About My Guest

My guest for this episode is Beth O'Hara. Beth O'Hara is a Functional Naturopath and Functional Genetic Analyst specializing in Mast Cell Activation Syndrome, porphyria, glucuronidation, and genetics. She holds a Doctorate in Naturopathy specializing in Functional Naturopathic Approaches and a Master's in Marriage and Family Therapy. Beth found her own root causes contributing to her chronic health challenges and has been able to heal. After decades of seeing traditional health care practitioners, she felt like she was at the end of her rope. The medications often made her worse. Being given the wrong supplements didn't help either. Finally, she discovered a three-part approach that helped her get well: Genetic Analysis, Functional Naturopathy, and Emotional Wellness. Combining the three approaches allowed her to explore genetic root causes underlying her own MCAS. She analyzed her lab testing to better understand imbalances in her body and chose supplements personalized for her individual needs. Stress reduction techniques form a big part of her self-care as well. These things all allowed her body to heal. Since then, her passion is working with others with MCAS to find their root causes and support their healing. Beth has been in practice for over 10 years and has helped hundreds of clients. Together, she works with you to identify your unique root causes, analyze your lab results, and develop a customized healing plan based on your needs, goals, and lifestyle.

Key Takeaways

- What is glucuronidation?
- What are xenobiotics?
- What is the connection between bilirubin and glucuronidation?
- How does glucuronidation impact bile acids, hormones, prostaglandins, coritcoids, fatty acids, and fat-soluble vitamins?
- Where in the body does glucuronidation take place?
- What are the genetic factors involved in glucuronidation impairment?
- What is the connection between glucuronidation and chondroitin sulfate, heparan sulfate, the extracellular matrix, and TGFb1?
- What are some of the substances to be avoided to take pressure off of the glucuronidation pathway?
- What role does glucuronidation play in phenolic or salicylate sensitivity?
- How might intolerance to CBD be a clue for glucuronidation impairment?
- What is beta-glucuronidase? How does it impact glucuronidation?
- What is the connection between glucuronidation and the blood brain barrier and neurotoxicity?
- How does glucuronidation impact the clearance of mycotoxins from the body?
- What are some tools for optimizing glucuronidation?

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Transcript

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[00:00:01.06] *Welcome to BetterHealthGuy Blogcasts, empowering your better health. And now, here's Scott, your Better Health Guy.*

[00:00:14.10] *The content of this show is for informational purposes only and is not intended to diagnose treat or cure any illness or medical condition. Nothing in today's discussion is meant to serve as medical advice, or as information to facilitate self-treatment. As always, please discuss any potential health-related decisions with your own personal medical authority.*

[00:00:34.27] Scott: Hello everyone, and welcome to episode number 109 of the BetterHealthGuy Blogcast series. Today's guest is Beth O'Hara, and the topic of the show is glucuronidation. Beth O'Hara is a functional naturopath and functional genetic analyst specializing in Mast Cell Activation Syndrome, porphyria, glucuronidation, and genetics. She holds a doctorate in naturopathy, specializing in functional naturopathic approaches, and a Master's in Marriage and Family Therapy.

Beth found her own root causes contributing to her chronic health challenges and has been able to heal. After decades of seeing traditional health care practitioners, she felt like she was at the end of her rope. The medications often made her worse, and being given the wrong supplements didn't help either. Finally, she discovered a three-part approach that helped her get well. Genetic analysis, functional naturopathy, and emotional wellness. Combining these three approaches allowed her to explore genetic root causes underlying her Mast Cell Activation Syndrome.

She analyzed her lab testing to better understand imbalances in her body and chose supplements personalized for her individual needs. Stress reduction techniques formed a big part of her self-care as well, and these things all allowed her body to heal. Since then, her passion is working with others with Mast Cell Activation Syndrome, to find their root causes and support their healing.

Beth has been in practice for over ten years and has helped hundreds of clients. Together she works with you to identify your unique root causes, analyze your lab results, and develop a customized healing plan based on your needs, your goals, and your lifestyle. And now my interview with Beth O'Hara.

[00:02:23.23] Scott: I recently had Beth on the show on episode 101, and usually don't have guests back quite so soon. But then she approached me about sharing this topic, and I just couldn't resist the opportunity. I not only wanted to learn more myself, but I wanted to share it with all of you as well.

The show is going to be full of meaty conversation, so apologies in advance to our vegetarian listeners. Parts of this discussion may seem a little deep at times, but you don't want to miss the connections that will get made throughout our conversation. Thanks again for being here, Beth.

[00:02:52.20] Beth O.: Thank you, Scott, I'm so excited to share this because this pathway we're going to talk about isn't taught in much depth in practitioner courses or in detox training. And I think this can be a major game-changer for people who are having detox issues and trouble tolerating things.

[00:03:11.26] Scott: As someone who's had major detox issues, I'm excited to see how much this changes the game, so this is going to be a fun conversation. We did talk about your personal journey and how you got to doing the work you're doing today in episode 101.

So maybe we can start with you sharing with the listeners, what aspects of chronic illness you work within your practice? And what are some of the challenges that people have that they reach out to you to help them on their own health journey with your work as a functional naturopath?

[00:03:39.28] Beth O.: Sure. So I know you know that my journey started with my own chronic significant health issues, so I was fairly debilitated. And I saw over 50 practitioners and spent over $150,000 trying to find answers. Everyone kept telling me I was the most complex case they had ever seen, and they didn't know what to do with me. But it was through learning genetic analysis really and diving in the biochemistry because I had to figure it out for myself.

I figured out I had Mast Cell Activation Syndrome and histamine intolerance, and I really specialized in those. I worked quite a bit with mold toxicity, with autism spectrum disorders and people that just fall through the cracks - that's who I was, I always fell through the cracks, and that's what I really specialize in now, are people that blanket protocols just don't work for and they're ready to do the deep work and dive in and find out what's going on that root level.

[00:04:39.10] Scott: And I think it's beautiful when our own healing journeys then can help other people, and that's certainly been your journey, and now you're helping lots of other people as the result of your own experience.

When did this whole topic of glucuronidation first hit your radar? And what drew you - I know you've done a huge deep dive here - so what drew you into looking at its importance in chronic illness and to start connecting the dots.

[00:05:03.00] Beth O.: Well, really, I was preparing for the Environmental Toxins in Genomics conference that we had in Denver, and it was about a year ago Bob Miller approached me about presenting at that conference, and asked me which detox pathways I wanted to cover. And I run a really busy practice, I love doing research, but I thought well let me be easy on myself for once, and I'll pick acetylation, which also is a histamine degrading pathway, so it was one I'm presenting on.

And then I said let me do glucuronidation - I don't know much about it, and in all of my detox courses I've taken and practitioner training, there are usually only a couple paragraphs written about it, it can't be that hard, and so I thought I was choosing an easy subject. Luckily I started the research well in advance because as I was diving into it, I started with toxicology textbooks, very little written in there. So then I had to go to all of the research articles and research studies, which I know you know takes a lot of time.

