Podcast Introduction and Hormone Testing Overview

If you're trying to decide what to wear for the day and you are looking through just a little hole in the wall and it's wet outside, so you think, oh, it must be raining.

That's akin to just looking at a serum or a salivary sample. You get a single little value at single point in time and your trying make a judgment call.

But if you were to look over the walls or open a gate door, you'd see it was actually just ton of sprinklers on full blast. So now your entire wardrobe has changed.

based on the amount of information you were able to gather. And with urine, you're opening the door. You're taking in a lot more landscape to determine, how do I want to dress for the day?

How do want treat my hormones? What could be driving these things? Welcome to the TBD Fit podcast on Dr. Talks. I'm your host, Daniel Keeley, and I will be guiding you through this wellness journey in terms of optimizing health and longevity, where we unpack the science, the dos, dos.

and everything in between. Come join us. Look forward to seeing you inside. Welcome back to the TBD Fit Podcast, where we bridge the gap between functional lab testing, root cause medicine, and real patient transformation.

I'm your host, Daniel Kiela, I am a functional medicine practitioner. And today we are diving into a very often misunderstood topic, how urinary hormone metabolites can reveal deeper metabolic dysfunction that drives so many of the symptoms patients struggle with.

Then we'll look at kind of all the breadth of testing. And in terms of symptoms, obviously there's fatigue issues, mood, cognitive issues. Weight gain, low libido.

Just to name a few, our guest today is someone who's redefining how we understand hormones on a very biochemical level, which I appreciate. Welcome Dr.

Liz Bartman, who is a naturopathic doctor specializing in nutrigenomics, environmental medicine, and hormone balance. As a chief medical officer at Endo Access, she leads innovation in data-driven hormone analysis in root cause supplement design with her deep background in advanced hormone testing and functional genetics.

She's helping clinicians and patients uncover the biochemical why behind complex symptoms and truly achieve sustainable precision based outcomes. Dr. Robin, it was a pleasure hearing you speak.

At the microbiome labs symposium, your presentation was very thorough, very informative, which is what I appreciate since I'm looking always at the literature, always hit the data.

It is a genuine honor to have you here. Thank you so much, Dr. Daniel, it's such a pleasure to be here and I'm excited to dive into hormones. This is a great topic.

It is, especially with the black box label that was recently uplifted. So let's start though with your story. I love for patients to, and just our community in general, to understand why our guests are doing what they're doing.

What's kind of your backstory? Yeah, so I began in cellular molecular biology and chemistry. So my background is really in research. I know I wanted to be in healthcare in some degree, but I just wasn't sure where.

As I was going through my education, the Human Genome Project had just kind of become public. And so that really caught my eye and my interest. At the time I in a lab, we were doing some research on the BRCA gene, which is very fascinating.

opened me up to, well, instead of this very algorithmic approach to healthcare, how can we look at the whole person and how we can really look the cellular dysfunction that can be driving disease?

And that's what led me into naturopathic medicine. And then through really my training with natropathic medical care, looking at the whole person, which I just fell in love with using herbs, nutraceuticals, lifestyle interventions, all my key things, really began to explore how can our genes, our environment, and really, even our gut transform our health.

Every time I would come into exploration of how to, explore what we would call the phenotypic expression of Our genetic predisposition. So we don't express all of our genes all the time.

We don' always have the high risk for everything that we have, you know, in our genetic maps. But we can see in real time, how are we expressing and where do we need to point more focus.

And over and over, I would say, or I will reveal that urinary hormone metabolism really is the way that can we see. It's that lens that is going on metabolically that could show us how our gene, our environment and our gut are all interacting.

And it's like, so profound and yet such a simple test in many ways. And yet, it is so amazing what it can reveal at a deep level if you know what you're looking for.

So that's how I got here. Long story short. Well, initially, how are people finding you often? Are you starting before perimenopause, menopausal, kids?

How are you and when are starting this discussion around hormones, obviously environmental toxins and looking at obviously inner the hood. I treat. patients from pre puberty to post menopause.

So I really treat the spectrum. Often I am seeing peri and postmenopausal women and then their families. It's through that care that their desire to better their health that then they realize, oh, our genes never change.

Let's do this with my kiddos to see what they're up against. And if I had had better You know, just recommendations or advice early on.

Dr. Liz Bartmanu2019s Background in Functional Medicine

Maybe I wouldn't have these same conditions now that I'm dealing with. Let's start early. I think that is awesome and can really help guide and direct care.

But, you know it's never too late as well. We are seeing in epigenetic studies that you can change your epi genome and your health outcomes for the better, even into your 70s, 80s and beyond.

So it is never to late, but I do tend to see the gamut. through the perimenopausal women that are often coming because they're just not feeling great in their bodies during this significant hormonal change.

And I love that you said it's never too early, right? So when you're looking at toxicity, which is so ubiquitous, are you running a tox test on children?

Certainly on parents, but when does that start? Not necessarily. A lot of it comes through just environmental screening questionnaires, looking at their risk factors.

But remember, our toxic burden doesn't start when we enter into this world. It starts when are in utero. And even

epigenetic programming can begin when were are an egg and our mother's ovaries and her second trimester and development and your grandmother's.

So it really is multi-generational exposures and knowing that, going backwards, being able to unravel that through history, through risk factor assessment, and even a little through the genes, we can really guide optimal support early on without having to do a whole lot of invasive testing or testing in general, right off the bat.

Okay. So you're having a discussion obviously then with the individual, no matter what the age would, especially in terms of preparation from childbearing and then obviously things how it progresses and into the period of all-spouse volunteers.

Do you see trends over time in term of what's happening in, terms? I do see a lot of trends in various communities. That's what I see more of. It's like, I will see like on the East Coast, and I do suspect part of it is just like the exposure to coal and maybe fires and certain pollutants, but often an upregulated CYP3A4 clearance with estrogen metabolism.

And this can be an issue with, um, Cyp1B1 is the other one that I often see up regulated heavily on East coast. lab testing, which is curious, at Chimmy, when tracking trends, the CYP3A4, this can be a couple of factors.

It can upregulation due to certain environmental toxins. it can also be recirculation. That's where our estrogens hit because Cyp3a4 is the primary enzyme expressed in the liver.

Some of our other SIP enzymes, our CYP enzymes that phase one transformation of hormones occur in lots of other tissues. It's not just the liver, but C-Y-P-3-A-4 heavily in the livers.

When you have recycling of estrogens because of poor like astroblome activity or rather more active astrobolome, like very high beta-glucuronidase, you get this recirculation hitting enteropatic circulation, hitting the going into 16. And for whatever reason, I see this kind of push.

So I'm like, is it environmental or is that maybe biome? Is it the gut? Are certain foods augmenting kind the beta-glucuronidase response and that recirculation?

So it could be either of those. Then with the CYP1B1, when I that upregulated, either exposure to lots of fires because its primary role is to break down polyaromatic hydrocarbons.

So it's breaking down these things that we inhale, these toxins we inhaled, but it also happens to move estrogen into what we would call the ugly metabolite, the 4-hydroxy intermediate.

4 hydroxy on its own, not terrible if you're methylated well, or if your reducing it via glutathione. But it can be a problem if it oxidizes into a quinone.

It's going to damage our DNA and becomes a problematic child. So we want to keep a close eye on 4-hydroxy, compare it to methylation, and compare to oxidative stress markers like pyroglutamate and 8-OH-DG.

But also know that if its elevated, there's higher polyaromatic hydrocarbon exposure. And that can pollution. That can, again, be the coal mines and just smog in the air.

fires. And I do wonder with some of our fires on the West Coast as well. I'm over in Oregon, but we do get a fair number of forest fires, California has been on fire quite a bit the last few years.

We see this for hydroxy go up transiently as in those demographics. It's more about where you live, not necessarily global trends all over. So when you're saying kind of up regulated or faster or conversely down regulated, what are some in those circumstances?

How are you thinking about in terms of solution or nutraceutical intervention? So it's always number one, remove or

reduce. Why? if it's a bad pathway, right?

So for like, let's say sip 1B1. If we are concerned that polyaromatic hydrocarbon exposure is pushing for hydroxy clearance, we don't want to inhibit 1b1 because that's doing its job.

It's breaking down poly aromatic hydrocarbons. In fact, there are some polymorphisms that reduce expression of CYP1B that I always like to promote because in our GWAS studies, which are the genomic wide association studies They can see that in some of these promoter region variants, you get a down regulation or a, down, You don't express it as much, right?

You're not hitting on that gene to express that CYP1B1 enzyme. Therefore, your not pushing as estrogen into the 4-hydroxy pathway, which would be seen as like a blessing, because 4 hydroxy, when it oxidizes, becomes a quinone, that can increase cancer risk.

And in fact, they do see a slight decline in cancer risk with that polymorphism. That said, what they see is an increase in lung cancer. And it's because they're not breaking down the polyaromatic hydrocarbons.

So if we learn a lesson from our genes, we don't want to suppress it. But we do want eliminate things that would upregulate that pathway.

Environmental Exposures and Hormone Metabolism Patterns

If poly aromatic hydro carbons open the door on that enzyme and allow the toxins through, but also estrogens through into this dangerous path, Let's clear out the estrogen in a safe way.

And we can do so with methylation, right? So it's producing a catecholestrogen. So catacol-O-methyltransferase can help. We call it the garbage bag. Put it into a little garbage big and take it to the dump, all right.

Glutathione also helps reduce for hydroxy, as well as the quinones. If any oxidative quinone are forming, if we have optimal glutathion, that'll be beneficial.

