Why TSH Can Read Normal While Cells Run Low (Part 1 of 3) Transcript

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Thank you, Integrative Peptides. Now let's jump into today's conversation. You can't give thyroid to treat obesity for weight loss, but every obese patient is low thyroid and dramatic improvement in everything.

Every Hashimoto's patient, is Low Thyroid. and would dramatically improve with thyroid treatment. Not T4, not Armour, straight T3. And the science backs it up.

Welcome to the Peptides and Longevity podcast. Each week, we'll dive into groundbreaking science, innovative therapies, and the future of health and longevity.

Dr. Holtorfu2019s Thesis on Thyroid and T3

Alongside visionary physicians and experts, We'll explore how peptides, cutting edge medicine and lifestyle strategies can redefine the way we age. Get ready to transform your health, optimize your vitality, And unlock the secrets to a longer, healthier life.

Let's get started. Hello, this is Dr. Kent Holtorf and we're having today the presentation, The Haltorf Way, How to Revolutionize Your Practice with Peptides, Bioregulators and T3 Therapies for Hashimoto's, Hypothyroidism and Beyond.

So I was trying to figure out how to break this lecture up because there's a lot of information. And so we decided to do three parts but if you can learn thyroid is not that difficult you'll be amazed how many people you treat and get better when i basically my story of people i will go back into it but headline babesia bartonella going to anesthesia just because.

It was just too aggravating to talk to a patient. Hey, they're asleep. So I figured that I could handle that. But then I didn't realize you have to get up so early.

I ended up heading off to the integrative conferences and really learned T3. And I went and took over a family practice. and I changed the name to the ByWave Optimization Center and was able to convert it to cash in less than a year.

No advertising, it was just, we would get so many patients. And T3 would basically, what got me initially, dramatically better, high-dose T-3. When you look, and we're going to go through this, I hope by the end or even before that, that I'm going to prove to you that the way that we practice and are told to diagnose and treat thyroid hypothyroidism in the country, especially Hashimoto's, which we'll talk about, is wrong.

It is not evidence-based. it is leaving people untreated that could dramatically benefit from T3. And so many people can benefit Amazed it is a magic pill and then if you add peptides to that just doing those two things.

you can get essentially everyone in your practice dramatically better that you weren't able to without those tools before.

Immune Dysfunction, Aging, and the Thymus

It allows you to go from basically A to B to patients improving much quicker, much easier, safely. That's what I hope we can do and go through it. So the agenda, so part one, it's probably not the most exciting part, but discuss immunity and chronic illness and aging, thymus has to do with it, we'll talk about thyroid physiology, which people go, oh yeah, I know thyroid physiology, But when you really look at it it like the endocrinologist refused to essentially understand it.

So many people, if you have any stress, toxin exposure, chronic illness, elderly, they're low thyroid. If they are obese, you can't give thyroid to treat obesity for weight loss.

But every obese patient is low thyroid and dramatic improvement and everything. So we'll talk about also the unique associated pathophysiology in Hashimoto's, which I think you'll be surprised about.

And then part two, which we will schedule and let you know, we'll talk more about thyroid hormone transport, Which is really the key to understanding thyroid and appropriate treatment, thyroid replacement for Hashimoto's patients.

And it really is, except for a rare exception, every Hashimoto's patient is low thyroid, and would dramatically improve with with thyroid treatment, not T4, no armor, straight T3.

And the science backs it up. So we'll show you that. Then part three, we will get into more the immune-modulatory part. What they found out, as much as I love T-3, actually patients feel better if you have a choice to, okay we're going to modulate your immune system versus giving you thyroid.

they will feel better with immune modulation, actually, with the Hashimoto's patients. But when you combine them both, that's when get the big difference.

So just giving thyroid, it'll lower the antibodies. It's gonna fix a ton of issues that they have. And we'll talk about therapeutic protocols and that combine peptides, bioregulators and all that so we can get you going and treating patients very quickly.