And I was pulling articles, pulling articles, describing glucuronidation in the human body, and these articles kept saying glucuronidation is probably the most important phase two detoxification pathway. What? How could this be? We always talk about glutathione; we talk about methylation. Some people talk about sulfur and sulfation, but I've never really heard anybody talk about glucuronidation, and getting into it is extremely critical.

And I think it's why for some people that detox protocols keep failing with them, this is why. Learning this has completely changed my practice and how I'm approaching detox support, and I'm just on a mission now from what I've uncovered to get this information out there because I think, as practitioners, we need to know about this, we need to be addressing it. And as patients also, we need to be bringing it to the attention of practitioners so that they get on board. And that's what I'm hoping we can do today, is help bring that education out there because this is crucial.

[00:07:18.09] Scott: So cool, I'm so excited about this conversation. As part of detoxification, we have phase 1 and phase 2. Phase 2 has all of the components that you just talked about, so we have glucuronidation; we have sulfation. We have the infamous methylation with its wonderful marketing department; we have acetylation. And so help us understand at a high level, what do each of these things do? Why are they important in the context of a chronic illness discussion and in our health recovery?

[00:07:46.02] Beth O.: So if we zoom out just to orient people to how detoxification works. All toxins go through the same detoxification pathway, where we're talking about chemical toxins, metal toxins, or bio or mycotoxins, those toxins made from pathogens like viruses and bacteria or fungal growth in the body. And if they're going to be

addressed by the liver, they come through the phase one which is like the CYP that's the CYP450, we also have PON1 which some people may have heard of, those pesticide breaks down.

And that takes these toxins and makes them into an intermediate, and that intermediate is actually even more toxic than the original compound in most cases. Then we have phase 2, these different pathways you've just described, and their job is to quickly conjugate this intermediate.

So conjugate just means they're adding a compound to the end of it to make it more water-soluble, less toxic so it can be excreted either through the kidneys, through the urine or out through the bowel through the stool. And glucuronidation is responsible for 40 to 70% more of toxin elimination, so that's why this is so huge, and it's involved in a large majority of medication breakdown also.

[00:09:16.27] Scott: So let's then talk a little more specifically about glucuronidation. So you mentioned that it's 40 to 70% of our detoxification from a phase 2 perspective. What exactly happens during glucuronidation?

[00:09:29.09] Beth O.: So there's a whole pathway that occurs leading up to producing something called a glucuronide, and that's just a type of compound. And so the whole point of glucuronidation is to take the glucuronide and conjugate it onto the toxin. So that again, it's a safer compound in the body, and it can be excreted.

Glutathione is doing a similar thing, glutathione is getting added to a compound, or a methyl group is getting added to a compound to make it less toxic. Here we have a glucuronidation compound added to that chemical, so that we can excrete it and break it down. Just that so many toxins are the type of compound that binds preferentially to those glucuronides; that's why it's so critical here.

[00:10:28.27] Scott: So let's then talk about xenobiotics in the context of glucuronidation. So what is a xenobiotic? How much of our toxic burden do we think xenobiotics comprise? How much of a role does glucuronidation then play in detoxification in terms of what makes a chronically ill person chronically ill? And is it a key player in terms of improving our terrain, so that over time we have a terrain that does not support these microbial overgrowths?

[00:10:57.20] Beth O.: So xenobiotics are any chemical that's not naturally produced in the body. So this can be medications; this will be toxins like BPA and plastics or dioxins in bleached compounds; all kinds of things that are created as pollutants include pesticides, anything like that that's produced not in a living organism, that's produced by man-made processes. Many of these xenobiotics are highly toxic; if we even just take fragrance, it's still common.

So fragrances in scented candles, it's in plugins that people plug into the wall, deodorants, lotions, shampoos pretty much any personal care product you pick up at a regular grocery store is going to have fragrance in it. Those fragrances are immune disruptors, they're hormone disruptors, and they cause DNA damage. Even those that are the type of xenobiotic they're extremely toxic, and we don't think about it, but they're not regulated.

And this is a huge piece of chronic illness and a big reason why autism is on the rise. Why Mast Cell Activation Syndrome is now as common as between nine to fourteen percent of the population, which is a huge number of people dealing with this. And it is a massive player; we are living in a toxic world, and then when we think about the amount of mold toxicity was exposed to, it's just a type of biotoxin. And we've seen from Dr. Klinghardt's research that EMFs electromagnetic fields produced from our cellphones, our Wi-Fi routers these different means, are increasing that mold growth potentially as much as 600 times.

And so those mold toxins are extremely hard, they'll clog up all the detox pathways as well. So we put all this together, our bodies can't keep up, and everyone really needs a detox protocol these days just to have, even if they're healthy to maintain good health and if people have chronic health issues, got to have a good detox program to be able to get well.

[00:13:22.08] Scott: If we think about improving health as improving our terrain so that over time, we're less toxic. It's sounds like glucuronidation is fairly critical in any program that's attempting to optimize and improve our biological terrain.

[00:13:38.25] Beth O.: I think we've definitely got to be taking it into account. And we've got to individualize these approaches, because people have different genetic predispositions, and then people have different exposures. And so we need to be looking at which pathways need more support for people, and glucuronidation is the heavy hitter. We actually need to be looking at that even more attention than we were already giving methylation and glutathione.

[00:14:09.21] Scott: So if we extend then beyond the xenobiotics, we know glucuronidation plays a role in many other bodily processes. And so some of those can be metabolizing bilirubin, it's involved in bile acids, in hormones, prostaglandins, mineralocorticoids, glucocorticoids, fatty acids, fat-soluble vitamins, salicylates, serotonin, melatonin - I mean this is a huge list, so let's talk about some of these in more detail. Talk to us a bit about the importance of bilirubin and bile acids in the context of a glucuronidation conversation and optimizing health.

[00:14:46.10] Beth O.: Sure. So bilirubin is often measured on a complete metabolic panel, and just standard blood work, you kind of get that routine checkup with your doctor. And it's a marker of how this glucuronidation could be working, how an aspect of it could be working. And it can also give us information about liver health and function. Bilirubin functions as an antioxidant in the body; it also has mast cell stabilizing properties.

And so we don't want that bilirubin to be low, we don't want it to be at the bottom of the range or tanked out. What we'll also see that can clue into a type of glucuronidation issue is when bilirubin stays chronically at the top end of the range, and it doesn't have to be above the typical lab range which is at the top end. That's telling us that one of the glucuronidation enzymes called UGT1A1, that's the one that breaks down the bilirubin.

And if that's not functioning properly, and it stays just slightly chronically elevated, then we call that Gilbert's disease or Gilbert's disease, which in Western medicine is considered a benign condition. I'm really questioning that because

there's a huge overlap with people with chronically elevated bilirubin and detoxification challenges, and a number of people in my practice have this issue; I don't think it is benign.

[00:16:21.21] Scott: And so then the connection between glucuronidation and the bile acids?

[00:16:26.20] Beth O.: So bile acids are really important for the elimination phase of detox, that's phase 3. Where the phase 2 conjugates, which again is just where the toxic intermediates have been bound up to make them less toxic.