That'll work in our favor. NQ01-NADPH-quino oxoriductase is an enzyme. that gets activated in the cell wall around the nucleus of every cell. When quinones enter into the nucleus and are about to damage DNA, we turn up ubiquinone conversion into ubiquinal CoQ10.

So it's an active free radical scavenger, protects our mitochondria. Very good as a great antioxidant, right? Good for cell activity. We have up regulation of turn-up, rather turning conversion on from vitamin E into active chocotrienols and then turning the expression up on P53, which is basically saying if the cell is damaged too much, it is a tumor checkpoint.

We turn off the cell. We go into programmed cell death and start anew. I'm going to try this over again. Healthier DNA this time. If all these things are working, we can support NQ01.

Neutroseutically, Nq01 is turned on by NERF2. Nerv2 is this great antioxidant promoter within every cell in our bodies. It tends to be tightly regulated by a protein called the KEEP protein.

but we can uncouple it and allow it to enter into the nucleus to turn up expression A on the NQ01 protect and give us antioxidant defense. B, it turns up the expression on glutathione, catalase, and SOD conversion.

So it can be a potent way to protect our cells. Remove the offender, if we can. Use air filters. If it's smoke, make sure that you're getting good, just like one protection, one antioxidants.

Vitamin C is a great one there. Resveratrol could be a good one too. Sometimes get some work, like on the mucous membranes. Give some ubiquinol. Get some CoQ10.

You know, give, nerve two promoters. Nerve two, promoter's the big one, sulforaphane, which comes from broccoli sprouts. There's lots of ways that we can protect ourselves.

We can promote glutathione activity. N-acetylcysteine is a precursor to glutothione, as is glycine. But you can make sure methylation is working well.

You can give cofactors for COMT. Magnesium is the big one, but we need some methyl-B vitamins. Not a lot. we don't want to supercharge the cells, so we could give a little for just balance.

And we also need taurine, we needed choline. These are key amino acids for both detox, methyilation, and free radical protection. So the list goes on and on, but those are like some big ones.

Well, when you're seeing that representation, obviously there's the, the nutraceuticals for ideally everything coming from food, better still than plugging the gaps, with some of these nutruseuticals, certainly all of which that we're suggesting here as well.

I love that when we transitioned into NRF2, until first thing that has become almost as stable for probably close to a decade. One thing I, and again, it's not necessarily ubiquitous because the context is relevant, When you said from broccoli, broccoli sprouts, it's what we try to start growing sprouts here to accommodate for that.

Um, and so it definitely a labor of love, but nutrition, again, the power of getting the proper nutrients through food and then correlating it with what you see in terms of the testing.

Now to, to wrap it all down further into the test thing, let's compare, I guess, three different tests often, which are urinary, salivary and blood. And when you use which tests and maybe the pros and cons of each.

Absolutely. So we'll start with blood, which is our serum panels. It's your gold standard. If you're looking for a diagnosis, you'll use blood because that's what we've validated against.

Those are the reference ranges strongly associated with health or disease. The issue with serum is that. A, if you're going to get a diagnosis, you have to test correctly.

And that sometimes is overlooked. Timing, If you are a cycling female, timing of your cycle matters, as well as time of the day, because hormones are very pulsatile.

They can fluctuate quite a bit. We don't just make a hormone and then let it stay steady, stay constant throughout the. Just like cortisol, we think about cortisol.

The rise in the morning to wake us up and the steady decline throughout. All of our steroid hormones, all of which are our sex hormones have a similar pattern where they go up in the morning and they tend to decline throughout the day.

Estrogen is the most steady state, but again, you really do get those peak levels to no function in morning. So timing matters. If you're looking at specific promoters like LH, FSH, the signals from brain to ovary serum soup, it's the primary way you are going to see those.

Again, timing matter. But it is a single point in time. And as I mentioned, steroids, steroid means it's a cholesterol-based molecule. Our sex hormones and our stress hormones are all cholesterol based.

So cortisol, pregnenolone, progesterone estrogen, DHEA, testosterone, DHT, right? Even vitamin D. These are cholesterol base molecules. This means they're fat.

What is our blood? Our blood is mostly protein and water. If you were to take a glass of water and pour olive oil into it, what happens? It separates.

And that's kind of what happens in blood, right? Our free hormone is not going to show up very easily or readily in the blood because it doesn't mix well with the water.

What happens is if we're going circulate our hormones in our blood we have to circulated them with binding proteins like little taxi cabs. They just get into their cab and they get taken to the next cell that may need them.

The primary binding protein are sex hormone binding globulin and that is mainly for testosterone and estradiol and DHT. We sulfate our estrone and our DHEA to allow for circulation and cortisol and progesterone can bind to cortisol binding globulin.

All of them can also weakly bind albumin, so when you're looking at blood, you are looking a protein band. That is not active, that does not mean it can get out of the taxi cab, it just means it's in a cab and it is being circulated.

It's being driven around, right? And it was in the cab at the time of blood draw. So it's not your daily average. It is simply a single point in time.

So those are the limitations on serum. Again, the pro is that it is your gold standard. It is what we base our reference ranges on for diagnosis of various medical conditions, but you're only seeing primarily protein bound.

You can get free fractions. They're not that reliable, according to the research. In fact, calculations for free testosterone often are more accurate than trying to capture free-testosterone in blood because it's just so hard.

it It's like a precipitate you are trying extract. And so that also brings me to point with serum that not all serum tests are created equal. You can have serum tests that are LCMS.

You could have a serum analysis that is immunoassay. Equilibrium dialysis comes up. That's going to be primarily used for that free testosterone if you're trying to capture that precipitate that little bit.

And then you do see gas chromatography used as well. But that's in more volatile for more vital compounds. When you're trying to like be really precise and capture absolute values, especially very small hormones like estradiol, we don't need very much of it.

It's just very potent. You really want accuracy. So LC-MS is the way to go. And not all labs will default to LCMS. you have to be careful what tests you run and again, what time of day you ran it and if you are a cycling female, What time in the cycle you've run it?

So there's a lot of variables. With saliva testing, salivary testing has the benefit of being easy to do. You just have to spit into a tube or chew on like a swab, depending on the level.

It is showing you free fraction of hormone because it's what's inside the saliva glands. And remember, if a hormone is free, it is a steroid. All of our cells are a fatty bilayer, so they can allow free hormone to passively diffuse without any transport.

As long as it's out of the taxi cab, it can enter into the home. Saliva is showing us what's in the Home. It doesn't show us if it is active, or how it has been broken down, but it shows us it was there.

Again, in some cases, especially in the case of estradiol, it's a very small fraction of hormone. So timing matters and how you're testing and capturing that value matters.

More and more labs are going to LCMS for salivary testing. It hasn't become a universal though, so I'm still default to immunoassay, which just is not as accurate.

That said, salibary cortisol is the way to go if you are looking for diurnal pulsations with free cortisol. You just have to know salivary glands are very active with 11-beta-HSC enzyme activity.

So you do want to look at both free cortisone and free cortisol to how much cortisol pass through the tissue at a given point in time. And I would argue, just like you'd do with salive cortisol, you should test four to five points throughout the day for all of our sex hormones if you're doing salivary testing because, again, they are so pulsatile.

In fact, just to throw this out there, progesterone was tested every 15 minutes in women. And this was back in the 1980s. But it was a landmark study on this hormone.

They found pro gesterones can vary 80% in 90 minutes, which means if were to test a woman at 8 in morning and again at 9.

How Urine, Saliva, and Blood Testing Compare

30, she could have radically different pro-gesteron levels. So if you're not getting multiple points for progesterone throughout the day, it's like, what are you even doing?

What do you know? Who knows how much they're actually making? From the study, they actually said women should be testing progesterone four times. And if your doing a blood test, that's a lot of hoax.

No one's gonna do that. Again, most of our hormones peak in the morning, so the consensus was, well, we won't torture women, let's just test in morning and that gonna be good enough.

But there can be a lotta fluctuation. And that brings me to urine. When you're looking at urine, you are gathering results at multiple points throughout the day.

So you picking up on the peaks and the troughs. You're getting the total daily average, not a single point in time. Whereas saliva and serum is a signal point and time at each time that you test, right?

So if you doing salivary testing multiple times per day, great. Now you have more data points to then average out. And that's great. That's good. But with urine, you're getting that no matter what, because you are testing four to five different times to get multiple values for that basically 24-hour shot, like urine value.

So the other value of urine. Now we'll get into the urinary testing. Urinary testing is revealing bioavailable hormones. So it was what could be accessed by the receptors.

And that's important because, again, serum, it's what's in the taxicab. But if it can't get out of the taxi cab, what value does that give us outside of, well, they can produce it or not?

It is a value, but now we want to see compare that to urine to say, oh, what actually got into the cell? And then how did this cell transform it? So how does it circulate?

How did it get access into tissue? Then how is it broken down? You can reverse engineer those metabolites to reveal a metabolic journey. You could only do this with urine because it's the only way you can see clearance.

It's an end product. And that really can be profound. I think of, I like to give the analogy of You know, if you're looking, If you are trying to decide what to wear for the day and you looking through just a little hole in the wall and it's wet outside, so you thinking, oh, it must be raining.

That's akin to just looking at a serum or a salivary sample. You get a single little value at single point in time and your trying make a judgment call.

But if your were to look over the walls or open a gate door, you'd see it was actually just ton of sprinklers on full blast and that in fact, its quite hot and sunny.

So now your entire wardrobe has changed based on the amount of information you were able to gather. And with urine, you're opening the door. You're seeing, your taking in a lot more landscape to determine how do I want to

dress for the day?

