THE SPECTRUM OF HEALTH
PODCAST

Podcast Session #62

The Thyroid Debacle

With Dr. Kelly Halderman &
Dr. Eric Balcavage

Dr. Christine Schaffner speaks with one of her most popular guests, Dr. Kelly Halderman, along with new guest Dr. Eric Balcavage. Together, they discuss their new book, The Thyroid Debacle, the Undiagnosed Epidemic of Cellular Hypothyroidism.

For more, visit: https://www.amazon.com/Thyroid-Debacle-Eric-Balcavage/dp/1628603984

Dr. Christine Schaffner: Welcome to the Spectrum of Health Podcast. I hope you enjoy my conversation today with Dr. Eric Balcavage and Dr. Kelly Halderman. We're talking about their new book "The Thyroid Debacle: The Undiagnosed Epidemic of Cellular Hypothyroidism." I've had Dr. Kelly Halderman on the podcast before. She has so much information to share with us not only about thyroid, but also about the concept of Phase 2.5 Detoxification. And now I interview Dr. Eric Balcavage, who is a wealth of knowledge, I learned a lot about the thyroid in this podcast and I hope you do as well. And please, please pick up their book, which is excellent, called "The Thyroid Debacle." We'll have all of this information in the show notes.

00:43 CS: Well, I am so excited to interview my friend, Dr. Kelly Halderman, and her co-author, Dr. Eric Balcavage. We have two wonderful experts today to really dive deep into this whole thyroid debacle, as they call it. As many people know, I treat chronic illness and I would say probably 95% of my patients have some type of thyroid dysfunction, and it's not always clear from the lab work. It's a complex situation, often we look at labs, but also clinical history, and of course, they feel better when we treat the system. And of course, the system is so tied into every other system, not only the endocrine system. We have to look at the body as a whole, and we're going to do that in this wonderful podcast. So welcome, Kelly and Eric, I'm so honored that you're on the podcast today.

Dr. Eric Balcavage and Dr. Kelly Halderman: Thanks for having us.

01:43 CS: You both just wrote a book. Can you tell us a little bit about why you felt this topic was so important that you spent the time and the energy to educate us all?

01:58 EB: Well, I think with where we are today with thyroid physiology, the title of the book says it all. We've got people who are being diagnosed with hypothyroidism, we're using one lab marker essentially to diagnose them. That lab marker is not a great lab marker to be assessing thyroid physiology on its own, and we have patients that are being told they don't have a thyroid condition until this one marker goes out of range, and then they're put on thyroid medication and they still don't feel well. And then they're told that, "Hey, you just eat too much, you don't exercise enough or you're just crazy." I think where we are is really a debacle with what we're doing with thyroid physiology, and we really need to take a different look at thyroid physiology, how we look at thyroid physiology, and really how we interpret blood chemistry and blood physiology, and really look at our patients to begin with. I think the book, even though it's written about thyroid physiology, I think it's a great book to help anybody understand the concepts of how to be healthy and how to assess health and physiology.

03:18 CS: And when you say that one lab marker, are you referring to TSH?

03:24 EB: Yes. The standard process is if someone thinks they have a thyroid condition, their doctor will run a TSH or a TSH with Reflex to T4, which means they'll run a TSH test, if their TSH is elevated or above lab range or below lab range, then they will run the next test, which is usually a T4, or a free T4. But if TSH within normal range, then the person is told they don't have a thyroid condition. But the problem with that is that the literature says that by the time TSH goes above the lab reference range, the person's lost 90% of their thyroid gland function. That's not the beginning of a thyroid condition, that's the end of a thyroid condition.

04:18 EB: The other problem with using one test, TSH, to evaluate thyroid physiology is that we're assuming that it's not impacted by anything. The number one problem we struggle with in this country and many countries is chronic low-grade inflammation, and inflammation will suppress TSH making it seem normal when it really isn't. And the third problem is, is that one of the most common medications we take is something called Metformin, which is a drug for blood sugar problems and diabetes. There's a number of other medications, but definitely Metformin suppresses TSH, essentially hiding a thyroid condition. And so we've got one marker that's suppressed by the primary problem we have in this country, and it's also suppressed by some of the most common medications we take. It's just crazy that we're using that one marker to determine the state of somebody's thyroid physiology.

05:16 CS: Yes. Just thinking of my practice, I would miss a lot of thyroid dysfunction if we didn't test not only TSH, free T4 and free T3, but also the reverse T3. Not to jump ahead a bunch, but it's more complex and I agree, I think it's just amazing how long these things take to change to become a new standard of care for people to get this information. I know you're doing your part with this book to educate people so they can feel empowered and go to their doctor with this. You two coin this term "cellular hypothyroidism." What do you mean by cellular hypothyroidism, and how is that different from a primary hypothyroidism condition?