But yeah, so let's talk about, we have to cover some basic science. We'll talk immune dysfunction, hypothyroidism, Hashimoto's and chronic illness. Really when you look at, when people get sick, old, stressed, they get essentially an immune shift.

TH1, you can throw Tregs over there to TH2, TH17. And it basically is the cause of chronic illness of aging and you've been the nice thing is you fix that.

It works for essentially every chronic illnesses every disease of ageing i know it's a bold statement but. When you do it is like wow it kind of like you have all the specialist they're just missing the boat.

and you'll be able to treat these patients better than the specialist. So I'll just quote, the immune system health is shown to be the biggest determinant of overall health and longevity.

Toxin stress age and chronic infections are major triggers of immune dysfunction, which result in the T1 Treg, a TH2 THFU chip, and a clear correlation between the increase in mean longevity and the leading indicator of cell immunity.

So everything's a vicious cycle, and I did this in 2003 for the fibromyalgia fatigue centers. We had 23 centers focused on treating the sickest patients.

You can see everything is a genetic predisposition, but genes only matter about 10 to, at the most, 20%. It's

epigenetic. And oftentimes, let's say they have chronic Lyme.

Most people that have Chronic Lymes don't have any symptoms, then all of a sudden they'll have a triggering event. and psychological stress is the biggest one is a nightmare on the immune system and just set everything up i can also be a number of talks and mold exposure again stress another infection but like a sign of a psychological issue divorce death in the family like that but.

Divorce is worse than death, actually. And so you get everything's a vicious cycle. You get immune dysfunction, autoimmunity, i.e. Hashimoto is one major thing.

Now you with the low TH1, you can't fight intracellular infections, so they all come out. Then that adds to the immune suppression and makes everything worse.

We get pineal, hypothalamic, pituitary hormone dysfunction including thyroid. So all these patients are low thyroid, but they have a low TSH, you know, low normal T SH and a high normal t4 and they low, normal free t3 and the high, normally reverse t three.

So these patience, if you go into cardiologists and say, well, their T S H is on low side, t four is high. Oh, they're high thyroid. Exactly the opposite.

The immune activation coagulation, mitochondrial dysfunction, The cells go into cell danger response and then they become senescent and you know, inflammar aging and all that.

So you need to essentially, one therapy is get rid of the senesen cells. But when you have low TH1, including a low natural killer cell function, that is what gets rid the the Senesen Cells.

And you also with low Th1 you can convert IgM to very weak antibody to IgG. So they can't essentially fight chronic infections. They can detoxify, you need energy to detoxified.

You need to energy transport thyroid into the cell. you get GI dysfunction, gut brain problems, brain gut problems. Leaky gut, leaky blood brain barrier.

Again, everything is a vicious cycle. So the shift is a marker for illness and aged phenotype. Increased morbidity, mortality, increased inflammation, increase autoimmunity, ends with mast cell activation, neurodegeneration, increasing cancer risk, reduced longevity and quality of life.

Yes. All these conditions are associated with again, this immune shift causes and then the, but the immune ship is driving all these things. So you, I'm not lying to you.

Here's a couple of studies showing chronic fatigue syndrome have this. chronic Lyme disease, stress or aging, get the shift, you get mast cell activation.

I won't go too much into this, but the patients have like, MCAS, Sears, and we have an ebook out that you're welcome to download on the peptide protocol for the rapid treatment of Sear.

All these things, they got POTS and others, the typical thing, very difficult patients. we can reverse that actually very quickly with immune modulation.

And, you know, thymus is key and it starts involuting around 15. It hits its main ear around 40 and then that's, a little lag and that is when all the disease of aging start because your immune system now is totally out of balance.

And you can see here's the thymus activity and then here is basically disease of aging and you see it just goes right together. And this is another representation of that showing how the immune function is inversely correlated with basically all the disease of aging, cancer, mortality, morbidity.