How do want treat my hormones? What could be driving these symptoms? That's that in nutshell. Yeah, that's perfect. So in terms of urinary testing, is that gents, given those three, Is there a specific order of operations or do you just default?

I mean, urinaries is kind of the way to go. The most complex. Urinary does have the limitation, a few limitations. Again, it's metabolism. And it's specific to kidney, your kidney clearance.

So kidney health is important. If you don't have good kidney help, you could get either under or overinflated values simply because your kidneys are over or under excreting creatinine.

And that's what we calibrate to. There's also a gene deletion called the UGT2B17. It's a copy number variant. You either get two good copies from mom and dad, or you get one copy and a missing copy, are you getting two missing copies.

40% of the population has at least one missing copy of this gene, and it is responsible for clearance of testosterone into urine. into the form we look at in urine, testosterone glucuronide.

So if you are a carrier or have two, the homozygous variant of the UGT-2B17, you may not process your testosterone into urine as accurately as you actually make an excess testosterone.

I always like to do serum testosterone total and SHBG levels, binding protein, CAB. How many taxis do we have in circulation? How much testosterone is bound to them?

And then compare that to the urine. but I do often do urinary testing first. And then I like to look at serum as well for those other peptide hormones, for the LH, FSH signals.

For men and women, L H is very important. So I'd like examine that value. Um, and I did like do serum for other markers aswell, including that ring function, cause that's going to augment our urinery clearance.

I liked to lick the blood sugars, fasting insulin, because that can be compared to our urine to reveal kind of what is happening metabolically. Although our yearning will often tell us What other lab tests we may want to consider as well.

So you could do, I like to do them both together. You could them urine first and then let it guide you to what other tests you want. Do you have, obviously as ranges have grown and individuals have.

But do you specific reference ranges that you try to adhere to? So we mentioned LH, FSH, testosterone in terms of females. Yeah, so for FSH, F SH is our basically a marker of antral follicular development.

So it's how responsive, how many follicles do we have active and responding and accessing for for egg development, right? For they all surround like so our granulosa cells are the primary responder to F S H.

And Fsh is going to come down knock on the door. I called them the little knocks from their brain to basically allow for that expansion and selection of an egg each month.

When follicles are declining, you're going to have a louder knock, right? The brain's like, hey, factory, what do we got? We're still not making estrogen.

Where's our primary egg? Why aren't we getting hormones going? The knock gets louder and louder, FSH gets higher and higher. And that can be a sign that there is ovarian resistance to that signal as a result of likely declining ovariant reserve.

So it's a good fertility marker and it is a great indicator of perimenopause and then suspected, are you getting into

those Late peri, possibly going into post-menopausal soon.

Yeah. So ideally we want FSH to be, I like to see it under five, like two to five range. And when you get above 10, that's a little more resistance, right?

You're getting into a like, there could be some stress. of menting that follicular activity. In the upper teens, now we're into perimenopause. Above 20, yeah, you're likely, if you were still cycling, your likely not going to cycle for much longer.

But that's showing a significant resistance and likely transition into postmenopausal years. And then in post menopas, FSH can get into the 80s, even into 100s.

We don't like it up there. The other thing, so F SH is actually a promoter of aromatase, and that's what it's doing in the ovary primarily. It's saying, hey, let's ramp up estrogen activity through this enzyme pathway that is taking our cholesterol precursors and moving them into estradiol.

Aromatases is also active in adipose tissue. So as FSH gets higher and higher, it can't knock on the ovarian doors anymore, its going to start to knock adepose, that can increase aromatization activity in that visceral fatty area.

This is where women that are going into menopause tend to see their bodies shift and morph because we're trying to set the groundwork for our next best source of estrogen, unfortunately.

With this in mind, we like to keep FSH down and when you bring in bioidentical hormone replacement therapy for estrogen that is actually a marker you can track because you see response by looking at that FSh declining.

If you had a woman in her postmenopausal years and her F SH was in the 80s and you get her on bio identical estradiol therapy, You should see her start to drop if she's utilizing that hormone accurately.

It's not going to get back into the teens. We don't want it that low. But you may start seeing it in the 20s to 30s, and that's great. That's where we kind of want.

And that 20 to thirty range for postmenopause. For fertility, I like to see it kind in that five to seven range. Again, ten is kind pushing it. Perfunctional.

LH should be a roughly one to one. LH can start to elevate, especially to FSH ratios with conditions like PCOS. It's not a guarantee that that's the cause, but it can, we can to see a, you know, often they'll say like a three to one ratio L-H to FSH, can be an indicator for PCO-S.

That's another marker in cycling women that you can bet another ratio. Okay. So that was FS-LH. Other markers, anti-malarian hormones, another one I like to watch.

I'm sorry, in serum that, that wouldn't get it in urine. Anti-milarian hormone is a marker, and other marker of like, uh, follicular activity. And we really do like to see that in like the two to three range.

When you're starting to push into the four, five, six range, that's lots of activity without maturation. Again, they're trying to look at that as a marker for potential PCOS.

It's the lower though that can be like a low ovarian reserve or poor, like higher oxidative stress damaging those eggs. So under one, we're looking at low of ovariant reserve.

Under 0.1, We're Looking at premature o varian failure. if a woman is under the age of 40, not cycling, and has an AMH under 0.1. So a good one to, again, for fertility marker, great one, to check.

And also for that PCOS concern, it is the most common reproductive endocrine condition for women. T and sex hormone binding labulin? Yeah. For women, cycling women?

Testosterone? Generally, we like it between 20 to 45. I would say it's age dependent. In our twenties, we are going to be often maxing out our androgen synthesis, just as men do.

And then you see a steady decline of one to 5% each year after age 30, depending on stress levels and just your overall cortisol activity, cholesterol synthesis and precursor balance and oxidative stress.

But on average, you're going see just a one-to-two percent decline over time. That's true for testosterone and for DHEA. For testosterone in serum, total testosterone, 20 to 30 is a great range for like a postmenopausal woman.

I like to see 30 to 45 for a cycling female, although PCOS can be diagnosed with a testosterone total of about 40 if symptoms are also consistent. So irregular cycles and androgen excess symptoms, especially if coupled, but not critical for diagnosis, that have coupled with like cystic ovaries.

But SHBG levels, we really like see for women. 50 to 85 is kind of my ideal functional range. You will see the blood tests come back saying 150 is normal.

It's like, oh yeah, it's pretty high. 85, is that good? Like 50-85, you're guaranteeing a pretty good ratio of free to total testosterone. For men, for total testosterone, again, same thing that you're gonna max out really in your mid-20s with a steady decline each year after you age 30. So it depends on if you wanna get back to your age 20, which I would say for most men that's a good target.

It's like, get you into your 20s. 600 to 900 is kind of the range for men is my ideal range. Now I will go to 500 if SHBG is lower. Some men don't need as much if they're getting a lot of free access to it.

So SHPG levels, 30 to 55 for man, you want to keep it pretty low. You want that testosterone to remain free. That is your primary dominant hormone. And I really I like to aim for that 600 total.

I'm in an SH BG. I mean, if it's less than 30, there's a lot more free activity. I might aim, again, lower on testosterone total because you're getting more access to it.

For free testosterone, I'm in 150 to 224 is a good goal, which would usually correlate with a total of 600 to 900 and an SHPG that's lower. On Quest, because on LabCorp, it means slightly lower, What was that?

Key Hormone Reference Ranges and Metabolic Markers

So on Quest, right? Those are the targets, I believe, for LabCorp. It's slightly different, but our numbers seem to be in line. Are any other markers that were referenced ranges in terms of markers, that you're keeping a close eye on?

I like to monitor fascinating insulin levels because that's such a metabolic driver for so many kind of aberrant enzymatic pathways that can throw off how hormones both behave and then also are processed out of the body.

So like higher insulin levels can drive up SRD5A1 and 2, which are your 5-alpha-reductase enzymes, the cheap ones. And 5 alpha-reductase is taking testosterone into DHT, Which is more anabolic.

It's four times more potent, roughly, than testosterone, but it does chiefly act and chief produce within the prostate. We don't want it to get too high because we want to protect the prostates, But we don' want too low or shift it away because you need that performance from DHT as well.

So it's like this Goldilocks hormone, but insulin, if fasting insulin is elevated, you're going to be pushing potentially more into that pro-inflammatory path.

And for fasting, insulin levels, it kind of runs the gamut in the functional world. I feel like everyone has their favorite range. See it as low as like two being optimal.

That might be a little too low for some people, I like to see it no higher than eight. When we get into the eight to 10

range, that's pushing into insulin resistance and above 10, even the labs will go up to 20 as normal.

By 20, you're already insulin resistant. Um, but like under eight is great. Two to eight, is a great range. Yeah. That's what I say. Optical for me is two to six is below six, But then great is anything up eight.

Uh, and then we've got kind of some question marks happening here. Okay. So fasting insulin, I know you mentioned thyroid as well. up and thyroid 0.5 to 2.50 is kind of my range, although in the functional world, often people will want it under two, like 0 to two is good.

I think there's a little wiggle room. You don't want to like. over-treat if people aren't symptomatic in that 2 to 2.5, that's like your gray zone. But TSH, just like FSH it's that knock on the door.

So the higher it gets, the harder it is working to try to activate thyroid. Higher levels of TSh, hypothyroid. I always have patients that are like, oh my Tsh is high, I'm hyperthyroid, no, reverse.

If you're below 0.5, yes, you are now hyper because your brain is suppressing the signal because plenty of thyroid around saying no, no feedback, we don't need it anymore.

Free T4 and free T3 you want to monitor as well. free t3 is really the bigger one because that's your active part of the thyroid. And so I really like, you know, honestly, B 3.8 is a great free T3 value.