06:06 EB: When we talk about cellular hypothyroidism, what drives hypothyroid symptoms is insufficient T3 reaching the receptors inside somebody's cells, and that can happen with a totally healthy functional thyroid gland, with normal levels of thyroid hormone in the blood, but the individual cells of our

body actually have control of what happens to the thyroid hormone once that thyroid hormone comes into the cell. The cells can either activate it, convert to T4 to T3, or they can deactivate T4 into something called reverse T3. If T3 is in the bloodstream, they can bring it in and they can take it right to the nuclear receptors or they can deactivate that to one of four forms of T2. But what drives hypothyroid symptoms is a reduction of available T3 reaching the receptors inside the cells. And it's our opinion, and we talk about this in the book, that that happens long before the gland becomes dysfunctional in most cases.

07:05 EB: Some type of cell stress, excessive cell stress, triggers what we call a 'cell danger response,' then part of the natural adaptive response to excessive danger at the cellular level is to slow down metabolism and ramp up inflammation. And one of the ways the body does that is by deactivating thyroid hormone locally, and that's the beginning of a thyroid condition. And then if the stress is persistent and chronic, then eventually we start to see changes at the thyroid gland, but the thyroid gland is not the beginning of the disorder. Primary hypothyroidism is what most people get diagnosed with, that is when you've lost 90% of the function of the gland, and the primary cause of that loss of function is what's called an immune or autoimmune process, something called Hashimoto's thyroiditis. And that is gland dysfunction, but what's really driving the symptoms is cellular hypothyroidism, and many times that's happening for weeks, months, years or decades before the gland finally becomes dysfunctional.

08:16 CS: Such a great point. And there's such an opportunity to help people before the gland becomes harder to regenerate and repair, right? You mentioned the cell danger response. Some of my audience is familiar with that,

and for some people this might be a new term. But what you're sharing is essentially because of certain stressors in the cellular environment, that changes how we convert thyroid into the active form of thyroid, and it seems like that stress or that inflammatory response creates more reverse T3, which essentially inactivates the cell. Am I getting that right?

08:55 EB: Well, we look at it like it's a deactivated form, but I think the key is that the lab markers aren't the problem, they're the story, right? So when we see the increased reverse T3, definitely in allopathic medicine, they don't pay really any attention to it, but in functional medicine, we pay attention to it and sometimes people think it's a bad thing. They think that the reverse T3 blocks T3 from binding to receptors and that high reverse T3 actually is what's causing hypothyroid symptoms, and that is not the case. Reverse T3 actually has a benefit. While it's not necessarily an active form of hormone, there is a huge benefit to the deactivation of the thyroid hormone in cells, especially when there's infection. White blood cells specifically utilize and actively deactivate T4 to T3 just for the purpose of liberating iodine because iodine is an antimicrobial.

09:57 EB: So it's actually part of the defense process. So if you have bacteria or an organism inside the cell and the cell is deactivating thyroid hormone, not only is it slowing down normal metabolism, not only does that reducing thyroid hormone prevent the transport of things like glucose into a cell that could actually support an infection, but it also produces iodine, which can be used as an anti-microbial. So there's a benefit to some degree of having the cells deactivate that thyroid hormone and increase reverse T3.

10:31 CS: I love that. I never have really heard it explained that way, and that makes a lot of sense. The body is so wise, right? It's adapting to a stress response. I've been educated to learn that if reverse T3 goes up, it can be there's a chronic infection that's affecting the body. I learned other reasons of why that is, but I love that, that we use a lot of iodine, not only to support thyroid function, but yes, as an anti-microbial and an anti-parasitic. That's a really wonderful explanation, that the body is actually trying to treat the pathogenic stress, I love that. I love learning something new every day. So you all also say that there's a spectrum of hypothyroidism. What do you mean that hypothyroidism is a spectrum disorder?

11:35 EB: Well, when we take a look at hypothyroidism, it happens in phases. Cardiovascular disease doesn't start when you have your fatal heart attack, diabetes doesn't start when you get diagnosed with diabetes, it starts at the cellular level as a regulation issue first, and so thyroid physiology starts that way too. I think the gland is rarely the issue first. It's not that it couldn't be, but I think it rarely is first, because if the gland was the primary issue and all the downstream chemistry worked, then all you would have to do is put thyroid hormone into the system and everything would be turned back on and then people would be great, but our patients don't show up that way. Our patients show up frustrated.