Why TSH Misses Tissue Hypothyroidism

And even the CDC admits that 80% of age individuals are affected with at least one chronic disease as a result of the involution of a thymus, right? And like the HIT trial came out and said, oh, we can rejuvenate the thynus.

Okay, yeah, a little bit. Even the study was not that impressive, but Why don't we give thymic peptides? They're cheap. They are totally safe. So it's crazy.

How much chronic illness can we prevent by just doing that? And you look at everyone's immune system nowadays, it is trashed, certainly promoted. therapies for pandemic is just disastrous on the immune system.

But this is a review article saying like, hello. Everyone, we've forgotten about the thymus, right? You can do all these things in terms of checking your biological age, and they get expensive with telomeres and methylation of the DNA, but the CD4-CD8 ratio actually is shown to be a very accurate measure of your biologic age.

So ideally it should be CE4 to CD8 greater than 2.5. The lower it is, the more aged you are and the worse your immune system. And there's just some essentially backup on that.

Thus the CE-4-C8 ratio may be used as an integrated marker for a biologic age. Again, so many things, a negative effect from the involution, increased thyroid dysfunction, septal infections, I've kind of gone through all these, cancer, obesity, diabetes, all the syndromes, chronic fatigue syndrome, fibromyalgia, and really they all have the same underlying pathophysiology.

Like we have ALS patients, come in wheelchairs, they're jogging and essentially treat them with some differences and the same as all these multi-system illnesses.

And so here's a question, what you were taught in medical school and residency about how to accurately diagnose and often we treat hypothyroidism Hashimoto's was wrong.

If you go to our nonprofit National Academy of Hypothyroidism, I have several review articles, each with 500 peer reviewed articles showing just that.

They're wrong and they won't change. Keys understand the thyroid, understand that the pituitary is different than every other tissue in the body. They have different diadenases, transporters, they react differently to stress.

The pitutary will maintain or increase the uptake of T4 and T3 in low-energy states and illness, where the rest of the bodies, it goes the other way. So we rely on the TSH but is actually the worst tissue to look at.

to try to judge the thyroid level in the rest of the body. The TSH level, in a chronically ill person, has an inverse correlation with what the tissue level of thyroid is per end of periphery.

When T SH is low, the pituitary level T3 is high, but the other body is starving for thyroid. So it's really the worst tissue to estimate thyroid levels.

So the production of the ROX is regulated by the classic hypothalamus pituitary thyroid access for the biological activity of thyroid hormone, which is availability of active hormone T3, the nuclear receptors, is mainly regulated at the tissue level by diadenases and the thyroid hormones transporters.

So those are the rate limiting steps, not the TSH. So, what steps are required for thyroid activity? Well, you have to have hypothalamic pituitary function, okay, then secretion of TSH, and then you'll have intact thyroid function which essentially secretes T4, that needs to go into the cell and convert T3, it has to bind the receptor and you then have downstream activation.

Okay, with all these steps here, are these abnormalities and dysfunction that are present common or uncommon in the chronically ill, depressed, obese, stressed, inflamed, and age individuals, like just essentially most everyone

you talk to.

Okay, how about hypothalamic pituitary function? Let's look at thyroid function first. That's what is thought to be, Pashimoto's is the most common cause of hypothyroidism, but it's not.

It's like the one of the least common, it just the fact that it is only one that they recognize. Problems with thyroid functions, not common. Hypothalonic pitutary functions dysfunction is common secretion of TSH disorders, very common.

Problems with thyroid transport in the cell, which is due to mitochondrial dysfunction, Very common, problems with conversion T4 to T3, very common receptor binding, common downstream activation.

We're not sure, not enough studies on that. So the reason that dysfunctional say thyroid function in which primary hypothyroidism, they say it's a Hashimoto's number is the most prevalent reason.

Only reason it's considered to be it because it is the only one the doctors check for and can easily be identified. Because all these other ones result in, again, a low TSH.