Then the bile acids help delivers those toxins to the intestines so they can get excreted. Glucuronidation is a part of that process that connection goes from glucuronidation to the bile acids, but then glucuronidation also breaks those bile acids down for recycling, so that they can get used again and not build and build and build. If those bile acids build, then we could end up with gallbladder issues or liver issues.

[00:17:11.08] Scott: So is that then to say that if someone has impaired glucuronidation and they're not able to move these toxins into the bile and into the intestines. But even if we're taking lots of binders, for example, we may not really be able to fully benefit from them because we're not really getting the toxins to the place where they would bind with those materials?

[00:17:31.19] Beth O.: Exactly, we can be stuck a little further up the pathway, and that's why we want to think about these pathways as a whole and map out what each client needs. So some people need more than just a blanket protocol.

What's generally done is let's give binders, let's give some glutathione; we'll do just some general liver support you should be good to go. I think we can drill in. We really have tools now to drill in deeper for people, customize these protocols for people. So when I get clues at their glucuronidation issues, we're going to really focus on that because I think that's where blanket detox protocols are backfiring on people.

[00:18:14.26] Scott: So let's look then at the connection between glucuronidation and hormones prostaglandins, which are involved in inflammation, corticoids. The impact of less than optimal glucuronidation appears to be pretty far-reaching from what I'm hearing in this conversation so far. So tell us about these and what happens to them when there is an impairment.

[00:18:35.16] Beth O.: So when we don't have glucuronidation activity, we can get a buildup of estrogens, we can get a buildup of thyroid hormones especially reverse T3, so that's something to keep an eye on in the track. And that's another pattern that I look for in my practice, is do we have chronically sub-elevated, so not outside the range, but sub-elevated bilirubin. Is there high reverse T3? Is there estrogen dominance? I start to wonder if this is a glucuronidation issue.

So these hormones aren't going to get broken down, we can also get an elevation of the glucocorticoids. And so as that goes up we're going to have higher cortisol; we're going to have issues that come along with that metabolic syndrome and so on. And then we've got the prostaglandins that glucuronidation breaks down, serotonin is converted to melatonin, and melatonin is a type of hormone glucuronidation breaks both of those down, so I strongly suspect that this is involved in serotonin syndrome. When people are getting toxic high levels of serotonin from certain serotonin medications or serotonin supporting supplements, so this pathway is just really far-reaching.

[00:20:03.07] Scott: Is there a connection between glucuronidation and the fatty acids like our Omega as the fat-soluble vitamins like A, D, E, and K and if so, what is that connection?

[00:20:14.11] Beth O.: Exactly. So glucuronidation is also required to break those down. So we think about taking vitamins in and getting used in processes in the body, but then they also have to get broken down, and then the metabolites are broken into to get excreted. I think this is a big part of what's going on with people that are having trouble tolerating vitamin D, vitamin E, vitamin A, I even have some people that struggle with vitamin K.

When I first started seeing people react to vitamin D, I didn't know what to make of that, especially because vitamin D is such a great mast cell stabilizer, it's a great immune support. I think it's building in toxic levels, and I've had people come into my practice who they were only taking maybe a thousand and two thousand IU's of vitamin D, then we measured because I always follow up on it. And their vitamin D levels were like well above they're around 130 for the D25 OH, which is astronomical, too high. Then again, another good place that this points to an issue with the glucuronidation.

[00:21:26.26] Scott: So where in the body does this glucuronidation process take place? So the liver seems like an obvious place, but is it limited to the liver, are there other places, other organs glands where this also occurs?

[00:21:38.06] Beth O.: So the expression is mostly in the liver, but also throughout the rest of the body in smaller ways. So some in the kidneys, even the stomach, the colon, the small intestine, the brain. The skin, the lungs, even the adrenal glands, all of the sex glands can undergo some glucuronidation activity, but the majority of it is happening in the liver.

[00:22:01.23] Scott: When we have an issue with glucuronidation, is it always that there's an under-functioning, or can there be an over functioning of the glucuronidation pathway?

[00:22:11.08] Beth O.: It's a great question, Scott. I typically see the under-functioning, and right now, what we know with the genetic analysis of this pathway is that typically, the variants are slowing down the activity. But we could have an up expression or an upregulation of any of the phase 2 pathways. And if we think about how these pathways, the speed of the phase of these detox pathways are working.

Well, anything about all three of them, and we don't want to speed up phase 2 faster than phase 3 can keep up with, and we certainly don't want to speed up phase 1 faster than phase 2 and phase 3 can handle. So I think of it in that way in terms of what's happening in relation to each other. So we wouldn't want to go in and stimulate

glucuronidation, without having, for example, the bile, the kidney, and the binder support on board to handle the excretion process.

[00:23:13.23] Scott: You work a lot with functional genetics, and so talk to us about the genetic implications here. Are there specific genes involved when there are glucuronidation impairments, what is the role of the UGT enzyme that you mentioned in terms of glucuronidation, and how many people in the chronic illness community have impairments in this realm compared to the healthy population or control.

[00:23:38.00] Beth O.: So this is the long pathway, and it starts with a gene called HK1 that produces glucose 6-phosphate. And some people may have heard of G6PD, so glucose 6-phosphate also feeds into that pathway. So if people have issues with the HK1, the very beginning of the glucuronidation pathway and they have G6PD that could compound that issue.

And so that G6PD makes NADPH, which is crucial in the body for energy production, antioxidant detoxification recycling. I think that's an important piece to recognize that we've got a branch off there into two different pathways. And then we go through a few intermediate phases, some genes one is called PGM1 and PGM3 and 5, and then there's one called UGDH. So these are kind of building the glucuronide compound as they go along, and then we get into the UGT's, and there are 13 different types of UGT's in the human body. The UGT1A1 is the one that's been the most talked about, and that's the one that breaks down the bilirubin.

So that's involved with Gilbert's, Gilbert's, how everyone pronounces that. And that one's really critical, but the other ones are just as important. We're still learning a lot; there's a huge amount of research to be done. But for example, the UGT1A3 is the one that's involved with steroids, hormone breakdown; a lot of medication breakdown also does a little bit of bilirubin breakdown.

And then there are others like the 1A7 breaks down phenols, which includes foods that are brightly colored blues and reds like berries, supplements like curcumin, resveratrol these are phenolic compounds. Salicylates are forms of phenolic compounds. And so we can really drill down looking at the genetics that which of these enzymes might be affected.

[00:26:00.12] Scott: So in the chronically ill population compared to the healthy population or the control population, are there significant differences in the mutations or polymorphisms in these glucuronidation genes?

[00:26:16.29] Beth O.: We don't know for sure yet about the generally healthy population. Well, what we do have is a database of over 35,000 people with chronic health issues, and we can look in that database and see how common some of these are. And so for the UGT1A1 if we just take that, some of them variants are very common, but then because in each set of genes or multiple SNPs that we look at, multiple wheels.

