteract or poise it off overgrew with the hydrogen colony is now being consumed by the methane gas so that regulator is now gone and the hydrogen producing colony overgrows, and you have a significant overgrow and now that was increased energy harvesting the gut which has been correlated to cause the weight gain.

Robb: Interesting. So from a practical standpoint, what are folks doing, what are you doing on a clinical setting to kind of address that stuff?

Michael: And that's the interesting piece because there has been some published interventional studies. I love where we're going with that which is just one thing that have a fancy mechanism that you can just spit out there, but you have to be able to say, or it's like they'll be able to say that if you treat that you actually have a favorable outcome. And in the published literature it's been shown a reduction in total cholesterol, a reduction in LDL cholesterol, and an increase in insulin sensitivity. That's been tracked.

And then recently, I had a patient that came in with both hydrogen and methane positive SIBO and we put her on an autoimmune Paleo diet and we treated her for SIBO, not at the same time. First is the autoimmune Paleo diet then we have actually figured out the SIBO diagnosis and

started treating her for SIBO a few months later. She lost 53 pounds over the course of about five months.

And in my newsletter, this will be going out in a week or two where I actually sat down with her and we discussed her experience, and again there is no way I can know for sure but I think about half of her weight loss came from the diet and half of the weight loss came from the SIBO because when we put her on a diet, we saw the initial rapid weight loss, like it's often seen when people switch to autoimmune Paleo diet. And then her weight loss kind of started to decelerate and stabilize until we put her on the SIBO treatment, and then she started losing more weight incrementally over the next few months as we treated her SIBO.

So I think she got maybe 20 to 30 pounds from the diet and then another 20 or 25 pounds from treating her SIBO. And of course, her gastrointestinal symptoms all cleared at the same time.

Robb: Interesting. So we're getting kind of an order of operations take care of potentially the food, food lifestyle features first and then we start working up the nutraceutical, pharmaceutical line if we need a heavier hammer to maybe stir the treatment course.

Michael: Yes, I can't tell you how vitally important having some kind of algorithm follow is. I think a lot of doctors out there are searching for a way to put this all together and I say that specifically based upon feedback that I received, and I'm not sure if I mentioned this on the show but I taught a half-day workshop on management, diagnosis and treatment of gastrointestinal disorders in San Francisco.

It was a great workshop, we sold out, we're standing room only, and I was super happy with how everything went because the feedback we got was just remarkable. People were saying it was the best seminar they had ever been to and I think it was because I laid out a framework for – okay you have all these tools but you don't need to use them all at once, you don't need to use them all for every patient, and here's a logical algorithm to work through with these things so that you don't waste money and you don't over treat and you're efficient with your care.

Robb: That's awesome, that's fantastic. Doc, I'm just really, really excited for the work you're doing. I just feel like you're becoming an absolute cog in this whole scene and you've really improved my understanding of all this stuff

and I feel like I had a decent steeping in all this and you've really raised the bar on our understanding both on the mechanistic level and then particularly in finding good treatment algorithms again so that we can find some replicable ways of tackling this and aggregating data and comparing notes so that we can continue to help people more effectively, it's awesome.

Michael: Well thanks a lot Robb, coming from you that means a whole lot, so thank you.

Robb: My pleasure. I'm looking forward to episode 5. How do folks track you down on the interwebs and we'll need to shoot off some fireworks and some party favors with the new sites launched, how do folks track you down right now?

Michael: Now the best way is the website which is drruscio.com. That will be hopefully shortly with a whole lot of cool bells and whistles and still talking way in the book and the seminar that I mentioned is being picked up and sponsored by a huge company and as I've been told, not officially yet but hopefully will be put on a national platform in mid to late this year. So, if there are practitioners listening that want to try to get some information it will be available there and maybe a more advanced version on my website at some point. So some cool stuff coming down the pipe here.

Robb: Awesome, awesome. Well doc, thank you again for all that you're doing. I'm looking forward to the next wingding.

Michael: Awesome Robb, thanks so much.

Robb: Okay, take care. Bye.

Michael: Bye.

[1:00:42] **End of Audio**