The TSh does not go up. And also, you know, if the Tsh was compensating, for lack of thyroid, why would a high TSH be considered low thyroid? It would fix the problem, right?

But anyways, all these common causes of low-thyroid aren't detected because they tend to have, and you'll see this pattern so commonly, Low normal TSH, high normal t4, because it's not getting into the cell, OK?

So it goes up in the serum. So what's in a serum doesn't mean what is in cell. And then they'll have low free T3 and high reverse T 3, which used to be a better test, but they got away from the RIA, reactive amino assay, Because they didn't like the radiation.

But now the reverse t3 is kind of crammed into a middle reference range. So this is your view of, you know, basic thyroid physiology, hypothalamus, erotropin-releasing hormone, tells the pituitary discreet TSH, which then tells thyroid discrete mainly T4, and then it gets converted to T3 in a healthy state and a little bit of reverse T-3.

Now, in an unhealthy state, what happens, you end up getting TRH, because that's what you get, TSH, too much thyroid, makes T4. Now most of it goes to reverse T3, and not much T-3.

We will get right back to the episode, but first a quick thank you to our sponsor, Integrative Peptides. If you are enjoying today's conversation and want to support your health and longevity, visit integrativepeptides dot com and use the code PODLIFE for an exclusive listener discount.

That's integratvepep-tides-dot-com. Code P. O. D. L. I. F. E. Now back the the and could be anything from stress, toxin exposure, inflammation, aging. So I put this diagram together.

You look in the middle. What things affect the deidonases? Let's first look at the peripheral cells in a body. They have type 1 deiodonase. That will convert T4 to T3.

Then they have Type 3 deodonasis, which converts T 4 to reverse T 3. In the pituitary, it does not have It has mainly type 2 diadrinase, which converts T4 to T3 in the pituitary.

Now, if you have anything such as diabetes, depression, dieting, inflammation, stress, obesity, PMS, leptin resistance, aging, autoimmunity, insulin resistance by a malignant cardiac syndrome, any stress inflammation aging.

So what does it do? Basically suppresses type 1 diadenase. increases type 3. So what happens in the periphery, you now have less T4 T3 in this cell, and you know have more T 4 to reverse T 3 And there's time to present, oh, it's just an inactive metabolite, but all the studies show that it blocks T3 receptor, reduces T4, T 3 conversion.

But what happens in the pituitary? It actually stimulates type 2-D adenase, which increases T 4 to T three. So now there is more T-3 in pitutary. And what do you get?

You get a reduction in TSH, so which the endocrinologists go, Thyroid is very easy. If the TSH is low, your thyroid is high. But no, that will hold for a completely healthy person without any stress or toxin exposure or chronic illness or has to be young.

And so I'll find that person for me. So it is not the ideal and basically, oh, you just got to look at the TSH. It's a terrible marker. Study showing type one, you had an ACE, a sensitive marker for a purple thyroid status.

I use it in the mouse, but same thing for humans. So it determines the level of T3 inside the cells of the body. Now, when you also just look at, let's just at and I put this together for bunch of studies and tracked free T4, free, T three and TSH by age.

And as you see, as she get older, and especially as you get an elderly person. T4 doesn't do much. Again, we talked about it not being able to get into the cell.

So the serum level stays basically the same. Okay, so, but their TSH goes down. Are these elderly people or older people becoming hyperthyroid? Is their thyroid working better?

If that's the case, because the TSH is the all-knowing, right, so low TSh, they have to be, thyroid has to better, and they're more thyroid activity in the body.

Well, then why does free T3, basically, it's a steady downward decline? So they are low-TSH with low t3. They kind of don't like to answer that question.

And here's also with just inflammation, okay, and we'll just look at CRP within the normal range. So 0.4 to 2 versus the free T3.

Diiodinases, Stress, Dieting, and Low T3

Look at just a direct inverse correlation, even going from, you know, 0 point 4 to less than 2. Free T 3 goes from 3 point something down to 1 point. And then there's another study which showed basically exactly the same thing.