12:21 EB: The scientific literature is showing that the patients are frustrated and doctors are frustrated, because patients have biochemical euthyroidism, their TSH is normalized with thyroid hormone, but they still have weight issues, they still have constipation, low libido, thinning hair, they have still all the symptoms. So we look at it at the cellular level and consider that hypothyroidism starts there. And so when there's excessive cell stress, you get the

deactivation of thyroid hormone as a net, that triggers hypothyroid symptoms and what symptoms you experience depends on what tissues are being most impacted by thyroid physiology. It's not like a switch in your house where either everything's turned off or everything's turned on. It's more like some systems are turned down before others, and so everybody can have different hypothyroid symptoms because it all depends on what cells and tissues in their body are more stressed, at least in the beginning.

13:24 EB: But we start with this cellular hypothyroidism, slowing down of metabolism, and this can develop for weeks, months or years, and TSH can still be normal. And then if it stays for an extended period of time, the danger response that caused the deactivation of thyroid hormone gets ramped up, and that cell danger response triggers the release of more inflammatory chemicals, which upregulates the immune system, but that cell danger response also releases things called DAMPs and PAMPs. I don't know if your listeners are familiar with those terms, but DAMPs are pieces of the genetic material or pieces of the cell, and PAMPs are pieces of the organisms. If there's a bacteria, a virus, or some type of organism that's infecting a cell, the cell will spit some of that information out so that it can alert the immune system that, "Hey, this is the problem, this is the thing you want to come get and clean up."

14:21 EB: And it's interesting that the thyroid cells have little receptors. They're called 'pattern recognition receptors,' and they reside on the thyroid cells. If those DAMPs and PAMPs bind to the thyroid cells themselves, to these receptors, it'll initiate the thyroid cells to go into a self-destruct mode. And it is often the thyroid cells themselves that are inviting the immune system, this inflammatory cascade, these white cells to come into the thyroid and

actually create destruction. Now, we call that an autoimmune condition, and we say that it's a mistake, but I don't know. I really don't think it's a mistake. I think it's more of a calculated response to, "Hey, we've got a lot of cell danger, we've got a lot of stress, we need to globally slow metabolism down," and then you see the slow down of metabolism or you see the damage to the thyroid gland.

15:12 EB: And we call that Hashimoto's or an autoimmune condition, and so that would be that middle phase. And then after weeks, months or years of this attack at the gland, that's when the person finally goes into their doctor, their TSH is elevated, their T4's low, and their doctor says, "You have primary hypothyroidism." But that's the end stage of a thyroid disorder, not the beginning. At that point they've lost 90% of the gland function, they can't even make any more. And now it's like, we're in an emergency situation.

15:43 CS: That's an excellent description, and kind of a reframe of this whole still conventional framework of an autoimmune condition. As you mentioned, autoimmunity in a conventional setting is explained as the immune system turning on your own tissues and damaging a gland, like your thyroid gland. And so, again, I know all of us on the call here trust the body's intelligence and nature's intelligence, and we have to take a step back and learn and see the bigger picture. I guess my question is, when I think about autoimmunity or Hashimoto's, we think sometimes there can be a viral trigger or even a toxin trigger, an exposure like a heavy metal that can turn this process on. What are some of the primary triggers for the cell danger response for a cell to feel threatened and produce these DAMPs and PAMPs that turn on this inflammatory process?

16:52 KH: I think that the main triggers that I see and that I think across the board that we're dealing with are biotoxins, such as Lyme, Epstein-Barr, CMV, certainly heavy metals as well, and mold. I think mold is under-appreciated as something that can set off the cell danger response. And Dr. Eric always talks about, and he's taught me about the allostatic load, and that we're really not in a state of homeostasis anymore because our bodies are challenged. We're challenged in the environment that we live in and in the air we breathe, the water that we drink, the food that we eat. Our bodies are constantly trying to detoxify and balance back, and so this allostatic load, it just is overwhelming. And so then, let's say you get bitten by a tick and you have an infection with that, you're just so overwhelmed. And I think for setting off autoimmunity, and Eric will comment on this as well, it just depends on your unique physiology. I do believe that my Hashimoto's had something to do with the chronic Lyme disease that was undiagnosed for years and years and years.

18:09 KH: I do think that played a role in it. Will I ever know 100%? No. But I do know that I've been in and out of the cell danger response multiple times in my life. I've had runs of Epstein-Barr, I've had Lyme disease, I've been in moldy homes before. So it is a matter of trying to quell down...Well, first to get at the root cause, and that's what we as practitioners do, we try and figure that out, then stop that process. And what part three of our book focuses on is the recovery phase, the strategies, we call them fitness factors. And that's everything from dietary fitness, to environmental fitness, to your detoxification, and really making sure that we're looking at this comprehensively when we're trying to recover, when we're trying to quell down the cell danger response, reverse it and bring our health back. Eric, I'll let you comment on that as well.