And some of the snips under the UGT1A1 it's like 0.1% of the chronically ill population have variants there. And if we drill through all of them, some of them again it might be 1%, 2%, 5%, 30% it just depends on specifically which SNP we're looking at. And while we're talking about these UGTs, it is interesting to connect that there is a UGT enzyme that breaks down porphyrins. So we did this last time when we were talking about the heme pathway in porphyrins, and this is also involved in porphyrin breakdown.

[00:27:33.23] Scott: Let's talk about the connection between glucuronidation and things like chondroitin sulfate, heparan sulfate, the extracellular matrix, which is a big conversation and should be in recovering health, and even things like transforming growth factor-beta 1. How do those fit into the conversation?

[00:27:51.14] Beth O.: So part of the glucuronidation pathway, this is that UGDH enzyme that we talked about just a minute ago; it's the step right before we get to the UGT's. And what's so interesting about that is that is involved in the creation of the sulfate compounds that we're talking about. So the chondroitin sulfate that's critical for joint health, and the heparan sulfate. So those are both important like you're talking about in the extracellular matrix for the health of that, and so that we can get nutrients across the cell membrane.

What's so fascinating about this to me is that heparan sulfate is also used by the mast cells to stabilize themselves. And so when I'm evaluating genetics for people that come in with Mast Cell Activation Syndrome, it's so much more complex than just looking at a few genes that are involved in histamine breakdown or histamine receptors.

We have to look at all of these other pathways that link in with what the mast cells need and so that UGDH is one that's always on my radar to see can this person even create the heparan sulfate that they need. And also, when we see patterns on micronutrient testing, the intracellular levels, if we see a pattern of high blood serum levels or if we see moderate serum levels, but very low levels inside the cell, it starts to make me wonder about that extracellular matrix. And does that need more support, are people able to make these sulfates that they need?

[00:29:28.23] Scott: Transforming growth factor-beta 1 one is commonly known in the mold and Lyme community based on some of the work in biotoxin illness and mold illness. What's the connection between TGF-beta 1 and glucuronidation?

[00:29:41.25] Beth O.: So the UGDH enzyme there it's actually upregulated by TGF-beta 1, and so it'll make it go faster. I don't know the full implications of that yet; it just came up in the research, so that's something that's on my radar and something I think we need to track and find out more about. Also, can up in the research that that enzyme can be downregulated, which means it won't function as well when we don't have enough oxygen in the body, which can happen either because of heme pathway issues, so we can't make the oxygen delivery molecules like hemoglobin. Or if people have airway issues like airway obstructions because the dental plate is too narrow, or they have sleep apnea and those kinds of things.

[00:30:31.20] Scott: So is an elevated TGF-beta 1; is that potentially also something that makes you think about exploring glucuronidation, or are we not there yet?

[00:30:41.18] Beth O.: It's on my radar, it's something that I'm working on piecing together, it makes me wonder if we're speeding up. So we talked before about upregulation, downregulation if we might be speeding up this enzyme using up a lot of NAD because that's a cofactor there, we have to have NAD. But I don't know exactly what happens yet if we have a buildup right there before we get to those UGT enzymes, so...

[00:31:08.10] Scott: Stay tuned, everyone.

[00:31:09.24] Beth O.: Yes.

[00:31:10.15] Scott: So you mentioned the heparan sulfate being used to stabilize mast cells, and my understanding is that heparan sulfate is also involved in coagulation or the thickness of the blood. So is there a connection between mast cell activation and hypercoagulation, and is that the result of impairments in glucuronidation potentially that are leading to lower levels of heparan sulfate?

[00:31:34.10] Beth O.: Yes, that's a great question. It's another one that I think we're still exploring; I see people with Mast Cell Activation Syndrome kind of on both sides of the spectrum. Some people bleed way too easily, and then some people are having coagulation and clotting. You especially see that in women with menstrual pain, I think there's a connection with that an iron dysregulation. I think so many of these factors play in together, and again we're not really clear yet on the role of coagulation and glucuronidation.

[00:32:07.15] Scott: What's the role of glucuronidation in terms of how pharmaceutical drugs are cleared from the body? And what are some of the medications that we need to think about that might be impacted by impairments in glucuronidation?

[00:32:21.05] Beth O.: So a vast majority of medications are broken down by this pathway, and the research shows that it's anywhere between 40 to 70 percent. And we have these wide ranges, so we have 40 to 70 percent of pharmaceuticals; we have 40 to 75 percent of antibiotics. One of the questions I had looking into this is why such a wide range, and it's because this pathway is so critical there are xenobiotics we don't even know how they're detoxed yet, but it's assumed that they come down these pathways.

So in the medications that are cleared by this pathway, aspirin, and any other salicylate, so aspirin is salicylic acid. Also, the NSAIDs like acetaminophen, naproxen, or benzodiazepines like Ativan, valium, valproic acid, there's a drug called Meprobamate, and steroids are cleared by this pathway. Also, propranolol, which is a beta-blocker, there are a number of others.

The reason is it's critical to look at this pathway, especially if people are prescribing, or if you've had some subclinical elevation of bilirubin. Then if your glucuronidation pathways have slowed down, these drugs can build to toxic levels very quickly. But we have the tools now to start looking at this, and this is why I'm so big on educating about this and helping to get to some more practitioners and patients radars, so we can prevent these drug reactions that people are having as much as possible.

[00:34:05.16] Scott: Beautiful. We know that glucuronidation plays a role in clearing compounds from the body like nitrosamine from cured meats, tobacco, rubber, pesticides. Heterocyclic amines from blackened or fried or barbecued or browned foods, a lot of the polycyclic aromatic compounds, the xenobiotics we talked about, the PCBs all these substances are really important and can lead to a toxic body, and then detoxification being really critical to regain our health.

So how much of a difference do you think we can potentially make in our health by better supporting glucuronidation to improve detoxification of all of these substances? Do you think that dialing in glucuronidation in a protocol leads to small shifts or larger shifts based on your clinical experience?

[00:34:55.14] Beth O.: I think dialing it in can be a make or break for people in terms of again whether that detoxification protocol is going to work. And where I work with people, where I start with everyone that comes into my practice is let's clean up your environmental exposure, let's clean up what you've got in your diet. So I do a full audit, we audit the personal care products, we audit cleaning products, we look at food diaries what people are eating.

We want to take the load off the pathway, and especially because we're so loaded up. I think about my great uncle, and his name was Wilford, so Wilford he and his wife Ethel, so we have these great old-fashioned names; they took care of me a lot as a child. I remember him coming home every day for lunch, and he would drink buttermilk and have a can of sardines for his lunch, and he worked on roofs. And so he was always out there working with all of the tar and the chemicals, and he smoked two packs of Pallmalls a day.

And he lived into his 80s when he died of melanoma; he didn't die of lung cancer. But when I think back, he didn't have the same exposure, so toxic exposures his whole life that we have. And so we've got to take the load down, we've got to take the load off of this pathway especially when we're looking at how much is cleared through it, and we're looking at the ongoing issues that we're having with chronic Lyme and people not recovering, chronic mold toxicity all these kinds of things.