You want to compare it though to RT3, reverse T 3, because even if you have optimal free t3 if your also then pushing a lot into RT 3 reverse t 3 you may not be getting that activity still.

And that could be an early sign of underlying metabolic dysfunction. RT3 does push up in situations that are protective as well. So I always like to point that out with athletes in particular, if you are actively training for a marathon or for high intensity, high performance.

Event testing, thyroid, don't do it because you are going to be pushing into RP3 to conserve energy and that's okay. That's your body's response. But post in recovery, check it out and see where you're to help with that recovery process.

Yeah. What about if TSH is extremely low? What are your thoughts when you see something like that? Like non-reportable or like the 0.001? Exactly. That's hyperthyroid.

Yep, so often graves. And you do want to look at your thyroid antibodies as well. There's a number of reasons why your thyroids will be suppressed. Over-medication can be one of them, so always making sure your medications are balanced for thyroid.

And that post-pregnancy Graves is a common cause for a just wiped out thyroid response, like a Grave's thyroid, a suppressed response. The issue with Graives, or the issue of hyperthyroid in general, the heart is very sensitive to thyroid and so when you're seeing that suppression, you really want to make sure we're figuring out why.

Usually it's going to be autoimmune. And we're figuring out, do we need medication intervention to kind of bring it back up? Um, or do you need, because you don't need more, right?

You need to like suppress xiring activity to bring TSH back out. Right. Um. And do, we needed to protect the heart. So really looking at heart rhythm, blood pressure, pulse, but it's, that's a complex topic.

Another rabbit hole for one too. Okay. so going back to the urinary testing and let's say enter in endoactive. Let's just tell us kind of what you guys are doing and how you're changing the landscape in terms of testing and then obviously the intervention.

So, as you probably as your listening through you're like wow there's a lot that can go on with urinary clearance that's, a, lot of information and it is a.

Lot of, information, and not every physician wants to remember all of this not, every you know provider within the functional space has time to. sort out every different nuance that, can augment urinal clearance and so often how they're taught.

And, you know, when I was taught just the basics, foundation, it's here are the hormones. Here's what we expect. You're the metabolites. It's not about the ratios.

Not about that reverse engineering of that metabolic story. But that's really where the interventions come in, right? That's where the information that's driving symptoms can be found.

And so I feel like, you know, often people are just looking at the tip of the iceberg when it comes to hormones, even through urine. Urine is giving a little more insight, but it's still just the tips.

There's so much more going on under the waters. With endoaxis, what we're doing is we are taking, we scrubbing all of those urinary metabolites and we comparing them not just to their parent hormones but to each other.

And we're associating it with all the other kind of drivers, either the inhibitors or the drivers for these different pathways and why an outcome might present the way it does.

And, we are giving that to providers in a report that details that metabolic journey for them so that they don't have to know, what are all of the promoters for aromatase?

What do I have know here? We give you a record that says, it looks like this patient is over aromatizing. Here are the things to consider, here are further labs.

Here is nutraceutical intervention if you want to pursue that. We have supplements that we have curated for these different patterns and they are kind of like interchanged together to give different combinations unique to that patient's needs.

So I like to say we're treating The why, we're treating why is the hormone, why's the symptom present? Why is a hormone behaving the way it is? Not the what, what is actual hormone itself?

What is this symptom? But why? Is that a problem? How does it work if you mentioned this is for practitioners? So we draw, well, provide a kit for the patient.

They do it in the convenient of their home. they send it, then we get the results back. And then everything is kind of indicated in terms of here's, the actionable steps to improve hormone management.

Right. So once you've performed to the lab, so you would purchase the kit through a company that is not us. And so through Precision Analytical, the Lab that does the Dutch test, you get your kit, You send it home with your patient.

They do their tests. they send that kit back into the laboratory. You'll get the report from the Precision Analytica lab. That is a bunch of dials and a lot of analytes and it can be very confusing.

you upload it into Endoaxis. It is free platform for providers and you'll Get the Report back that tells you, yeah, here are your action items. Here's where you need to look.

And then in terms of the, the BHRT onboard, presumably it doesn't provide any sort of actual insight there, right? It's just a nutraceutical intervention or am I mistaken?

No, we actually do discuss whether they're in a good range or not based on their urinary clearance while on hormones. But we do talk about the nuances.

So if you're looking at topical estrogen as an example, you can stay on that for urinery testing. That's great. We

actually want you to because that's going to reveal well clear tissue utilization and clearance of that hormone.

Same with patches, so the estradiol patch. The caveat is vaginal estrogen because you could actually get some accidentally onto the paper because And there can be that cross contamination that can overinflate values.

So you just have to be careful when you apply and when your test. Test first, then apply. It's kind of the take home. Oral hormones are going to go through first pass metabolism.

That means it's just that you dump about 80% just out through that conjugation out into urine without ever using it in your body, but it is going look really high in urine.

You actually want to discontinue oral. depending on the hormone and the kit will actually tell you for anywhere between 48 to 72 hours. Progesterone is the caveat.

You can stay on progesteron because they have, again, it has such a short half-life, they've been able to calibrate kind of an expected oral range, but they can't really do that with estrogens and testosterone because of the dynamic clearance that happens in the body.

Injected injections and pellets collect halfway between. your either reinsertion or your injection date. That's kind of the, so you can test and then our report does give you ranges based on testing.

So yeah, everything looks within a therapeutic ideal. You have some wiggle room to go up or down depending on patient response. Or as I see more often, the people will be on, will say that they're on estrogen therapy and testosterone therapy, and their urine is like flatlined.

They're like, okay, you're not actually utilizing that hormone. If you were using it on the day of testing, that's pretty low. And we'll talk about that as well.

Do you see that often prevalent where a female patient postmenopausal on hormones but testing low? Is that atypical? Yeah, I do see. Sometimes. Yeah. I wouldn't say it's like very, very common, but it is more common than I would expect for women on hormone, to be honest.

And I think a part of that is that for a very long time, we were kind of afraid of estrogen replacement therapy. and I mean, some women are underdosed and so it.

The, the old mantra of start low, titrate slow, although true. I think some providers are just very conservative and they start a little too low and then you titrated a bit too slow.

And so, and you're seeing that in urine where it's like, yeah, they're on a lit all, but I don't think they are getting enough. You may want to bump them up a.

In conversely, what if you see high or. Notably high, yet patient indicates they feel great. Look at the metabolites. How are they processing it out? If they're pushing heavily into like that four hydroxy or 16 hydroxyl pathway, these are more proliferative with greater risk for oxidative burden.

And again, that DNA damage. I would just be cautious with their ratios and make sure that, you know, if we don't want to augment the dose, they feel great.

Maybe we support detox and we supply oxidator burden and maybe tweak the dosage just a little. Are they methylated well? Do we need to support methylation?

That might also be something that we want to target. And then of course, are they having bowel movements every day? Because if you're not pooping, you are not clearing out your estrogens.

If you on hormone replacement therapy, if for women of any age, cycling or on HRT, You need have regular bowel

movement for optimal hormone. regulation and clearance.

So definitely wanting to work on the gut there too. But really, you know, we have our bone protective ranges for estradiol. We know what we want to get above really around 60 picograms per milliliter in serum for our estrdiol, and that's using ultra sensitive testing.

There are functional docs that say, you know, I really want to get it into the 120 range. I think that's a great goal. There's some that'd say I want it to the 220 range, and that is closer to like opulatory surges of estrogen.

Yeah, everyone has to kind of, this is a new territory for estrogen therapy these days because we are realizing, hey, it's actually not a danger like we thought it was.

But we really don't know where that optimal dose is yet. And so we're just, we see, but we know bone protection starts at 60. So that's what I try to achieve in all my patients.

That's why we say in the report too, it's like, hey, this is again where serum can come in, check an estradiol in morning. They need to be above 60 to make sure that they're getting enough estrogen to protect their bones.

on their therapeutic response and how they detox. In urine, the goal is to get them kind of at the low end of what I call the gas gauge. On a Dutch test, there's like these little dials that you'll see with these stars on either side.

I'd call that kind the empty tank and like full tank of gas. It is based on luteal ranges. So a woman on BHRT, if you are getting them into low and lutial range, that's around a 60. picogram per milliliter in zero, kind of in that bridge between the post-menopausal, the gap, and then the low-end storm.

So it's a good target to be above post menopause range on HRT. Otherwise, what's the point? When you fuel the gas tank, in terms of starting, so if someone comes to you and they're just low across the board, are you starting someone on Just right away, EHEA and T and estradiol or anything else, progesterone.

Are you doing things in a stair-step fashion or how and when do you think about introducing specific replacement options?

EndoAxis Platform and Interpreting Urinary Metabolites

I like to test first, right? I always test on guests. Sometimes patients don't need DHEA right off the bat. sometimes their adrenals are doing great. There are more recent studies showing that possibly the weight gain that's happening in perian postmenopausal years is not driven by the estrogens per se, but by cortisol.

So I do like the test the adenals as well, which is why the Dutch is great because the dutch plus and complete both look at the hormones, their metabolites and cortisol response.

So sometimes it is that balance. Are they in a catabolic, high cortisol drive, kind of pro-inflammatory, androgen clearance, low DHEA, at the expense of D HEA?

Then maybe I do want to bring in D Hea to balance them out. Or are they fine on DHea? They're pretty balanced. Adrenaline, it's more just the estrogens that are on a fluctuating scale and progesterone that's on the decline because we're not ovulating regularly.

Then I might just start with an intervention, initially a pro gesterome. Sometimes I'll do progesterone in a low dose estrogen. We are finding that the earlier you bring in estrogen, kind of the better response women get into their late perian and early postmenopausal years.