19:07 EB: Yes, I think when we're talking about what can trigger a cell danger response and trigger cellular hypothyroidism, it could be anything. As Kelly said, it's the load. Sometimes we're looking for the one sexy thing. Is it Epstein-Barr? Is it this one thing? But sometimes it's not a thing, sometimes it's our emotions. Sometimes it's our poor breathing, sometimes it's our chronically disrupted sleep. Sometimes it's trauma, sometimes it is poor diet, sometimes it's chronic infections. Sometimes it's all of those things. But the issue is that sometimes it's sexy in functional medicine, you'll see Epstein-Barr causes it. Yes, maybe it did. But that's not everybody's cause. And we have to get away from the idea that the same stress caused the same result, that everybody has hypothyroidism for the same reason. You can have it for 10 different reasons. What triggered my Hashimoto's wasn't necessarily infection-related.

20:06 EB: Mine was, I was training in my 40s like I was still in my 20s for endurance activities, and I was sleeping four hours a night because I thought that's cool. And I'm trying to study, I'm working all day, I'm coaching my kids. I'm trying to do everything...Business, family, train, work and sleep, and what does that do? That just runs you into a hole. So I didn't do anything most people would think would be terribly unhealthy, but poor breathing and lack of sleep and over-training was probably what drove me to have Hashimoto's. And so I'm doing my blood work, I'm thinking, "I'm gonna be great." But I'm insulin resistant, I've got Hashimoto's and my inflammatory markers are elevated, and you have to ask, "Okay, what am I doing wrong?" If you asked the average person, "Is that a healthy person?" They would say, "Oh yeah, they're doing all the healthy things," but just because they're healthy activities...

21:10 KH: That brings up a great point, Eric, because we were just discussing how with hypothyroidism, we think there's a typical phenotype. But I'll tell you this, for the audience, if you don't know Dr. Eric Balcavage, he's one of the most fit people that I've ever met in my entire life. When you told me you had the Hashimoto's, I was blown away. So that is a really good point that several things, even the emotional...We know from the work of Dr. Naviaux that emotions and trauma that we go through can set off the cell danger response as well.

21:48 CS: Yes, it's so multi-factorial. And you all brought up so many great points. I can echo that, yes, I think sometimes we get fixated, it's this one thing or everybody has this. Especially with the chronically ill patient population, if it was one thing, they wouldn't be so sick and it would be easier to get them better. So it's kind of this whole broad perspective and looking at all these buckets and really treating people comprehensively. I guess I'm curious how you both would work up a patient, just thinking of everyone who's listening and saying, "Oh my gosh, this is me. How do I communicate this to my doctor? How do I start investigating what are my own triggers that I need to start addressing?" Are there any clinical pearls of assessment or labs or work-up that you want to share today?

22:45 KH: Oh, there are plenty, Christine. In our book, in part three, you will be blown away. We went into such depth, I'm so proud of this work. There's the part one where I talk about my allopathic training, what I learned in medical school, and give the reader a perspective of why the doctor is treating the way they are, how they learned that. So I come in and do that. And then in part two, Dr. Eric lays out all this beautiful science that has taken him years to

accrue, and we just heard some of the pearls that still blow me away as a practitioner. And then part three is where it's loaded with pearls. We go into things that I didn't even know existed, such as these respiratory techniques, and there's 10 fitness factors. So really in there are concepts that I think are valuable to a lay person, a practitioner, it's very valid for anyone. And it is very valid for not just people with hypothyroidism, but also those suffering from just chronic conditions. They're really helpful. Because who couldn't benefit from cleaning up their diet? We know that. But having them all laid out in ways that there's calls to action and there's things that people can do, like you were asking, Christine--that's what we're really proud of.

24:11 EB: And we talk about, from a lab perspective, the tough part is, if you're going to an allopathic physician trying to convince them to do a different kind of thyroid panel or run a more comprehensive blood chemistry panel, it's going to be really difficult because they're working within a set of guidelines. If they step outside those guidelines they may get their hand slapped like, "Why are you doing that? Those are not medically necessary tests." But when we're looking at patients who have thyroid issues, I think one of the things that's really important is that, A, we run a true comprehensive thyroid panel. Typically, if you ask them if they got a comprehensive thyroid panel done, they'll say, "Yes, it's a TSH and a T4," and they don't realize that there's probably nine or ten thyroid tests that can be run to assess thyroid physiology, and all of them have some value. And so we want to look at all of those tests so we can determine what's going on with somebody's thyroid physiology. And then on top of that, we think it's really important to look at, along with that comprehensive thyroid panel, to look at a comprehensive metabolic panel. And it's not as simple as just looking at a lab panel for highs and lows.