[00:36:40.00] Scott: What are some of when you're looking at the people's personal care list and their food log and things of that nature, what are some of the common thread offenders that you're seeing relative to this conversation?

[00:36:54.23] Beth O.: Just foods in general, processed meats. People really like to grill out, and we don't think about that charring, but that charring actually is fairly toxic, it can do DNA damage and that charring itself that's a compound that has to be broken down by the body, glucuronidation breaks that down. I think about how much pesticides the person might be consuming, especially if they're eating conventional apples, blueberries, peaches any of these foods that are really important to buy. Organic carrots, and so I give people a list of the top pesticide residue foods to make sure they're at least getting those organic.

And then a couple of things that aren't food-related, but most people may not be thinking of is rubber products are broken down by this pathway. And so if children have jaundice issues, and it could be related to this pathway, rubber baby bottle nipples could be a problem. I didn't use to ask about this, but it's on my radar now.

Are people using rubber condoms, or are they using latex condoms? Because whatever touches the skin we absorb. Even things like rubber balloons, that's going to come through this pathway. And then otherwise the nitrosamine and the cured meats; people who are eating things like ham, deli meats, bacon then we want to look at that and see again what's the total load, so I'm doing a total toxic load assessment for that person.

[00:38:36.04] Scott: Let's come back to the conversation about phenols and phenolics. You mentioned some of the substances that might contain these so tea, grapes, coffee many of the berries. Some supplements like you've mentioned quercetin and resveratrol and curcumin. If someone's reactive to phenols, does that reactivity potentially reduce or even resolved when we support glucuronidation?

[00:39:02.14] Beth O.: It can definitely get better, and this is where again we've got to look at the whole picture because COMT is also involved in breaking down those phenols. And so if people have those COMT variants, and then they've also got glucuronidation, they're likely to have a much larger issue with these kinds of compounds. I think this is what's going on with a lot of people who aren't tolerating herbs and herbal supplements well.

I see that in my practice, and it may be that they're tolerating certain water-soluble vitamins like B vitamins very well. And then they try some curcumin, or they try some quercetin, and they've got this double glucuronidation and COMT issue so they just can't break those down.

Another thing I see more and more in my practice is this salicylate intolerance, where people are struggling with salicylates in food. So it used to mostly be when you looked at histamine, you looked at oxalates, you looked at glutamate levels. Now we're having to look at salicylate levels in foods for people as well, and that's dependent on glucuronidation and glycine.

[00:40:21.26] Scott: Beautiful, yes, that was actually my next question. Some of the more sensitive people that I've interacted with salicylates were a big issue for them. And so is there then a connection between salicylate intolerance and Mast Cell Activation Syndrome? Or the production of histamine? And is that also one either by better supporting glucuronidation or introducing glycine, is that one that you also see reduce or resolve?

[00:40:50.07] Beth O.: Right. So you're exactly right so that salicylate can trigger Mast Cell Activation Syndrome, as can oxalates and glutamate, so we have to think about all of these in terms of people with mast cell and histamine issues. I do see it improve, and I see it improve when we make sure that glycine is supported, sulfation is a big part of breaking down salicylates also. So we have to make sure that sulfation is supported, and we support this glucuronidation pathway.

One of the things that are happening with genetic analysis information becoming more easily accessible online is people are making simple equivalents. So they're looking at well if I have a CBS variant, then I can't eat sulfur foods. And I'm having more people come into my practice who either saw that online and stopped eating sulfur foods on their own or they had a practitioner tell them to stop eating sulfur foods, and then their health dramatically got worse.

And one of the problems there is that we need that sulfur again for stabilizing the mast cells, breaking down the salicylate, so then they end up with this massive intolerance issue. So instead of just to stop eating sulfur foods, we need to support the pathway, so it works well because these detox enzymes aren't isolated; they feed into each other. So glucuronidation is going to feed into acetylation and sulfation, they all can work in tandem depending on which toxins being broken down.

[00:42:34.12] Scott: And supporting sulfation for those that maybe are reactive to some of the sulfur compounds. Are we talking about molybdenum or something else?

[00:42:42.14] Beth O.: Right. So we can do molybdenum, I like to do urine testing of sulfite and sulfate levels to see what's happening there and that conversion. And then I also like to check the methylation panel, and check the actual blood levels of things like homocysteine, methionine, cysteine see what's happening in that pathway and see if people are flowing down too fast through the CBS and then we can actually support that homocysteine going back up to methionine if we need to.

So finding where those blockages are, and supporting the pathway with the cofactor. Sometimes people are taking too much B6, and they're forcing that pathway too fast. So there's so much we can do to target for people. And then when people have lost all of their sulfur foods, they can get them back on slowly sometimes just by introducing like 1/2 of a broccoli floret; this is what I did for myself because I lost all of my sulfur foods at one point, I was down to 20 foods. And then like 2 or 3 days later I did a whole floret of broccoli, three days later two florets of cauliflower, just slowly build it back on board.

[00:43:57.02] Scott: So wrapping up on the salicylate conversation, you mentioned glycine is that supporting the salicylate sensitivity because it's somehow interacting with glucuronidation? Or is it via a different mechanism?

[00:44:10.10] Beth O.: It's a different mechanism. So this is a great example of how a lot of times, we have these pathways working in tandem. So for salicylates, we need glucuronidation, we need glycation or glycine, and then we also need sulfation and sulfur compounds because they're all involved in that.

[00:44:32.03] Scott: So when we have impaired glucuronidation, let's talk about the vitamins, the fat-soluble vitamins like A, D, E, and K. Does that potentially also lead to toxicity of those substances, as you mentioned some of the pharmaceutical drugs that that could happen. And then when we support glucuronidation and that leads to increased clearance, might we then need at some point an increased dose of those fat-soluble vitamins?

[00:44:59.12] Beth O.: Right, exactly. So I think with people with glucuronidation issues, we have to be careful with how many fat-soluble vitamins we're piling on at one time. So if somebody has a glucuronidation challenge and if somebody's got a lot of mycotoxin issues. If they've had chronic Lyme, then I'm already assuming that this pathway is clogged up.

And so I don't want to jump them on high-dose vitamin D, and this was a good case for a slow increase to see what people can tolerate. Because you may find that 5,000 IU is the person is fine with vitamin D, but then suddenly we add in a little bit of vitamin E, which is also a fat-soluble vitamin, and then we've tipped the bucket, and they can't handle that load anymore.

[00:45:47.28] Scott: I was one of those rare people that felt significantly worse with CBD when I tried it several years ago. And so how might intolerance to CBD be a clue for a potential impairment with glucuronidation?

[00:46:03.29] Beth O.: So this is a really great point, Scott because CBD is so popular these days and lots of people are trying it. But CBD is also one of those compounds that's broken down by glucuronidation, and if this pathway just loaded up with toxins or if somebody has genetic variants in this pathway, they may not do well with CBD until the load is taken off, and the pathway supported. Can I ask if you had problems with mycotoxins?