So I think there used to be this concern that we don't want to start estrogen too soon because there's so much fluctuation happening in perimenopause.

And I there is some truth to that, especially in the early window, but If they're starting to get a lot of hot flashes, night sweats, the weight changes, The brain fog, yeah, and the mood changes.

You might want to bring in a lower dose just to give them a little stability. So even when they are high, they still getting a gentle, like it's there.

But when the drop, you still have this like buffer. Progesterone is usually my first, because that's the hormone that declines first for women. So I usually will bring in progesterole and see how they tolerate it.

I then discuss the bringing in of estradiol and then testosterone. And sometimes again, that sometimes I'll bring testosterone first. If estrdiol is looking great and testosterone is the one declining.

Again, it's all very personal to the patient. Testosterone is one of those in women, not to go on another tangent, but it not well researched, right? We get a lot of parallels from men, But testosterone in woman is a very different hormone.

I think it is very important hormone and it an unsung hero that does need and deserve further attention and further utilization. But it another one those I thing is like underutilized for women.

So one that I do consider especially if they're low, if their looking robust, Adrenals are great. I'll just start on the progesterone front. Does that make sense to answer your question?

110%, everything, this is the practice of precision-based or personalized medicine, right? So everything is contextual naturally. And the T interestingly is, is I think, um, certainly in terms of libido as well, very, profound.

We have our post-menopausal patients who, I mean, it's changed their entire willingness or sexual drive activity and something even as simple sometimes enough with just liposomal DHEA by bringing that online.

That too will very much enhance a drive or sexually drive. But no, yes, it's contextual in terms of where to start. I just see patients all the time just starting kind of everything.

And then we have all these patients that come to us where everything is kind just either zone doesn't exist or too high and mismanaged. Then you have very severe fluctuations they're in.

But often enough, my problem is going back to the beginning of the discourse and you know, some of these hydrocarbons you had mentioned or environmental toxins is we don't do enough of a cleanup beforehand such that when the cars are on the road, they can deliver to where they need to go, right?

And then the receptor signal is available to receive that. And so our foundational is critical to do some sort of a receptor detox or a hormone balance to get rid of some of these built up or excess estrogens or any sort beta-glucuronidase, any sorts of conjugation issues.

And then also to make sure that as you said, it is looking at the gut. So that's been my focus for the last decade is if someone's motility is compromised, if they're not passing, you're just recirculating toxicity, administer hormones.

You're going to someone worse. So we don't take that lightly, but I appreciate kind of how you express and how look at things. I just wanted to confirm our ranges are almost identical.

A few of them were slightly by like five off. Well, depending on I'm looking at. So when you said, you know, luteal or postmenopausal, estradiol, certainly six in terms of bone protection, we do have targets.

And what you say, interestingly, is this is novel in some respects. What works for one, sometimes not for all, but certainly there are certain levels that I think we do find hold true across the board that we want to hit in terms of targets.

And then that's also qualitative in term of how someone feels. So. Absolutely. It is so, as you mentioned, so personalized and variable. Inflammation is a big driver.

Environment is the big drive. Blood sugar imbalance is big. If you're not, I always say. So if we think about like the therapeutic hierarchy, right, it's all about foundational work.

Clean up your environment, clean up you gut, and clean your diet. If that's not strong, any intervention you stack on top of that pyramid will only be as good as that foundation is strong.

So, if you don't have, you're not sleeping well, Hormones may help, but we've got to get that sleep cycle regulated, right? We really need to support with stress management.

And if your gut's not working optimally, we focus on gut. Diet's so important. Macronutrients change for women as we get older. We need really to focus more on protein.

I think we're seeing that recommended more now, it's critical that you hit those protein goals in our perimenopausal years for hormone success. I always tell my patients like, you know, we can give you thousands of milligrams of ashwagandha, but we're not going to be able to reduce your cortisol if you're in a high stress job or in marriage that you don't feel supported in, or, in you, know in situation that just is driving up your stress on a regular basis.

Sometimes perimenopause is a great awakening of what do I need to really take into account and really put into order to optimize my health long-term because it's almost like this mask gets lifted.

Hormones give us this nice buffer, but there's a lot of metabolic change that can happen that likely was always happening to some degree. But once you lose that estrogen and that testosterone, that progesterone buffer it becomes amplified.

Does that make sense? So like, you know, vascular changes and hot flashes and mood and like these are all fluctuations that were likely always there to some degree and blood sugars, thyroid stress are just making them worse.

So you always have that protection of optimal hormone balance. And then once that's lost, we've lost that buffer. Those things that we're there are now becoming amplified.

It's so important that you do work on that foundation. That's kind of my end. My mentor always said 10 years ago, he said, Daniel just assumed people are xenotoxic and cortisol resistant.

And so looking at the health of the nervous system, we can't heal for in a state of dysfunction or sympathetically driven, constantly operating cortisol, which kind of then sits in blocks receptors and then other receptors, the other hormones can bind.

cortisol management, where we're washing ourselves in cortisol and our babies were still. And I didn't see that in mind with our second pregnancy. Our baby was very different than our first where it was happy-go-lucky, you know, the nutrients were dialed in, supplements were dialled in.

Exercise was dial in and the second one, when crap hits the fan, that's to say it nicely, but things get real, real fast and cortisol is through the roof.

I see certain kids. have this presentation now of, I didn't know what terrible twos were until there was a second child. And it doesn't mean that there's any less love or anything like that.

But again, it's a very stark awakening of our decisions, how we live our life, what choices we make, What is the environment internally and also externally, marital relationships, et cetera, really is implicated in the health of the child, which then is generationally, right?

As they carry this with them. And then again, they now begin to wash their children with kind of the hormones that are on board at the time. In terms of genetics, I think you mentioned UGT to be seven, if I recall, but I know the UGs they serve a role in conjugation and.

subsequent elimination of the xenobiotics that I was talking about where we have all this kind of chemical soup that we're not living in and this constantly exposed to these endocrine disrupting hormones.

I know it has specific specificity for catecholestrogens too, which plays a role in estrogen metabolites, but what are some of these other hormones that maybe your, or excuse me, genes that maybe you're looking at in terms of optimizing hormones.

And I know you mentioned one related to testosterone clearance, but in general, are there specific kinds of classifications that when you, when your dissecting those, or are, there other very kind of specific genes that you were looking.

Terms of, optimizing DHRT or just in the general propensity for someone to have any sort of hormone imbalance. So there's definitely kind of my like short checklist and then my bigger ones, but a lot of our CYP enzymes are important to monitor.

And there is so much information coming out of like our GWAS studies that reveal kind impact both of underexpressed and overexpressions within some of these pathways.

C-Y-P 11A1 is then a mitochondrial derived C Y- P pathway that's converting cholesterol into prognatholam. It's kind your second step in steroid synthesis.

The first step is STAR, which is your steroide acute regulatory protein. That is not a polymorphism. That's basically a gene that if you had any issue with it, it would be incompatible with life.

So that's not what I look at because if your making hormones and you're alive, you've got it working. But the next step, CYP11A1, that an important one because that is going to be that conversion, going into pregnenolone and even a little further into progesterone with the help of 3-beta-HSD.

I do like to look at the CYPs for estrogen detoxification, so C-YP-1B1. 1A2 plays a little role in our estrone conversion and estradiol conversion into 2-hydroxy as well.

It's primarily caffeine though, that's an important one, especially if you have any issues with COMT. If you're a slow methylator and you don't metabolize caffeine very well, it's like a double hit on not doing your estrogens any service either, because now you've just backlogged your estrogen clearance through that path.

pushing into the right pile because of caffeine. So that's a good one. And CYP3A4 is metabolizing roughly 60% of the medications and toxins in our environment.

It is a heavy hitter in the liver, and it does also metabolize estrogens into estrogenic intermediates. 16-OHE1, which does bind to estrogen alpha receptors, adds to that proliferation.

Does not cause a tumor to form, but it can cause one to grow faster if one is present and estrogen sensitive. So that's a cautionary one, but it also is good for our bum.

It gives us a little backup when you too is low. So there's like, again, it's that Goldilocks. Its partner is 16-OH-E2, also through the CYP3A4 pathway.

BHRT Dosing, Cycling, and Menu2019s Hormone Support

Colloquially, we call that estriol. It is not its own estrogen, it is a metabolite of estradiol, and it binds to estrogen receptor beta. That's why it helps with vaginal lubrication.

And can actually counter and protect breast tissue a little more when estrogens are a bit higher. So it's like a give and take, a yin and yang of all of our hormones, but a good one to look at.

Not just for medication response, But also then for hormone response. But that's 3A-4. Again, COMT, big one, because that's our catecholamine-O-methyltransferase.

That is for the catochole estrogens, the two and the fours, hydroxy intermediates. And that is not methylation, it's methyl detox. It's a little different, but it is dependent on the end product of our methyl cycle, which is SAMe.

So SAME is required to be donated by CompT. to our catecholestrogens to deactivate them. So I do like to look at that. Are they fast or slow metabolizers?

And what does the urine reveal? Is that a place we really need to intervene or are they actually doing pretty good? The risk with rapid, sometimes people are like, oh, it's rapid methylation.

That's great. They're clearing out their estrogents really quickly. It's a balancing act, right? Because too rapid a clearance can then cause, even if your estrogen's look great on paper, if you're processing them out rapidly, you are going to feel estrogen deficient.

And it can also process out your dopamine, norepinephrine, and epinephrin. So you're going to be more prone to kind of reward seeking behaviors and depression and agitation.