25:34 EB: We talk about this all the time, that when we look at lab values, it's more important to interpret labs based on your patient's signs, your patient's symptoms, and what they're experiencing. You could have a totally normal lab value, but it's inappropriate for your patient. If you had a patient that was sitting in front of you that was tired, fatigued, their hair was falling out and their eyebrows are falling out, they have no libido, you would say, "Wow, I think they're hypothyroid." But if their TSH came back normal, you might say, "Oh, then they don't have a thyroid problem." But that's reading a lab report, that's not interpreting it. So then we would have to take a look at, "Okay, if that TSH is normal but my patient's got all these symptoms, do I have indicators like inflammatory markers that may indicate that the patient does have inflammation, which would suppress TSH and create this symptomatology?"

26:26 EB: If you don't run a full thyroid panel, you can't see the rest of the markers. And if you don't run a comprehensive metabolic panel with some of those things like inflammatory markers, you miss it. And so I think that's really, really important for doctors to understand and patients to understand, is that we've gotten so far away from what a comprehensive metabolic panel is. When I first started in healthcare 20, 30 some years ago, a comprehensive panel was like 60 tests. Now it's under 14. And so what gets measured gets managed. If you don't measure it, you don't have to manage it, and then it doesn't exist. Yet our patients are struggling.

27:05 CS: Yes, such great points. And when you're looking at a comprehensive metabolic panel, for people who might not know what we're talking about, it's typically looking at electrolytes, liver function and kidney function. I guess are you taking into account what's normal for that patient? I know Kelly

actually educated me on the role of bile and thyroid hormone conversion, so looking at maybe liver markers, bilirubin markers. But what kind of pearls do you see in your patients and their comprehensive metabolic panels that points to thyroid dysfunction?

27:44 EB: Well, first of all, if they have inflammatory markers that have elevated. So I'll look at CRP, I'll look at homocysteine, I'll look at fibrinogen, elevated ferritin, low bilirubin. You'll see a lab report with bilirubin less than 1.3. Well, what does that mean? If a patient's got a bilirubin less than 0.5, they probably have oxidative stress and that bilirubin's probably being used as an antioxidant. We'll look at uric acid. There's a number of tests on there we can look at for inflammation. The other marker that's a really good telltale sign that the person's probably got some level of cellular hypothyroidism going on is elevated cholesterol. Thyroid hormone's critical to HMG-CoA reductase, and so if you have cellular hypothyroidism, they're going to probably have elevated cholesterol. That's even been talked about in the guidelines, but they just kind of ignore it when they're assessing thyroid physiology.

28:42 EB: A third thing to take a look at is insulin resistance. So whether you're looking at higher fasting glucose, higher fasting insulin or elevated hemoglobin A1C, to get glucose into the cell requires what they call transporters. Glucose transporters. GLUT transporters. And all those GLUT transporters require sufficient levels of T3 in the cell to have them activated. So anybody who's got insulin resistance, pre-diabetes, or diabetes has some level of cellular hypothyroidism going on. So those are just three or four things that we could say are simple things that if we run a more comprehensive panel, we can see. Definitely if we see problems with elevations of VLDL, we know that we've got problems with not only bile flow, but we've got problems with

potentially thyroid physiology. If LDL is elevated, you need thyroid hormone for your LDL receptors on your liver to work. So if you don't have good cellular levels of T3, you can't bring LDL into the liver to get it cleared out of the system.

29:51 EB: So there's so much value in looking at a comprehensive metabolic panel where you can say, "Okay, we've got problems going on at the cellular level. We've got cellular hypothyroidism." And we're not out to treat the cellular hypothyroidism, but that's just telling us the story that, "Hey, something's going on in the liver that's creating cell stress, that's deactivating thyroid hormone. That's why that LDL levels are elevated. That's why VLDL's elevated. That's why we've got liver enzymes that maybe...That's why bilirubin's low." So when we really interpret labs, not just read them for high or low, man, you can get a ton of information, and we can see the story of why our patients are struggling.

30:30 CS: I love this. I'm learning a lot. I know the connection with total cholesterol being elevated in cases of hypothyroidism, but a pattern that we see is there could be kind of a normal cholesterol, maybe a little bit elevated, but most of my patients have a high LDL, and so this is another lens to look at it. And looking at this is really another clinical indicator that we have to circle back and look at their thyroid. So, great pearls. I know that we've talked about this wide net to cast and look at this as a multi-systemic, multifactorial process, but as we get into treatments...A lot of people, depending on how they've been diagnosed, they're on some type of thyroid replacement hormone. And so there's a whole art of thyroid hormone prescribing. Where do you stand on thyroid hormone replacement? How do you approach that?