[00:46:34.06] Scott: Oh, absolutely, yes.

[00:46:35.25] Beth O.: And so that's another, many mycotoxins are broken down by this pathway, so this is a pattern I see frequently is people with mold toxicity, we can usually assume that this pathway is going to be challenged because of the sheer number of types of mycotoxins that are broken down here. There may be even more than we know about at this point because, again, this research is early in terms of mycotoxin breakdown and glucuronidation.

[00:47:06.27] Scott: Let's talk about beta-glucuronidase. So tell us a little bit about it, how it might lead to further problems from a detoxification perspective, where does it come from in the body, what might cause elevations of beta-glucuronidase and what do elevated levels potentially tell us?

[00:47:24.23] Beth O.: So beta-glucuronidase is produced in small amounts in human cells, but mostly where we see it elevated is when we have pathogenic bacteria in the gut. And so this is tested on many stool tests, tests for beta-glucuronidase.

So this is a really important part of the entire glucuronidation picture because what this enzyme does is it takes, if we step back and remember we talked about that glucuronidation takes these intermediate toxins, conjugates the glucuronide on there so makes it less toxic, and then it gets excreted into the stool.

Beta glucuronidase in the intestines when it's produced by these pathogenic bacteria comes in, and it breaks that bond, so then it basically just undoes the glucuronidation, so we have this toxin freely floating again and available to be reabsorbed through the intestinal wall, and recirculate. And so beta-glucuronidase greatly increases the toxicity picture for that person when it's elevated.

[00:48:38.13] Scott: So even if we have glucuronidation that's not significantly imperative, we have this microbiome imbalance that's leading to higher levels of beta-glucuronidase, where essentially negating our own healthy glucuronidation.

[00:48:54.05] Beth O.: Exactly. So even if there are no genetic variants, even if we don't have mold toxicity, somebody's elevations of beta-glucuronidase, then they're going to have toxins recirculating. Especially we see the association with this with increased estrogen levels and estrogen dominance. So estrogen is one that is really associated with beta-glucuronidase issues because remember again, estrogen is one of those compounds that is broken down by glucuronidation. So if it gets this glucuronide compound attached to it, it gets into the stool, and then the beta-glucuronidase comes in and breaks that bond apart, that estrogen again is free-floating. So we see this associated with some forms of estrogen dominance.

But we can think about that with any of these toxins that come through glucuronidation; beta-glucuronidase is going to give us a big clue if it's elevated that those toxins are getting reabsorbed. So if this is in the gut it's got to be addressed, and it can be addressed with something called Calcium D-Glucarate, and so that's a supplement people can take, and it's also in supplement called Glucuronidation Assist, and that helps to reduce that beta-glucuronidase enzyme, so that's the way we can support this pathway from the intestinal support and also just cleaning up pathogenic bacteria especially Clostridium.

[00:50:30.10] Scott: So is something like Calcium D-Glucarate, which we're going to talk more about later in our conversation, but is that only working with glucuronidation impairments when there's elevated beta-glucuronidase? Or is it possible that even if you have a normal level of beta-glucuronidase, and impairments in glucuronidation that Calcium D-Glucarate could be supportive?

[00:50:53.17] Beth O.: It could be supportive in general also by donating this glucarate compound for the glucuronidation process. The only real place where we want to be more careful about it is when women have very low estrogen. And so if the estrogen is low, we just don't wanna take too much of it. So typically people can do about 500 milligrams of Calcium D-Glucarate, without getting into too low estrogen issues. If somebody has hot flashes taking it, and then they can just bump it down a little bit more.

[00:51:30.26] Scott: So, in terms of beta glucuronidation, what's the connection between that and cancer? And also, the connection between beta-glucuronidase and the cell danger response?

[00:51:42.07] Beth O.: So there's quite a lot of research out there on cancer issues, especially colon cancer risk, liver inflammation even, liver cirrhosis, and elevated beta-glucuronidase. And again that's because these toxins come into the colon, and then all of a sudden think of it as the protection of the glucuronidation protection, keeping those from

affecting the body is stripped away, then they're just sitting there attacking and affecting the colon, so that colon cancer risk goes up.

Cell danger response is a really crucial model in chronic illness, so this model talks about that anytime we have ongoing toxins, toxicity or ongoing chronic infections the body goes into this cell danger response. Where it's going to prioritize dealing with the toxins, dealing with the pathogens, and it'll start to shut down certain pathways.

And I like to think of the metaphor of if the body is a super complex assembly line if we want to think about chemistry like that, and there are all these assembly lines happening, packaging up different compounds and moving them around the body. And then all of a sudden we have a fire over in sector four of this factory, we're not going to keep the assembly lines running we're going to shut them down, and then go put out that fire. And so that's what's happening when we've got things like the heme pathway issues we talked about last time, the porphyria, Mast Cell Activation Syndrome is an aspect of the cell danger response, autoimmunity, certain kinds of cancers.

And so I tell people that the Mast Cell Activation Syndrome, the heme dysregulation isn't the problem actually, the problem is the root issue. And the elevated beta-glucuronidase is fading into the cell danger response also, often because we've got an imbalance in the intestines where we've got an overgrowth of either too many beneficial species or overgrowth of pathogenic species in the intestines. And then you have to think well why is that occurring? Well, it can be occurring because we have too many toxins; it can be occurring because we've got mold toxicity downregulating the immune system, so these things is just all tie together that's the real beauty of all of this.

[00:54:24.20] Scott: We have talked about some of the potential ways to explore glucuronidation impairments in terms of testing. Can you just summarize some of the key ways that you explore this in your clients?

[00:54:37.08] Beth O.: Sure. So checking the bilirubin, so that's a big one. And also, looking for the thyroid hormones and seeing do we have elevations in reverse T3, but even T3 or T4. Do we have elevations in estrogen? Or even just an estrogen dominance picture where there are more estrogen than progesterone. There's also a few panels would actually measure glucarates, and that's a glucuronidation compound so we can check that glucarate and then again the beta-glucuronidase is key to check that on stool testing.

[00:55:15.16] Scott: So the glucarate is in some urine organic acids tests correct, and then the beta-glucuronidase in many of the different stool tests that people can do?

[00:55:24.24] Beth O.: Right. So glucarate is on I believe the Genova ION has glucarate, also Doctor's Data has a Hepatic Detox profile that has glucarates, so those are glucarate options there.

[00:55:40.29] Scott: There is a long list of different gut bacteria that can create beta-glucuronidase, and so when we're looking at balancing the microbiome, reducing beta-glucuronidase. Are we generally thinking more in terms of antimicrobials, or are we thinking more in terms of something like spore-based probiotics, or possibly a combination of both?

[00:56:00.23] Beth O.: It depends on what the picture is that we're seeing. So I like to take a look at what the whole gut picture looks like. Looking at a stool test, and I like to look at the organic acids side by side, the gut markers. Seeing what we have this looks like somebody's dealing with SIBO, is this just some general dysbiosis because of mold toxicity?