And you are kind going of be seeking those things that bring pleasure and gratification. All right, so it is a balance, but too little. Now you're backlogging dopamine and you are up and ever.

You're more anxious, now you aren't sleeping. And now your agitated all the time, you feel like your heart is racing and your backloging your estrogens.

So again, even if estrogen is great on paper, Now, your feeling estrogen dominant because you not methylated well. so kind of the two sides of that coin.

Other ones, So the UGTs, it's a whole family, just like the CYPs, You have the UgT2B17, which is that deletion testosterone. you have UgT1A1. and 1E1, which are gonna be more your estrogen breakdown and some of your xenoestrogens as well.

It's also processing our 16 hydroxys as a sulfation. I do like to look at salt 1A1 and I like do look to salt 2A2 and those are going to be involved in that sulfations of estrogenes and the DHEA.

So DheA in circulation comes chiefly all from 2a1 salt, 2 a1 in the adrenals. It allows it to circulate out. And I like to know, are they activating that pathway?

Inflammation pushes that away. It says, no, we don't want to circulation DHEA right now. We need to push it into the tissues because it's going to support immune function.

That's a precursor to some of our immune cells or kind of supports that synthesis. So it is a nice one to look at and to now if they push that too heavily or not just by proxy.

Again, NQ01, another big one when it comes to hormones, especially BHRT. Because if they have even one variant in the NQ01 and the commonly researched RS loci in a promoter region of Nq01, they've down-regulated expression, which means they're not going to protect their nucleus the same way as they would if it was fully functioning.

And in those cases, the may benefit from ubiquinol instead of ubiquinoe as a supplement because they don't convert their inactive CoQ10 into active Coq10.

They may actually benefit form delta-tocotrienols as well. And again, you just want to really promote Nerv2 in that case. So I do like to look at that guy.

Man, my list goes on and on. SRD5A1 and 2. If you're a promoter of testosterone through the 5-alpha reductase path, it means you are going to be even more sensitive to things that would upregulate that pathway anyway.

And so insulin is going be a big driver as are some of our inflammatory cytokines, namely IL-6. and some of the non-coding RNAs that get expressed and upregulate inflammation as well, anytime that gene is expressed.

You can look at IL-6 and IL6R1, which are the receptors for interleukin activity. That's again, it's just kind of furthering the depth that could impact our testosterone metabolism.

I mean, yeah, I would say that's a good comprehensive list. The aldoketareductase pathways, some those, again that is another family of enzymes that are heavy in the liver for biosynthesis and that pushes data.

So there are some that I may sometimes look at, but those are not well studied. So I wouldn't put heavy street cred on those. But it is one that when I see a beta systemic preference, I might think they may be needing some bile pool support, some liver support.

And they maybe dealing with some environmental toxins that are like kind of burdening their liver a little bit. Fatty liver can also push beta. Anyway, is that a nice summary?

I can go further. We definitely love further and we'll definitely have to do round two. I know we're approaching time here. A couple of questions that I have remaining.

So I knew when we had spoken initially in person, you had mentioned cycling hormones. Coming off hormones, what, how do you typically advise that? For cycling females coming out, which, so rephrase.

Possible females. Okay, coming off of hormones or cycling them. So some sort of a hormone break, hormone vacation, or hormone pause. Yeah. Are you, are you doing that with everyone?

Is that warranted for everyone. I know you said the goal is always to mimic kind of endogenous, you know, inherent cycles, which difficult sometimes to track, but how are doing all that?

Postmenopausal women, you follow the moon cycle is kind of what I say, because you're not going to have menstrual cycles any longer. Although when you cycle progesterone, particularly you may have a little light bleed.

Well, it is expected as long as you do pre-testing and you know that your endometrium is looking good and then screen on a fairly consistent basis. I'm not worried about light spotting with the drop in pro gesterones that can happen with cycling hormones.

But yes, I think When it comes to hormone activity, our bodies, again, like we said at the very beginning, we don't just make a hormone and keep it steady state all day long, every day, all-day.

It goes up and down throughout the day. Our estrogen and our progesterone receptors are used to those pulsations. They don't want to be bombarded all the time by higher doses, especially if you're doing higher dose therapy.

There is some interesting research coming out on progosterone and allopregnenolone. That's the intermediate that when you take oral progeterones in particular, but just any time progenitor hits the liver, you are converting into that allo-pregnanolones intermediate, and that's a neurosteroid.

it can cross the blood-brain barrier, and it's an allosteric modulator of GABA, meaning it increases influx of Gaba into those neurons, which can improve relaxation.

It's like that calming and sedating effect that progesterone is known for. There is a caveat, there are some women that will actually get a paradoxical response to progeterones, where they actually are getting an inflix more in glutamic acid response, a different receptor, getting the inflx often driven by inflammation, by high cortisol, like high stress.

But with allopregnenolone, it again, it's a neuro steroid. We want a little of it, we don't want it on and high all the time. And there are some emerging studies coming out saying we may actually be creating almost like a It's not quite the same, but almost like a benzodiazepine addiction in the brain if we're pushing allopregnanolone all the time.

So it's upregulating these calming receptors too much and the brand almost becomes codependent. And then when you try to take them away, they get worse and end up having to be on it forever because it like desensitized your brain and now you're dependent on this progesterone.

And we don't want that. Again, we want to kind of pulse in the natural cycles. So often I will have my postmenopausal women follow a moon cycle. New moon to full moon, they're just on their estrogens.

And then full-moon to new moon kind mimics like ovulation to men are, or to your next, sorry, to you next period, kind in a way to release of an egg. That would be with progesterone 200 milligrams nightly.

and so that cycle seems to really work nicely to mimic the cycle, a natural cycle for a woman who would be naturally cycling, because we don't make progesterone all the time.

When you rupture a follicle, that residual tissue after you released an egg is called the corpus luteum, and that's a sac that lasts 14 days out of the month, then that makes progeterones.

So women are really only getting projesteron during their cycle. For 14-days, we get a lot more projesterone for nine months during pregnancy, but those are like short windows of time, and usually only a few times in a woman's lifespan, if at all.

So I always questioned, like, why would we then give lotus progesterone all the time to a postmenopausal woman? Should we not be pulsing it? And that's, again, what the research is now saying, maybe we should pulse it.

And maybe, we shouldn't mimic that 14 days. 200 milligrams, oral at bedtime, support the nervous system, but don't over push that allopregnantal and that neuro steroid activity.

Allow your body to have a little bit of recovery and a reset as well. Estrogens are doing a lot of the heavy lifting when it comes to hypothalamic response and neurotransmitter synthesis.

Testosterone plays a role as well, but we're getting a lot of our serotonin support from estrogen, as Well as dopamine. So when estrogens are on board and at the right dose, sleep should still be good, even without the progesterone.

Some women get scared. They're like, But progesterone is helping me sleep. I need to be on it all the time. It's like well but estrogen is really the one, right?

It is the estrogen that's supporting tryptophan conversion to seratonin and serotonin then becomes melatonins. And that is our safe hormone. So you're still getting that nice gentle response, the right hypothalamic response in the brain with the complementary progesterone when you need it.

Again, if you are dosing in this way, you'll often get a little light breakthrough bleed. And I always just warn my patients that's to be expected as long as we're monitoring and reviewing the endometrial response.

Some women, they do just want to on that low dose all the time. If that is the case, again, we do this bioindividual to every patient. I'm not saying there's a right or a wrong, and again this is very new to you.

investigation, but I do think it warrants further approach and support to really dose per our cycle versus per constant dosing. And then with the estrogens, I will often just keep them on a lower dose for the first two weeks and then we can go up a little in the dose in second two week.

Again, to mimic kind of that rise and fall that estrogen has naturally during cycling years. So, so that's where you're doing so higher P and then as in your cycling off of P, right?

And then incorporating higher E and vice versa during those four weeks. So it's higher ENP at the same time together, and you drop off the P. And just bring in a lower dose E continuous.

So it's like new. So again, moon cycling, because it is 14 and 14. It's 28 day cycle, you know. New into full moon, I'll usually do just a lower dose.

I like to do a bias, but again there's, like, people say, well, then you're competing with estradiol, just give straight up estrdiol. You can do that as well.

really are paying, you're partnering it to the patient and to their needs. Estriol has its benefits, estradiol, has it's benefits. I don't overdose on the estriole and sometimes I might just dose it vaginally or topically like for skin support as well to just get a little systemic balance.

But just kind of a lower, low to low moderate range for that day one to 14 of that moon cycle. a new moon to full moon and then full-moon bring in the progesterone 200 milligrams at bedtime and up the dose on the estradiol a little bit.

Did that make sense? Are you ever adding in projesterones for males? Yes. And that was, I would not have said that even just like a few years ago. But I think more, so again, testosterone is the unsung hero or the under-researched hormone in women.

Testosterone is mildly androgenic. It is your precursor. coming off of pregnant alone converts into androstenedione and can support is part of that kind of testosterone conversion.

But so in that way, you know, progesterone therapy can shrink the testicles just like testosterone can, because it does create a feedback loop that drops androgen conversion within the gonads.

So you'd have to be aware of that. You'd just have balance it with testosterone. But I do think pro gesterones has its place and can help with sleep, can't help it mood.

I don't think the dose has to as high. And, but I hear, I see some functional docs in the male hormone space dosing just as highs. It's getting more exposure, more, there's a more.

experimentation happening, I would say, in real time. But I do think it is a hormone worth evaluating if a male can't sleep or has a lot of stress and doesn't have that stress resiliency and just isn't balancing or regulating their nervous system.

Progesterone helps a LOT with that, and men do make a little progesteron. And its primary role that we know in the research is that it supports sperm capacitation.