31:46 EB: So I think if somebody's got glandular hypothyroidism and their levels are down, they're going to need some support. But I think where we get into problems is now we see people arguing whether it should be T4, should be T4/T3, should be T3 pulsed multiple times a day. And we're trying to fix symptoms by putting more thyroid hormone into a system. If you can understand the concept that Kelly and I are talking about in this book that, "Hey, the problem's at the cellular level, the body is trying to deactivate thyroid hormone, it's trying to shut down thyroid physiology," and now you're trying to essentially fill a bucket up that has holes and your arguing about, "Okay, should we put water in there or should we put soda in there?" Well, the problem is you have holes in your bucket. So the real solution is to fix the bucket.

32:39 EB: When it comes to thyroid physiology, I rarely ever have to tinker with thyroid hormone, which thyroid hormone to use or recommend. Most of my patients come to me and they're already on thyroid hormone. I'm not a medical doctor, I don't prescribe it, and I don't take them off of it. But what happens is, is that many times what I'm doing is sending information back to their doctor who put them on the thyroid medication to reduce the dose or change the dose. Because as you fix the things that Kelly and I talk about in the book, we improve their fitness factors, we identify their stressors and reduce or remove those stressors, and support the healing and recovery, the thyroid often recovers. And so they don't need it. So the argument is, should it be T4, T4/T3 or T3, and my argument is, "You give just enough to support basic physiology and then get to work fixing why the body is trying to deactivate to begin with."

33:40 EB: If you don't fix that, the argument doesn't matter because none of those things will fix the problem. I just had a patient, I talked to her yesterday.

She's overweight, she's obese, and so her doctors were driving her TSH, her TSH was 0.001. Now, they didn't do the rest of the panel, they were driving it low. They didn't run liver enzymes, they didn't run anything else, all they were doing is running the TSH and she hadn't had anything run. I ran the enzymes, liver enzymes, her liver enzymes are off the chart, ALK-phos is like 400, her GGT's in the 300s, her other liver enzymes are in the 200s, her ferritin is sky-high, fibrinogen's sky-high. And I'm like, "Wait a minute, we've got major issues here. This is a person who's had breast cancer, we shouldn't be loading this person with thyroid hormone, A, because we can re-trigger the cancer, and, B, we're missing something." And matter of fact, I think we're overdosing her because her free T3 was way too high and her reverse T3 was really way too high, it was like almost into the 40s.

34:51 EB: I'm like, "One of the things we need to do is get her off of thyroid hormone. That'd probably be one of the best things we can do." And in 30 days of cutting that thyroid dose down, her liver enzymes improved 50%. This lady, by just doing the same principles we've talked about in this book, she has lost 40 pounds, her insulin's gone from mid 20s to 10, she's no longer in the diabetic range as far as hemoglobin A1c or fasting blood sugar, her blood pressure is normalized, her liver enzymes are improving, and I think they'll improve even more if we can continue to get her doctors to lower the thyroid hormone dose.

35:43 CS: What a great story. That's incredible. And I agree. I do prescribe thyroid hormone and a lot of my patients have the clinical signs and symptoms in the labs that it's indicated, but yes, I'm not satisfied by just that solution alone. I agree that when using medication like thyroid hormone, you need a strategy of taking people off of it. I think sometimes we just get in this habit

of, "Okay, we've found the problem, we add the medication and we're good to go for a decade or two." And then we don't look at the plan, that this is a short-term solution as we fix the underlying causes and really try to reverse this. So I think those are really excellent points, and obviously some wonderful clinical studies or clinical cases that you just shared, and I know that you guys experience every day.

36:43 CS: My other question is, when I think about thyroid health, I think about, of course, the thyroid's role especially in women's health, thinking about the thyroid in relationship to our adrenal health and ovarian function. Of course it's significant in male health as well. Can you talk about that interconnection with thyroid as well as adrenal and other reproductive organ function?

37:18 EB: Yes. We talk about this all the time, nothing works in a bubble. Almost every cell, every tissue has thyroid receptors. So what we see is that when we have this excessive cellular stress, we get slow down of thyroid physiology, that's an adaptive response. But we see all the other systems adapt as well. And so we see changes in adrenal physiology, we see changes in GI physiology, we see changes in neurotransmitter physiology, we see changes in hormone physiology. All these things adapt. Somebody will say, "Well, I have adrenal fatigue and I have dysmenorrhea and I have this condition, I have reflux, and I have..." You know what? They're given all these individual diagnoses, especially if they haven't been diagnosed with primary hyperthyroidism yet, and somebody's telling them that they're all not connected. And that's nonsense.