Here is an article from TSH, may not be a good marker for the adequate thyroid hormone replacement therapy. Total thyroxine, pre-thyroxy, and TSH made with sensitive immuno-diometric assay did not accept in patients with gross abnormalities distinguish youth thyroid patients from those who were receiving inadequate or excessive replacement.

These measurements are therefore of little, if any, value in monitoring patients receiving thyrokine replacement, but the poor diagnostic sensitivity and high false positive rates associated with such measurement render them virtually useless in clinical practice.

We consider that biochemical tests of thyroid function of little if any value clinically in patients receiving thyroxine replacement. Further adjustments to the dose should be made according to patient's clinical response.

So it's also, what happens when someone chronically diets? Okay, how many people are those? Oh, that's like no one, right? You basically significantly suppress diadenase type 1, resulting in a reduced T4 to T3 conversion with a fifth reduction in T 3. But also you get an increase in diidenase Type 2, So the TSH is now suppressed.

So even though there was 50% reduction in T3, there is no increase in TSH. In fact, the drop from 1.2 to 0.7. Another study by Liable, if an individual is significantly dieted in the past, had on average 25% lower metabolism, equal to someone who weighed 60% less.

Additionally, reduction was shown to be years later. And we see this every day of our practice. So we check

everyone's basal metabolic rate and we checked their relaxation phase of their brachioradialis, which the British Medical Journal showed that an algebra doctor looking at relaxation phases of actually the ankle reflex was a better test than blood test.

It correlated with who would benefit from thyroid, who was high, and who is low. And we also have the gold standard, the basometabolic rate. Anyone who's sick, Can't lose weight, chronically ill, older, they're in general 25% lower metabolism than someone their same age and weight.

So what happens is, especially if they diet, it drops the metabolism and it doesn't go back to normal with normal eating. So when they go, oh, go on another diet because you need to lose weight, you have to fix their metabolism.

Otherwise, its a futile exercise in trying to get them to loose weight. Yeah, so other studies also confirm that acute or chronic dieting can result in a significant decrease intercellular and circulating T3 levels way up to 50%.

which significantly reduces basal metabolic rate in the number of calories burned per day by 15 to 40%. And again, resume normal eating, it doesn't go back to normal.

This is just a study where they're showing that the TSH had no value in measuring the intracellular level of thyroid and looking at a number biochemical markers.

their total thyroid tivity level. The TSH had no value, but they showed that the T3 or the free T-3 reversely ratio is currently the best indicator of tissue levels of the thyroid.

Chorus sensitivity of T SH and T4 is worsened and is further decreased in the presence of chronic illness. A study in a journal of endocrinology metabolism, which is the old joke, how do you hide something from an endocynologist?

put it in their journal. Just kidding. So they examined the accuracy of using TSH to identify hypothyroid obese patients. They did TRH testing and they found that while TSE levels were not significantly different between normal weight and obese individuals, 36% of obese patient had severe thyroid dysfunction not detected by standard TSA testing.

Diabetes, insulin resistance, diabetes, and metabolic syndrome, semen reduction, T-partidium conversion, undercellular deficiency. T3, increased inversion of T4 to reverse T 3. Additionally, the elevated insulin will increase diidenase type 2. And so again, that's going to suppress the TSH.

Diabetic individuals have, on average, in this part of the study, a 42% reduction in T 4 to T three conversion. We're giving all these diabetics, so we got some good meds and Zembic and Mengero, but you got to fix their thyroid.

You got fix your metabolism. And so in this study, they investigated T4-D conversion for the diabetic patients and a 40% decrease in free T3 levels. Their free to T-4 ratio was 50% less than controls.

And it actually, TSH failed to elevate despite the serum T 3 being half of normal. Another study basically came up with the same conclusions. In this, study kind of again, the, same results.