So it's supports that kind of mitochondrial drive for sperm to be active, to good swimmers, and to able to implant into an egg. That said, again, supplementation, or replacement rather, can prevent spermatogenesis, just like a testosterone exogenous prevents spermetogenosis in men.

Just be cautious. With use, make sure to, like I would use it in a male who's done having children, if they needed the support. Just as I would cautionary with testosterone.

And I often balance them together. Yeah. In terms of aromatization, which is obviously very common with men and

why they're going on TRT off and up. To me, it's a disservice when you go to some of these clinics and some these men who have very high adipose that are just automatically put because their levels are low.

Unfortunately, they often just get worse because they are aromatizing. What are some nutraceuticals that you like that will help intervene? So, you know, that's another very interesting pathway for men, right?

Because you have, so you've GNRH from the hypothalamus, gonadotropin-releasing hormone, telling the pituitary, release LH. Lh knocks on the testicle door on light egg cells and says, okay, let's upregulate testosterone synthesis.

Then testosterone will aromatize a little in the testing to help with sperm generation. But most estrogen in men is coming from that adipose tissue. It's from peripheral cells.

When estrogen goes up, it does a few things. So it's coming from testosterone, which means it stealing in a way, kind of stealing testosterone. It's converting over into estradiol.

Estradiola will tell the liver, make more sex hormone binding globulin. So you're making more taxis and loading up your testosterone and circulation and not being able to access it as easily.

The other thing it does is it tells the brain, hey, we have plenty of hormone. We probably shouldn't be pushing so much because I'm getting a little too high over here.

It's kind of a safety, but in doing so, GNRH drops, LH, drops and testosterone synthesis declines. So it becomes this like vicious cycle. of what little testosterone is being made is getting pushed into estradiol.

Estradiola is telling testosterone to get all bound up, and then you're not making any more to replace it. It's a problem.

DHT, DIM, and Nutraceutical Strategies

The aromatase cycle for men is driven heavily by adiposity, but specifically metabolically active, like inflamed adecose tissue. So it's insulin resistance.

Its inflammation. its leptin resistance, its high cortisol. We have to treat those factors. And then we have lower that estrogen so that the GNRH cycle can return and we can start generating LH again.

You can screen serum L-H in men and see what is that pulsation. Do we need to support? We want to target the liver. We're going to support the lever as well.

But I find that herbs like Tongkat Ali. Tongcat Ali is a great herb for men who over-aromatize because it can help reduce CYP19A1 expression, which is aromatase, and it lowers SHBG binding affinity to testosterone.

So it's a really nice herbal support, really targeted for women. Women don't get as much value. but men can see some improvement there. Tribulus, another herb that's wonderful for boosting androgen response and kind of getting that LH response to return for androgensynthesis.

The other big one that I like for men is Damiana, which sometimes gets used for women as well as Fenugreek. Venugreek, it's like the testophen. Do you know that brand?

Yep. So that's balancing blood sugars as part of its role, but it boosts androgens. Damiana boost libido really is more of the nootropic. It's working on the brain side, the hypothalamic side.

In terms of inhibition of CYP19A1, It is about reducing the promoters first and foremost. Balancing blood sugar is whatever your favorite way is. Diet, exercise, very critical, But also cortisol management, And again, that blood sugar balance with herbs like berberine.

You can also think of some chromium, or you can think other ways that, you know, your favorites. I like milk thistle as well, again just to protect the liver.

But there's, there are a few. Chrysan's on that list aswell. Crysan is an interesting one. It's a flavonoid, but it is from passionflower, among many others.

Passionflower is kind of its primary extract. Chrysin is not well absorbed through the gut, and yet it seems to show in research to have impact on decrease in aromatization.

And the more we get to research into microRNAs, these little non-coding RNAs. The more, we start to think it may actually not be the chrysin itself as a flavonoid that's causing the impact to reduction of CYP19A1.

It's these micro RNA's that residualy send messages to reduce inflammation actually in the guts that then alter. CYP1981 expression. So I do still think it has a place, although many people will argue, oh, but taking it orally has no bioavailability.

I think you are still getting microRNAs and we just are at the cusp of research on what that means, because we do see impact with that a little bit. And I'd say I get a lot more impact from some of the herbs, and of course, diet and exercise and cutting out alcohol.

That's the other big one that drives up C-Y-P. In terms of DHT, That's often vilified or demonized. What are you, how are looking at, at DHT? Is there a genetic propensity and how you are maybe shifting that favorably, even though it is, would you say three times more androgenic?

It is the potent form of testosterone, right? And it's made chiefly in the prostate. There are some other tissues that will also convert testosterone and androsenedione into DHT.

And even in a gut, you can get reverse conversion from some of our corticosteroids into DHD, which is interesting. Specifically, clostridium shindens can cause this like reverse pool of androgen exos.

They're actually studying it right now in prostate cancer, as well as for PCOS risk factors. Ah, I'm so gut. But yeah, again, it's a Goldilocks hormone.

You want a little, you need it. It is the anabolic component, like the heavy hitter for anabolic activity for testosterone or for skeletal muscle activity and brain health, right?

And even bone. But it is a promoter to prostate hypertrophy. So we have to be careful. SRD-5A1 and 5A2 are your primary five-alpha reductase enzymes. There is research around the SRD5A1 as an increased risk for promotion into DHT, specifically when blood sugars are dysregulated and there's high insulin levels.

So again, it comes back to blood sugar balance, chiefly. There are five alpha reductase inhibitors. I'm always cautious with them because again, we don't want to suppress.

So like flat out use of a medication called finasteride, it blocks by Valfariductase and it can create what's called post-finasterid syndrome. And this is devastating, especially to younger men who are using this to help with like hair loss.

Because DHT, what does it do? It creates terminal hair growth or terminal, hair follicles within like the scalp hair. So that means that they no longer go through the telogen phase and no under growing.

They just kind of drop and then fall out. And then you get that male pattern baldness. It can also create darker hairs on the body, on their face for women as well.

This is more concerning usually for them. and it can cause cystic acne. So again, if it's too high and its going rogue and hitting these other tissues because it is the skin, it the prostate, and a little bit in the brain that we get this

conversion happening.

And it a skeletal muscle again that chiefly uses it for anabolic performance. But if its too much and not getting metabolized, that's when it becomes a problem again for hair loss or acne And we can inhibit, but by using finasteride to inhibit that's global inhibition.

And it can actually lead to erectile dysfunction, weight gain, and a lot of like just fatigue. That's very hard to recover from. So I'm not a huge fan of suppression hardcore, like, absolute suppression.

There are herbs, though. Salt palmetto is a great one. That reduces 5-alpha expression but won't directly inhibit it. You stop salt palmito, you're going to get DHT conversion once again if you need it, reishi mushroom, actually a good one, nettle root.

Nettle is great. Great for the prostate. Reduces SRD 5A1 and 2 expression. So reduces that DHP. Vitamin E. vitamin A. Very important as both free radical scavengers, but also protective to the cross state.

I like to use those anytime DHT is higher as well. Sardines. I always say the superfoods for men are sardine, pumpkin seeds. And again, because of the healthy fats, the omegas, vitamin A and E, and then the pumpkin seed with the zinc and selenium.

Those are your other two minerals that are really critical for prostate health, but also to regulate DHC activity and the gut. No, I love that list. In terms of DIM, you hear that all the time.

When is it appropriately used or maybe sometimes when it is overused? I had a 23 year old male patient come to me. He was like six foot seven. It's very tall, very athletic.

he could not for the life of him put on muscle. You like had this goal. His body fat was down to 12%. He wanted to get it lower. And he wanted it like into the 10% range.

Um, and he just couldn't push that threshold through him. Uh, he was having issues with libido. erectile function as well. When he had fatigue, he just wasn't like motivated and going through his history.

And this was just in one sitting with him. A prior doctor had told him he was over aromatizing and needed to take them to lower his estrogen and that would solve his problems.

Estrogen is a balancing act for men and dim also metabolizes testosterone. So two factoids there that were working in his disservice and we tested his urinary hormone analysis and he was low with his estrogen, like almost non-existent.

I was like, yeah, I think you're on too much dim. You need to come off of that. And that was the only change we did that and then just kind of tweaking protein intake and when to engage with like don't over push or over exercise, but also when, to have your rest days based on his genes.

And, and how to push for like each high intensity interval training. Anyway, it's a whole different discussion, but he came back and was like, yep, doing great now, salt.

And it was basically just taking out DIMM. So I think DEMM does have its place, what it needs to be used correctly. and it shouldn't be use continuously in men because it does lower estrogens and circulation and estrogen plays a critical role for men.

You don't need a lot of it, But it is increasing nitric oxide synthesis. If you don't have nitric oxide synthesis, you do not vasodilate. You need vasodylation in order to get blood flow into the penis for an erection.

So if your estrogens are too low, You may not have enough bloodflow. And that can be a problem. It can also, it's also supporting PEMT, which is de novo choline synthesis in the liver.

Um, and it supports BDNF in their brain. Acetylcholine and long-term memory consolidation and kind of neural flexibility. So knowing this, it was like, yes, you don't want too much estrogen, but again, don t want it in the toilet.

Having DIMM on board, I see it as a benefit in men who are struggling with weight but are doing things to work on losing that adiposity. But while in meantime, we need to lower that estrogen so that it's stopping the suppression of GnRH so we can get LH to return so you can testosterone back on-board.

Dimm in that situation when estrogens are frankly high, or way higher than ideal compared to testosterone, that's when we can start saying, yeah, let's use it for like three months and then take it out.