38:19 EB: We refer to it as multi-system adaptive disorder. And so when the body's in homeostasis, there's plenty of energy to run all these systems. But

when we're under excessive levels of stress, there's not enough energy to adapt to the stress, so something has to be shut down so that energy can be pushed towards the thing that's most important, and that's survival. And so what we see is all of these systems start to become compromised in time. And what people don't have is, "Oh, I just have diabetes but it's not related to my thyroid condition," or, "Oh, I just have IBS and it's not related to my thyroid condition," or, "I just have gall bladder disease and it's not related to my thyroid condition."

39:01 EB: It's all related. And I think it's super naive of us to really think that it's not related. It's just crazy to think that. So when a patient comes to see me and they say, "What's wrong with me? I have these five, six diagnoses." I'm like, "Listen, you have one problem, you have excessive cellular stress, and we need to identify what those things are that are causing that excessive cellular stress and help support you. And then what we need to do is support all these other systems that have become compromised, that's an adaptive response, that's not a mistake by your body." Then, they'll probably get better. We may have to support them, but they're not unrelated conditions, they're all totally related.

39:45 CS: I completely agree. That is a great explanation. I'm hoping our conventional world will wake up and see this one day. I'm so glad that functional medicine, naturopathic medicine, all of these other views of the body show that we're this interconnected, multi-system network, and we cannot just treat anything in isolation. I think that's a really wonderful point. I know Kelly's done a lot of work with genetics and you probably have as well, Eric. Do you guys have a place with all of the genetic information that we have access to on our patients? Do you highlight that or feel like that's an appropriate

place to look for why some people might be more susceptible or vulnerable to cellular hypothyroidism or hypothyroidism in general?

40:50 KH: Yes. In our book, in part three, one of the chapters is on genetic fitness. There are some specific polymorphisms in thyroid physiology that give us clues to why a person can be experiencing the symptoms. But moreover, I think that the name of the game is always epigenetics. So we wrote the chapter on genetics, and we put some of that information in there because that's always of interest to me. You're right, Christine. I really think that it has merit, and I think we shouldn't throw the baby out with the bath water. We're not treating any sort of gene. But I think it's part of the picture, and I think it's important, just like we discussed when we talked about Phase 2.5 detoxification.

41:37 KH: There were patterns that I was seeing, and I used those patterns that I was seeing in patients to really put it together, to put together the ACAT and how we're making bile. And that really helped me, and it helped a lot of people, and it still does to this day. So that's what we did in that genetic chapter, is we used some of the data there. But we were always stressing the epigenetic factors and how that'll always take precedence over your genetics and how you're actually expressing them. Eric, did you want to comment on that at all?

42:17 EB: I think the key is too, epigenetics is king. Because what we see is that while there is maybe a predisposition, I've treated hundreds of patients over the last 20 years and their genetics stay the same, and yet we're still able to get them to recover, to be able to convert T4 to T3 better. So there are definitely SNPs that put people at a greater risk of conversion issues, but

I don't think those are necessarily the primary. Those may be some of the few cases. I think in the vast majority of people, it's just excessive levels of stress. And what we see in thyroid physiology or this adaptation in thyroid physiology, is just that: An adaptation to be able to adapt to the level of stress. If we look at the body and the physiology from that perspective, like, "Hey, if there's excessive stress, the body's going to go into defensive mode. Inflammation and inflammatory processes are part of that defense," we stop looking at the body like it makes so many mistakes, and we look at it for this amazing piece of equipment that it is.

43:33 CS: Yes. Great points. And as you're talking, I'm thinking of a few patients and actually colleagues, and I'm thinking about hyperthyroidism. Do you all have the same approach for hyperthyroidism as hypothyroidism, or any comments on that?

43:52 EB: So when we're looking at hyperthyroidism, again, we're talking about an autoimmune disorder, maybe a set of circumstances that predispose somebody to have more hyperthyroidism than a hypothyroid condition, but it's still an autoimmune issue. So while it can have more grave consequences, the solutions ultimately come down to the same thing, what are the excessive stressors that triggered the immune system, depending on what's going on. With Graves, is it to make antibodies that are binding to the receptor that are causing the over-activation? Something triggered that response. So we get the patient out of crisis, we lower the success of production, but then the principles are the same. We need to keep the foundational principles the same, which is, "What can we do to improve their general health and well-being, and reduce their excessive stress?"

44:50 EB: I don't think that changes, whether they have a thyroid condition, a hypothyroid condition, a hyperthyroid condition, a diabetic condition, coronavirus. I don't care what it is. It's interesting, when we wrote the book, I had comments from people like, "How come you didn't put the protocols in for how to treat a thyroid condition?" I'm like, "Because every person's thyroid condition is different." If you want the protocol go to section three, or part three of the book, and that's the protocol. Improve your diet, improve your fitness, improve your sleep, improve your breathing, improving your mindset, improve your habits. You want a formula? That's the formula, but there isn't a supplement formula.