The authors also state chronic fatigue syndrome patients resemble those with non-thyroidal illness. So they kind of call it nonthyroidal illness, like let's say someone goes to the hospital and they're under significant physiologic stress and what happens?

Again, they get exactly the TSH suppression, T4 goes up because it can't get in the cells, pre-T3 is low, reverse T3's high, their cells are Dramatically significantly hypo thyroid and the argument is well.

It's temporary but these people are in chronic. Non thyroid illness right and show so she was cell danger response by a conjugal dysfunction. Now Leslie DeGroote is widely regarded as the founder of modern thyroidology.

He published 462 papers on thyroid and editor of the textbook endocrinology over 30 years and says that non-thyroidology should be treated with T3 because replacement of T4 would not likely restore tissue hypothyroidism.

And so the man says, sticking around with your T4. In this study, 94 Chronic Fatigue Syndrome patients measured thyroid function, and they looked at a number of different results.

They basically said the results were diuretic occlusion at TSH. Tests are inefficient to assess thyroid functioning, chronic fatigue syndrome, or fibromyalgia.

These Dutch and Spanish researchers found that THC levels were indeed similar between the chronic syndrome patients and healthy controls, but virtually every other thyroid measure was significantly lower.

So here's the group just saying stop treating with T4 and they are low thyroid, so they should be treated with D3. And fibromyalgia patients in this study, they found all of the fibomyalgic patients were hypothyroid using a stim test.

And in fact, the fibromyalgia patients tend to have low normal TSH levels at average 0.86 versus 1.4 in normal. So again, lower T SH. Well, I thought they're supposed to mean higher thyroid.

Absolutely not. In internal medicine, hey, PMS. They use TRH testing. 94% of patients with P MS have thyroid dysfunction compared to zero in the asymptomatic patients.

Other studies have confirmed that. Depressed patients, and we'll talk much more about this when we get into thyroid hormone transport and will look at more clinical tests with treatment, like treatment with T3 versus T4 or no treatment which will be the next lecture.

The way we treat thyroid in this country is wrong. Half the depressed patients found to be hypothyroidism but had thyroid function tests in the normal range.

And it's like the Starr Report, largest study ever done on antidepressants, showed that T3 was a better antidespressant with less side effects and much more efficacious for depression and work when the antidepressants did not.

Also had 135 bipolar patients totally treatment resistant. They tried on average 14 different medications, no response. The gave them all T-3, regardless of their baseline thyroid function test.

80% responded and right around 30% had total resolution assessment. This actually kind of belongs in the next section, but reverse T3 block cellular uptake of T 3 by 34% and T4 by 23%. So lectures about Hashimoto's, okay, let's get to it.

We can cover it pretty quick. But there's all this stuff to say, oh, Hashimoto's is got TH1, Graves is TH2. And I was just like, wait a minute. If that's the case, why do they all, and we say like this is a TH1 disease at mass and all these things, they respond exactly the same to treatment, to ones that are, you know, considered TH2.

So the problem is, is that they couldn't discern TH-1 from TH17. These are actually TH 17 dominant, not TH 1.

Hashimotou2019s, Obesity, and Misdiagnosis

And you also cannot go by cytokine testing because it's like the body will try to like increase TH1 and they'll test that and go, oh, is the TH 1 dominant?

No. The body can't raise it. It's low. So it is what it trying to do. not what it is. So again, circulating TH17 cytokine levels are altered in Hashimoto's and eventually, you know, basically just proving that all this dogma is wrong.

What's the standard approach with Hashimoto's? The endocrinologist feels it's simple. Easy to diagnose, easy to treat. Basically, if a TPO antibody or antithyroglobulin, they have high TSH, you just treat and bring that back to

normal.

If they had normal TSh, then you don't treat it. Well, is that the best practice? And long endoclinologists won't even check for Hashimoto's because they say we can't do anything about it!

which is crazy, but that's what they feel. So they don't even think about, oh yeah, Hashimoto's, they, you know, modulate the immune system and fix that.