And then really it's working on the gut. I would say I honestly rarely used them. It's mostly my morbidly obese male patients that I will use from time to time just to protect the prostate from that excess estrogen too.

In females? Dym can be great in women, but again, it's the same caveat. You don't want to lower it too much. I have had cycling women where if they're taking DIM every day during their cycle, they suppress ovulation because they are not getting the rise in estrogen that they needed, the right surge to release a follicle.

So instead, there are now prolonging their cycles. And they were like, hey, I just started this D. M. detox product and my cycle is suddenly 33 days long.

What's happening? You probably took it for, you know, took too high a dose at the wrong time during the cycle. DIM for women is great second half of the circle as a cleanup crew.

I usually will actually recommend it day 21, so mid luteal into day seven of next cycle, really just use it as that cleanup of when estrogen should be on the decline, help it move out if they were prone to keeping it in circulation and then clear it out.

in the next cycle as well, just so that they don't have as much painful, heavy menstrual bleeding. That said, DEM is only supporting reduction of estrogen through the upregulation of CYP1A1, primarily.

A little bit 1A2, a little 1B1 depending on genetics. It does not work on methylation, it does work not on bowel health. If you are not having regular bowel movements and or you're a poor methylator, DIMM is not a good solution.

I have had too many women get provoked on DEMM because they didn't work in methyilation and they did not worked on their gut. Again, if you not have bowel movement daily.

Calcium deglucurate might be a better option in tandem with just optimal fiber and just broad liver support and acetylcysteine, taurine and phosphatidylcholine.

If you're a poor methylator, those can also help as cofactors for methylation support. But then also again, bring in like magnesium, a little methylfolate if needed or folinic acid, which is calcium folinate, not folic, acid different, but if you are good with Methylating, sometimes people who are methyl sensitive, folinic, or calcium folinate, right, is that one, can be a better option than straight up methylfolate, but it just depends on the patient.

And then your B12 as well. Cobalamin tends to be more targeted towards COMT. But if your genetics MTRR or MDR are off, you're not methylating your b12, You might need methyl b 12 as Mostly, if they're not having regular bowel movements and they are poor methylators, I won't use dim or I'll try to stay away from it.

Yeah. There's so many better options out there anyway. Dim's just like this one little piece that can help. It's a profound reducer of estrogen in the right scenario, but it's not always the rights.

Yeah, I think it all starts in the mitochondria. That rate-limiting stuff that I talked about, STAR, steroid-acute regulatory protein, it is a mitochondrial stress sensor.

Safety of Hormone Therapy and Final Takeaways

It will down-regulate if there's any oxidative burden to the mitochondria, your gonads, which are gamete-producing tissues, so ovaries for women, testicles for men, are the tissues of greatest mitochondrion density.

in the body, the heart is a close second. So when you're looking at hormone support, don't forget the mitochondria. The other big concern there is the environment, right?

And the Xenoestrogens and the micro nanoplastics that we're exposed to that can't tend to disrupt and create oxidative burden to Those poor mitochondria.

So whenever I see low hormone synthesis or poor hormone conversion occurring, often it's the mitochondrion that I definitely want to focus on first and foremost, in addition, again, to the environment and to gut and nutrition.

I'd say that's my one last caveat that we've touched on yet. That's why we definitely have to do a round two. You look at all kinds of kind of all diseases related to hit on the Mitochondria, In terms of say someone's coming to you and everyone tells them hormones are not safe.

Obviously we have seen otherwise now. Context doesn't matter if they're, if their synthetic. Well, I don't want to go down necessarily synthetic, but obviously what happened with the, with a study that we're all aware of now?

The feeling comes to you and they're very reluctant or skeptical. What is the quick, maybe 90 second overview or what is education you're providing them in terms of not only is this very safe, but it's essential.

But I would say number one, I always meet my patients where they're at. I have tried to intervene for patients who did not want BHRT, who were kind of scared of it or resistant.

And yet, you know, we spoke about the tie. So we explained, the Women's Health Initiative, this was a flawed study. It was actually a cardiovascular study, It began as that, and then they converted it or kind of switched gears into hormones because they were looking at estrogen as a way to prevent heart disease in post-menopausal women.

And so they're choosing, like their selection was these obese, older, kind-of high-stressed, highly-oxidized women with heart-disease risk factors. Then they are like, oh, estrogen costs cancer.

This is a problem. Anyway, they cherry-picked and raised alarm over poorly done research. And we know this now. And in fact, the initial researchers of the study pulled out.

They were like, this, we can't be a part of this. and they've actually given a retort now to it and said like hey, these were the flaws and here's, you know, there's actually a great book called Mistakes Were Made But Not By Me that goes into some of these studies, including the WHI, that really go into the floors and like where they went wrong and how that was a disservice to women's health for decades.

What we know is that, and there's actually some great graphics. They actually come out of the NIH and then in the UK, they've done a lot of research and advocate for BHRT now.

There's a big movement there to bring awareness back to the benefit of BHT. And they have some really good graphics that I like to present to my patients that you can just look up.

BHRT use UK, and you'll find these graphics that show that like the risk of cancer from estrogen, your risk a breast cancer, from drinking more than two glasses of alcoholic, two servings of an alcoholic beverage per day, increases your risks for breastcancer much more significantly than BHT.

In fact, estrogen alone as therapy, can actually reduce breast cancer risk, right? This high risk from something like, and so I often will just tell my patients, I'm like you know, if you're drinking two glasses of wine most days out of the

week, that is a greater risk for breast, cancer than if we were to bring in estradiol therapy.

And the estrogen therapy is going to be supporting your bone, your brain, Your vascular health because of that nitric oxide. And so much more that we're still unveiling, right, is in terms of its role on like hypothalamic volume and response for dopamine and serotonin and BDNF, memory and consolidation.

We know that women are at greater risk for dementia and Alzheimer's than men. And part of that is this loss of volume in our brain for long-term storage of memory because of the drop in decline in hormones.

So absolutely critical, but again, meet them where they're at. provide them the information. Um, I will just give you a few stories of like, you know, bringing estrogen into two patients that just weren't right ready for it yet.

And I would say, don't do that. Don't push it, but just continue to offer support and then target these other tissues, the brain, heart, liver, right?

Our metabolism, our thyroid and other ways until they come around because the two times I kind of, like overly convinced and pushed. They had the worst outcomes on BHRT.

It was just like, oh, geez. Let's unravel this a little and bring it back. And a part of it was actually for both cases, they were mold patients. We were like what is going on with your hormone metabolism?

This is so unusual to have this strana reaction to such small hormone. I think again, part was the fear factor that was still in the patient's mind. So just getting them to that place where they do feel comfortable, giving them that information, You know, showing them these studies that are now retorts, the black box warning has been lifted off of estrogen therapies, right?

Like we were seeing the proof now that it is not the danger that we thought it was when done correctly, and safely and using bioidenticals, mimicking our own hormone.

But also test the environment, make sure they're in a good place. If they are mold sick patients, they not going to tolerate hormones very well, it turns out.

And that's something that you really want to support as well. That's my parting thought there. This is like an aside, but also why it's so good. That was, that was beautiful.

And the mold piece, especially because many of our patients as of late, it's been a very ubiquitous population in terms of who we're seeing now. That's one thing I'm very kind of steadfast as I refuse, everything is very contextual, but we don't start hormones with any of my patients.

I tell them exactly the order of operations. when we're going to intervene, when do we kind of clean out that toxic burden, that internal toxic environment?

And some of it is externally driven, but needless to say is when you said it turns out, if that's correct. So our hormones can be very protective on bone, breast, heart, you mentioned cardiovascular support.

Muscle is our organ of longevity. There's so much benefit they can provide. The context certainly matters when we are intervening, how we're intervened, our testing, what we were looking at in terms under the hood.

Obviously what you develop with endoaxis to me is absolutely brilliant. It sets practitioners up for success to how can be very, very targeted in term of treatment, both in the terms of nutraceuticals and obviously just BHRT included.

This has been an absolute masterclass, mini master class in all things hormones. We'll likely have to do around two in the new year, but I genuinely appreciate your time.

Where can people find you, follow you? Do all that. So I'm at Holgenic Wellness on Instagram. You can find me on the same as Facebook. Those are my two social media platforms right now.

I am not a social person. I'm trying to be though, I am realizing I like behind the scenes person, now I coming up. I thought I was doing more, going to get more engaged I promise.

We're also at endoaxis.com, you can learn more about the platform. Again, it is intended for professionals, but you cannot see our catalog of products.

If you are a patient, bring it up with your provider, see if they're engaged and interested. It's free for them, there's no cost. So if their interested, they can play around with it too.

What you've done is nothing short of brilliant. And I appreciate how detailed and again, thorough you are just in general, not only today, but every time it is incredible.

Thank you again for your time. Last parting question I have for every guest is if you could have one superpower, what would it be? You know, I think about this all the time, if I had one super power, think I would be able to transport myself anywhere around the world.

It is a common theme. Yes, I hate flying. I think it's the big reason there. Don't want to get on an airplane, but I love traveling. So they're awesome.

You are wonderful. This has been awesome to everyone listening. Thank you. Hope you guys enjoyed it as much as I did. Feel free to like and subscribe, share this with someone who would benefit in terms of anything related to optimizing health and performance, obviously, especially hormone related.

Until next time, keep asking questions, stay curious, and remember, your body's own data has the key to your healing. Thank you again, everybody. Until the next, time stay healthy, Stay wealthy.

Thank you for tuning in to the TBD Fit podcast on Dr. Talks, where we unpack all things health and longevity. If you enjoy the show, like, review and subscribe.

This allows us to have a greater reach and help others on their health