45:30 CS: I love that. Of course, supplements and herbs and all of that have a place, but this is a lifestyle and we want this to be sustainable and correct it. And things don't just turn around overnight with even the right supplement, so I completely agree. How about, is there anything else, any other words of wisdom or pearls that you want to share? We've covered so much and you've definitely shared some insights that have my wheels turning for certain patients and just looking at things through the lens you just shared. But is there anything else on your mind that you want our audience to know before we tell them more about your book?

46:14 KH: I think two things come to mind. We set out to write this book for both the lay person and a practitioner. So there are going to be parts of it for a lay person that we tried really hard, there was a lot of re-editing on Eric's end, of trying to make it very digestible for just even someone who really doesn't even know where their thyroid gland is. So we really start off and try and give a very nice layout so that it makes sense. I've actually had some patients where they think that TSH is produced by their thyroid. Even the best

doctors don't have time to explain thyroid physiology, so again, we set out to write the book for the lay person, as well as to hand it to their practitioner, because it is a manual.

47:07 KH: Part two has so many references. It just really is based on the good clinical science that doesn't get to your doctor's office for 17 years on average, so here it is, here's the future delivered to you. And spread the word, because you don't find texts like this, you don't find people like Dr. Eric who take the time. You know, Christine, it takes so much time to go through the literature and research and then put these concepts together and then, again, deliver them in a manual where it's for both, again, the lay person and the doctor. I'm really proud of that and I really want the people who are listening to know that. Don't be overwhelmed because it's really science-y, because it's going to help you. Part three is easy to understand and to put into action. Literally, you could just skip to part three if you're really chomping at the bit, and you can put those into action the day you get the book. You can start. So it's really, really, really good stuff.

48:10 EB: I would say that Kelly did a really good job of laying out in the beginning of the book...While people might want to get to that part three right away like, "Well, how do I fix it," it's important for people to understand the medical perspective. It may come out like, "Hey, you're anti-medicine." That's not really what we're saying, we're not indicting our fellow medical doctors, we're not saying that they're the problem. Really, it's the whole model or the system of how we're looking at the physiology. And we don't foresee that changing anytime soon, but that's good, because that gives functional medicine its place, and allopathic medicine has its place. But when people read

that first section of the book, they're going to get a better concept of what medical doctors are taught and why they do things a certain way.

49:02 EB: It's not because they're necessarily ignoring you, they're following guidelines, they're following what the rules are. And it's the Kellys of the world who are in allopathic medicine and get frustrated themselves within that model or have their own health crisis, and then have to reach outside that model and look for something else, that they then come to functional medicine and they say, "Oh, okay, there's a whole parallel universe here I didn't even know existed." But we want people to know that their doctors are doing what they're taught, this is just a totally different model. If you just go in there and start talking to them about cellular hyperthyroidism, they may look at you like you've got three eyes because it's just not something they've probably been exposed to. They definitely haven't been taught it and they probably haven't spent the time to read it and research it. But this will help somebody understand why, when they go there and they're frustrated and they say, "Why aren't you running those tests," they'll have a better understanding of why. It's not their doctor just blatantly disregarding them. In most cases, it's just that they're following the guidelines that have been mandated to them.

50:10 CS: I think that's such a great point because it's really important to understand where people are coming from, and to speak their language. It takes time to change your paradigm or change a narrative. And as you said, there are the Kellys of the world who are in the conventional system and who have their own health journey and see this other way. As I see it, we're in this health crisis, and there is room for all medicine, and how do we really work more together? Conventional medicine is really amazing in a certain, very specific set of circumstances. And then this is where we shine: Functional

medicine, naturopathic medicine, alternative medicine. We are so well-suited to help people with chronic illness recover their health and look at the root cause. So I just honor both of you and applaud you for taking the time to write the book. I know that you both are really busy clinicians, so to actually take the time to write a book is no small undertaking, and I'm just super in awe, and inspired, and impressed, and I'm grateful that you put your knowledge base and research out into the world, so thank you.

51:26 EB: We appreciate that.

51:27 KH: Yes, and everything you're doing, Christine, we are in awe of everything you put out education-wise and your brilliance. Thank you so much for having us on your podcast. We really appreciate it.

51:40 CS: Oh, absolutely. I know this won't be the last time. And I'm excited to share your book "The Thyroid Debacle." We'll have a link in the show notes for where people can find your book and learn more about you all. Please share both of your websites, so people can learn how to work with you both as individuals.

52:03 KH: Sure. Mine's just drkellyhalderman.com.

52:06 EB: And mine is rejuvagencenter.com.

52:11 CS: Awesome. Well, you all have a beautiful day, and thank you so much for being on the podcast, and I'm sure we will be talking soon.

52:17 EB: Thank you.

52:19 KH: Thank you.