And just, again, just treating with thyroid, even though their tests, their levels are normal, is dramatically beneficial. So studying the Lancet, probably heard of it, from thyroidopses and people with chronic fatigue syndrome, and with just chronic fatigued, not even chronic disease syndrome.

They found that 40% of the patients had lymphocytic thyroiditis. And now only 40 percent of those 40%, with the lymphocystic thyroidiasis, were positive for TPO or antithiroglobin antibodies, or had an abnormal TSH.

So they essentially missed 40% of people with low thyroid. And they also demonstrated that TSH is a terrible indicator of low thyrox. So after treatment with thyraxine, clinical response was favorable, irrespective of baseline TSh concentration.

Damn, that's a Lancet saying that. This is a study just showing that it's interesting, like HHV6 can affect the thyroid, but it is positive in the thyroid, in periphery it doesn't even turn up positive.

So causing a lot of inflammation. When you look at obese children, actually even obese adults, instead of Hashimoto's in obese, children is so much higher than healthy, lean children.

It's 60% versus 1.2% in this study. So they had 186 consecutive overweight children with workup, long story short, so 40% of obese children, with lymphocytic thyroiditis, were missed.

And we see it over and over, like the children that are overweight, their TSA, even when the TSH is like 8, they go, oh, no, we're not treating because if they just lose weight, that will go back to normal.

Well, Okay, let's just discount all the negative health consequences and I think even more the psychological health consequence of, you know, this kid is overweight, your destroying his or hers self-esteem and you're asking them to lose weight when their metabolism is 25, 35, 40% low.

It's mean, it's terrible medicine, that makes no sense. And then they'll say, well, T4 is shown not to help, so why treat? Yes, hello, P3 is showing to work incredibly well.

They'll use that, same with depressed patients. Well, depressed patient may seem a little high thyroid because their TSH is low normal. T-4's high, but we gave them T 4 and they didn't get better.

So prove you're crazy. Conspiratory thought that their hypothyroid, we proved it wasn't. No, you're giving the wrong med. So undiagnosed, Hashimoto's in obesity.

This was rats. T-front levels were 30% higher. It just fits exactly the pattern, right? And we won't get in there, we'll have a lot of treatment studies in part two and part three.

And this study also showed that T3 had significant immune-modulatory properties, so lowers the antibodies and also didn't affect that P4 is worthless and they should stop playing around.

So here, journal endocrine metabolism using TRH testing, they found that the TSH levels were not significantly different. between obese and normal children, but 36% of patients.

So here's their stimulation curve. They have central hypothyroidism, which by definition makes it so you cannot use a TSH for diagnosis. They like to forget that part.

If you go also to National Academy of Hypothyroidism, an article entitled, why doesn't my endocrinologist know this? I would recommend checking that out.

It kind of just gives a sense why. Let's see, your studies show the level and presence of Hashimoto's immune dysfunction. They found those with positive TPO or basically Hashimoto's.

they had a much higher level of fatigue, weakness, Your ability dry hair, nervousness, dysphagia, and as well as being more like a history of breast cancer, early miscarriage, low quality of life, including general health, physical activity, vitality, social functioning, mental health.

Even though their levels were so-called normal and they were not hypothyroid. Level TPO correlated with total number of symptoms per patient. TPL levels were highest in those with six or more symptoms.

Immune dysfunction would be more important for symptom relief than thyroid replacement, which we will talk about in the next section. So you want to modulate the immune system and give T3.

So, Hashimoto's and fibromyalgia are thyroid bubbles, autoimmunity or immune dysfunction are the culprit, and 52 patients, essentially the ones with the antibodies, were the one with worse symptoms.

Studies show that hospital patients have the highest known incidence of anti-pituitary antibodies. So basically causing pan-hypopit and pituitarian dysfunction.