Typically, if there's an overgrowth picture of Clostridia, or if we've got SIBO, we may have to use some antimicrobials, and then the spore-based probiotics can be extremely effective for balancing the gut. There are also human lactic strains that are really helpful, especially in Clostridium. And again, it's tailored to what that person is dealing with, but typically we're looking at a little bit of both.

[00:56:55.07] Scott: Talk to us about the connection between glucuronidation and the blood-brain barrier, and the potential for neurotoxicity.

[00:57:02.28] Beth O.: So there is a little bit of glucuronidation expression that happens in the brain. And even though bilirubin is an antioxidant, when bilirubin becomes too high, too elevated, then it can permeate that blood-brain barrier and start to accumulate in the brain.

Bilirubin is not supposed to be in the brain itself, and so then we can start to get neurotoxic effects. This is another reason why I really don't think that Gilbert's syndrome is benign, as traditional medicine says. And so if that bilirubin instead of getting conjugated by the glucuronidation, if it stays in the brain, we're going to get an increase in brain inflammation, and then all the downstream effects of that.

[00:57:56.03] Scott: I think one of the most interesting aspects of this glucuronidation discussion for me personally, and the community of people listening to this podcast is that it impacts our ability to clear mold toxins. And so many people today are really struggling with mold illness. I'm interested in your perspective, your clinical experience and the clients that you've worked with mold illness, and how significantly the shift was when you started to address their glucuronidation impairments.

[00:58:24.19] Beth O.: This is one of the reasons I'm so excited about this research. I'm really interested in the increase in mold toxicity and mold-related illnesses and the impacts it's having on Mast Cell Activation Syndrome and heme dysregulation in all these other areas. So when I started digging into this research, the first thing I saw was that there are certain mycotoxins cleared by glucuronidation, and I thought well that it seems important, let's go dig into it.

And as I was digging into it there were so many, so the Alternaria mycotoxins, many of the Fusarium mycotoxins like the different types of trichothecenes, Ochratoxin A and B, Sterigmatocystin, and Zearalenone - these are all broken down by glucuronidation, this is a huge chunk of different mycotoxins. So I was seeing people who were doing the typical mycotoxin protocol, but when they got to the antifungals, they were doing worse and worse and worse.

And it wasn't quite making sense why until I started studying this research and realized what's happening for these people that have these types of mycotoxins cleared by this pathway, is when the antifungals were brought on board the mold was dying off, more of these mycotoxins were being released and then there was nothing to catch them in the phase two detox, the glucuronidation wasn't being supported.

The other pathways where glutathione was being supported, methylation was being supported but not the glucuronidation because that piece has just fallen off the radar on so many places. We brought that on board then we started supporting with the glucuronidation supports. So we backed off the antifungals, did the glucuronidation supports, and then slowly brought that antifungals on board suddenly the person could deal with it, and they were handling more and more antifungals, and they were progressing much faster.

So again, we've got to support all aspects of the detox pathways if we're dealing with, whether we're talking about mycotoxins. I highly suspect that Lyme toxins may be going through this pathway, the research hasn't been done yet, but very likely the biotoxins from Lyme and co-infections, and a lot of these other chronic infections are probably dependent on this pathway in the detox phase.

[01:01:00.29] Scott: Yes. And to your earlier point, if we can just take some of the stress off of the pathway, right? So maybe we don't need to necessarily support all of these different toxins that are moving through it. But if we can, in my experience, mold toxicity is a huge player and chronic illness, and so if we can support glucuronidation in such a way that we're better able then to metabolize and excrete mycotoxins, that seems like a huge step forward.

[01:01:25.16] Beth O.: Absolutely. So if we lower the overall toxin stress, like the chemical toxins that come down through this pathway, pay attention to medications that people are taking. And if they've got these specific mycotoxins involved in this pathway, and they have to have them take their medication, probably better to choose a medication that's not dependent on the glucuronidation pathway.

We address the hormone imbalance, we address beta-glucuronidase, people are going to have a much easier time, and you're going to find here in the future we're going to have much more targeted detoxification protocols based on the toxin. So research project I'm involved in with a couple of other practitioners, we're mapping these mycotoxin pathways out, how are they broken down, and then how can we really tailor the support.

[01:02:18.16] Scott: Exciting times for sure.

[01:02:20.19] Beth O.: Very exciting.

[01:02:21.10] Scott: I've always felt that autism, Lyme disease, later MS, Alzheimer's, many different neurological conditions have more similarities than differences. We live in a soup of environmental toxins that creates a terrain that promotes chronic infections. You found that glucuronidation is reduced in children with autism, how significant do you think that is in terms of people presenting with autism spectrum disorder?

[01:02:47.21] Beth O.: I think it could be really relevant. So there was a study that showed an association between phthalates, so that's a very particular type of toxin and children with autism having reduced glucuronidation of that particular chemical. So they took that chemical because they knew it was dependent on glucuronidation, and the conclusion of that study was that there's a very strong link between autism and compromised glucuronidation.

Particularly because of all of the different toxins the pollutants, the food additives, different medications, the hormones that go for this pathway. And if we think about just our drinking water, we know that our general tap water is full of medications, everybody that drinks city tap water is drinking birth control pills and Paxil, and we get a lot of estrogens in the drinking water and our water is full of plastics now, they're finding plastic fibers in our water. And so these again have to go through glucuronidation, if that's impaired, we're going to have an entire plethora of implications, health implications.

[01:04:06.03] Scott: So, in terms of getting to some of the treatment approaches here, the first one you mentioned is kind of taking the load off the pathway. So reducing plastics, reducing the nitrosamine, benzene, the charred barbecued type foods, looking at the mycotoxin potential, addressing beta-glucuronidase, supporting COMT. Looking at the hormones, optimizing bile. Anything else in that realm before we talk about some of the substances that can help to support the pathway?

[01:04:41.13] Beth O.: Yes. Also, checking that COMT pathway because that's also involved in the metabolism of estrogens and those phenolic that we were talking about. And supporting that so we can support COMT in a number of ways like magnesium, low-dose lithium orotate is helpful.

Also, the brassica vegetables, so these are things like cabbage, cauliflower, broccoli, arugula as long as people don't have fog map issues, if they do have FODMAP issues even just arugula and cabbage are low FODMAP. But most people can tolerate those; those are big supports to the glucuronidation pathway.

[01:05:21.18] Scott: And arugula is bitter, so that's obviously bitter is better, and so we're supporting the bile piece from that perspective as I've learned from my good friend Ann Louise Gittlemen, so that's great as well. What are some of the substances then that we can use to support glucuronidation?

[01:05:40.07] Beth O.: So this is really tricky. When I was getting into the research, there is a ton of research on supplements, especially herbals and these glucuronidation enzymes. But the problem was that a lot of them will upregulate some of the glucuronidation enzymes, and downregulate others. So, for example, St. John's Wort has that kind of effect upregulation on some, downregulation of others. Or ginkgo upregulates some, downregulates others, great to see.

And so I wanted to come up with some supports that really support it across the board and through digging into it, what I came up with were the Calcium D-Glucarate that we already talked about, and then five herbals that will help

support glucuronidation without any downregulation. And these were dandelion root extract, pterostilbene, ellagic acid, rosemary, and astaxanthin. Those are all great glucuronidation supports.

[01:06:48.08] Scott: Fantastic. So let's then first talk about the Calcium D-Glucarate a little bit more. So we can find some of that in crucifers, it does exist in some foods, it can help lower cholesterol. Lower levels of D-glucaric acid can also be correlated with increased risk for hormone-dependent cancers. How is Calcium D-Glucarate working, and how much of this glucuronidation problem do we think it potentially supports?

[01:07:17.02] Beth O.: So what we know is that Calcium D-Glucarate will both reduce that beta-glucuronidase in the intestines if it's elevated, and at the same time increase the glucuronidation, so it's doing both. So it's working two different ways here, and there's a lot of research that supports that this can reduce cancer cell growth and inflammation and that it can eliminate certain carcinogens and tumor promoters.

So this is a really powerful compound; it can also support the metabolism of different medications that go down this pathway. So people that are taking NSAIDs or benzodiazepines or opioids even statins, this could be a good adjunct if they can't get off those medications. And then again like we talked about before, if this is being used in women with low estrogen levels. If we keep that Calcium D-Glucarate below 500 milligrams a day, then usually it won't be a problem for them either.

[01:08:25.23] Scott: For those people that are somewhat hesitant to take calcium, because they think that long term it can lead to other problems of aging in terms of blood vessels, atherosclerosis - things like that. Is there a downside to taking Calcium D-Glucarate in terms of getting calcium that we don't want?

[01:08:43.18] Beth O.: That's a great question. I tell people to take this with their meals, and that way that whatever oxalates are in the meals that calcium will bind to the oxalate instead of being absorbed. And calcium typically is more absorbed on an empty stomach, and then we can balance it with some magnesium also taken with the meal, and we can reduce those effects.

[01:09:08.03] Scott: Wow, the pearls just keep coming, good stuff I'm loving it. So you mentioned a few of the substances that you found helpful in this realm that only upregulate and don't also downregulate. I know that you've been working with the Functional Genomic Nutrition product, the Glucuronidation Assist. Tell us a little bit about that product; tell us how people can access it if they're interested. Do they need to work with their provider, let's hear a little more about that.

[01:09:35.01] Beth O.: Well, I'm really excited about this, there was quite a lot of work that went into formulating this product, and it does contain just the 500 milligrams with Calcium D-Glucarate. So it's going to be effective at that dose, that's in two capsules. So if somebody feels like that's a little too high, they could just take one capsule if they prefer they can get 250 milligrams.

But then it has all five of the compounds that we just talked about the herbal, so it has the dandelion root, it has the pterostilbene, the ellagic acid, the rosemary, and the astaxanthin. And we've been trialing this out for a couple of months now, and really seeing great results for people, especially people who are having trouble with her detox protocol, people that weren't tolerating antimicrobials and antifungals, and I'm noticing myself I had some elevated beta-glucuronidase levels. And even just with this, I'm noticing my gut starting to calm down some more, and some decreases in inflammation I think it's because it's supporting this pathway here.

[01:10:43.00] Scott: Beautiful. If people are interested in learning more about that product, where should they go?

[01:10:49.13] Beth O.: Right. So they can go to, if practitioners, can order at FunctionalGenomicNutrition.com or if it's a patient, you can reach out to us at MastCell360.com, and we'll be happy to help you get ahold of that. And you can take a look at the label there at FunctionalGenomicNutrition.com. Anybody can go and take a look at the label. And then if somebody's not a practitioner and would like to order it, they're welcome to reach out to us, and we'll help them.

[01:11:17.21] Scott: Excellent, that's exciting that we have some new tools coming. We've covered so much in this conversation, so can you maybe wrap things up for us by sharing what you expect to see in the chronic illness world as more information about glucuronidation starts to become better understood. And do you think that glucuronidation has any chance to become as popular as its sibling methylation?

[01:11:42.00] Beth O.: So I think where we're going, I mean we are in this world functional medicine where we're starting to personalize things, individualize things. But I don't even think we've scratched the surface yet; I think in five years we're going to have things so targeted down for the individual. And especially in terms of detoxification, right now I make custom detox map for each of my clients and go through the detox map and circle where we're seeing that it looks like they have challenges and how we're going to support, and how we're going to step from phase 3 to phase 2 to phase 1 to our antimicrobials.

I think we're going to have software going forward that's going to do this for us, and build these custom maps. So this is really exciting, we're on the cusp of a whole new world of how chronic illness is addressed, and I think we're going to get so much better results going from where we think about the gold standard right now is if you can reach 80% of people with your protocols or your approach, that's considered really excellent.

But then 20% of people fall through the cracks, and to me, that's not good enough. And we've got the tools to narrow that down, and with this kind of personalized approach like we're talking about today, I've been able to get that to 95%, which I'm really excited about. And the other 5% tend to be people who don't want to put the work in, because it is work, to work on these things for your health.

But I think glucuronidation is going to be a big piece of this as we move forward. And that's my goal, my dream for this research and getting this out there and talking with you Scott, is getting the knowledge out there and I hope that this will become as popular, if not more popular than methylation here in the next few years.

[01:13:43.25] Scott: Excellent, beautiful. So I know you just answered this question a few episodes ago, so maybe you have some newer thoughts around, what are some of the more recent things that you're doing on a daily basis in support of your own health?

[01:13:57.10] Beth O.: One of the newest things that I'm doing Scott is I'm doing quite a bit of Qi Gong, and just as energy movement in my body every day, it feels deeply healing, that's been really big for me, and then quite a bit of intermittent fasting. So to support the autophagy process but pulsing it, so what I'm doing now is they're doing it continually, I'm doing intermittent fasting five days a week, and then two days off to kind of support that cellular cleanup and then their cellular growth process. And I'm finding that seems to be working better for my body than the constant intermittent fasting.

[01:14:42.00] Scott: Beautiful. Thank you so much for your time today; this was a deep conversation. I think getting the awareness out there around glucuronidation will hopefully continue the conversation and move it forward. You've been very generous with your time in sharing your knowledge and wisdom, and experience. And people that are interested in working with you, how can they find out more about your client opportunities?

[01:15:07.04] Beth O.: My website is that Mastcell360.com or I'm on Facebook @MastCell360. And you can take a look I've got tons of free resources out there, I've got a free root causes report and chronic illness, and you can shoot us a message if you have any questions. I just want to thank you, Scott, really appreciate that we can team together and help people like this and get this information out there.

[01:15:36.21] Scott: It has been my honor and really fun conversation, so thank you so much for being here today, Beth, and I'll look forward to more of the things that you uncover that we can share together with people in the future.

[01:15:48.18] Beth O.: That sounds great.

[01:15:49.25] Scott: All right, be well.

[01:15:51.05] To learn more about today's guests, visit Mastcell360.com that's Mast Cell 3-6-0 dot com, Mastcell360.com.

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