They found 18% had multiple autoimmune oid glandular syndrome, pituitary antibodies were found in 9% of women with Hoshies, which is a 7-fold increase in the incidence of anti-pituitory antibodies.

So it's just another reason not to trust the TSH and mention that patients with auto-immune thieverities have a high sense of antipituitry antibodies, but Just as another little present, they also have the highest incidence of anti-type 2 deitonase antibody.

In first thought, you'd say, well, OK, that's going to reduce type 2deitonease, so there'll be less T3 and T2 teres. So their TSH would not be suppressed.

But lo and behold, it actually stimulates type two deidonases. And that was in 27% of Hashi patients. Again, showing that the Tsh is worthless in a Hashimoto's patient.

And here they said D2 activity was strikingly elevated in Hashimoto's patients, which is again the pituitary diatomace. There were 11 patients but under basically battery of tests.

In all patients the EMG showed myopathic changes and from a small amplitude and duration motor unit potentials. Oh, but they're normal, right? normal thyroid.

Here they found CFS patients of 71, only one had a level that even was barely in the normal range. And if you look, Hashimoto's patients go into anaerobic metabolism very quickly as their mitochondria are thrashed.

You can see here how they just essentially go anaerobeic very quickly because they don't have the mitochondrial function.

Treatment Approach and Closing Q&A

Very prone to SIBO, and we have a manumonics, but you gotta fix the thyroid to fix gut brain and brain-gut access. So in the study, 54% of Hashi patients were basically positive for SIBo versus 5% for controls.

If you treat the SIbo, it didn't fix a thyroid. So thyroid replacement for U-thyroid Hashimoto's. So treatment with T3 is shown to reduce thyroid deuteroid antibodies regardless of pre-treatment TSH and thyroid levels.

In study 33, U thyroid, in quotes, randomized two groups, one received thyroid the other was followed for 15 months without treatment. They basically did a follow-up with a battery of tests after 15-months.

The one's name was T4. But increase in T-4, a significant decrease in TSH, which actually we say is bad, but this was like showing they're actually their sideway was approved, the ultrasound actually improved, their goiter reduced, because in prophylactic thyroid hormone therapy can be used in patients with Hashimoto's thyroiditis even if they are youth thyroid.

Treatment with thyroxine reduces thyroid volume in non-broidered youth-thyroid children and may prevent goitre development. So you're totally justified treating these patients You actually are looking at all the studies, which we can go through.

There's so much evidence because people get scared like, oh, I heard a doctor suppressed the TSH and lost his license. I've done, let's see, over 50 expert testimony.

And every time I hear something like that, and I'm like oh my God, this doctor, that's not what they did. They kind of surgically get their license, but I was 51 in one and the one that I actually refused three times before I took it, but on thyroid, I mean.

they can't refute the evidence. So keep understanding thyroid, immune dysfunction, chronic inflammation, the pituitary is different than the rest of the body.

Again, that low TSH, high T4. Thank you for attending. Hopefully you got some out of it and I just made it hopefully understandable. And again, this wasn't the most Fun part, you're like, okay, but let's get to something that I can actually use.

And so that will be the next two lectures, really. So, which again, so part one, we kind of went through this. Part two, it's like we got to cover thyroid hormone transport.

But really then we'll get into the appropriate thyroid replacement for Hashimoto's and other conditions. We'll actually clinically treat these chronically ill or just the regular Mom wants to, needs to lose 20 pounds because she has a wedding coming up.

We'll talk about, you know, really with the peptides, immune monitor, the bioregulators, how they work and reduce biologic age. And so we'll have basically therapeutic protocols for you.

So you can go back to your office and call those patients that you thought you couldn't do anything for and you seriously. can and get them much better.

So thank you, and let's answer some questions. Before we wrap up, we want to thank our sponsor, Integrative Peptides, for supporting this episode. If you enjoyed today's conversation, visit integrativepeptides.com and use the code PODLIFE, P-O-D-L-I-F-E, For a special listener discount.

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