THYROID MIGRATING MOTOR COMPLEX-SIBO CONNECTION

What to Test & How to Treat It

SHIVAN SARNA
Bestselling author of Healing SIBO

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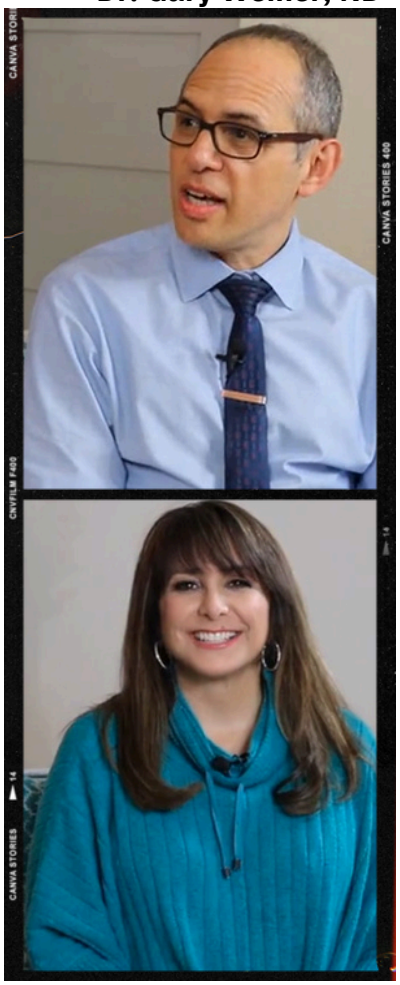
About this Masterclass

Dr. Gary Weiner, ND



Shivan Sarna

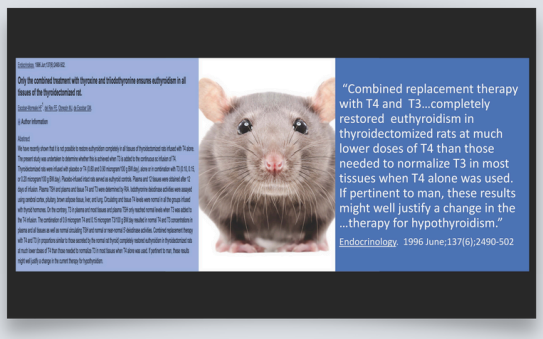
Hi everybody. I'm **Shivan Sarna**. Thank you for being here with us for the **SIBO SOS® Speaker Series**.

For this masterclass, we are joined by **Dr. Gary Weiner, ND** who, even though he has since passed on, has left us with a long legacy of clinical pearls and healing wisdom. The information he shared has been instrumental to my wellness that I wanted to continue sharing his work with the world.

This training is essential for anyone with SIBO and symptoms of hormone imbalance. What seems like unrelated conditions - sensitivity to cold, depression, fatigue - can actually be signs of a hormone imbalance that is preventing you from recovering from SIBO. Learn how to find out if you have hormone problems and how to treat them.

Here's everything you can expect to learn from Dr. Weiner's masterclass:

- ✓ How hormone imbalances (especially thyroid) can cause digestive issues
- ✓ Why typical thyroid tests given by Western Medicine doctors lead many people under-treated, poorly-treated, and under-diagnosed.
- ✓ The important connection between the thyroid and the Migrating Motor Complex.
- ✓ How hormones can help you heal when other treatments have failed.



A quick sidenote before we turn it over to Dr. Weiner. The images you'll see throughout this ebook (as demonstrated by the image on the right) are actual slides taken from his masterclass. I hope it helps solidify some of the concepts he explains in this material. That being said, let's get started!

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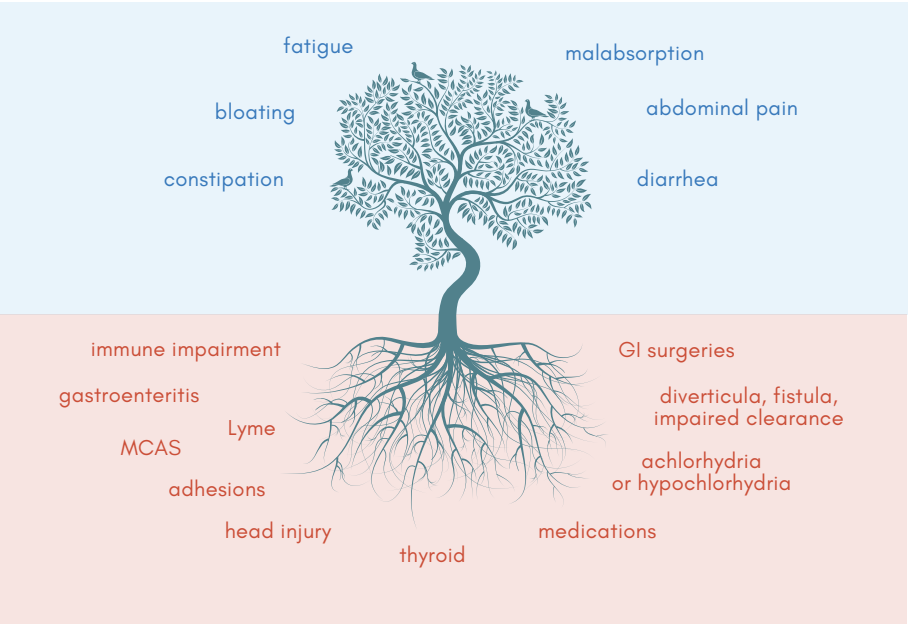
Introduction: Could Your Thyroid Be Contributing to Your Unresolved SIBO?

Dr. Gary Weiner: Alrightee... well, good afternoon. Again, it's very nice to be invited to do this. I want to speak to you about this very significant relationship, SIBO and the thyroid.

In the community, we've been looking a lot lately at the underlying causes of SIBO because we're really learning that **if only it was as easy as just giving antimicrobials and a prokinetic and be done with this—it's not**. Those of us who are getting the really difficult cases know all too well that there has to be a search for underlying causes which, unfortunately, are not always just one, but there are a lot of causes.

And so, as the curtain opens... We have here the Tree of Life with the main root of SIBO and the branch and all these symptoms that you know so well that we're always looking at in patients with SIBO.



And the important thing to know is that, underneath it, it is lots of roots with other causes of the SIBO itself.

Dr. Pimentel recently at the SIBO Symposium in Portland called SIBO a meta-phenomena, and warning—it was a doctors' conference mainly—not to be confused into thinking that it's a primary phenomena.

I think, probably, if most of you out there are patients, you may fall into that same trap of thinking of SIBO as it in itself the root, when in fact, there are deeper causes that you've been exploring with different speakers in this series that Shivan has organized.

You can see in the root there that thyroid is one of those causes, a dysfunction, or dis-thyroidism is, in fact, a risk factor for small intestinal bacterial overgrowth.

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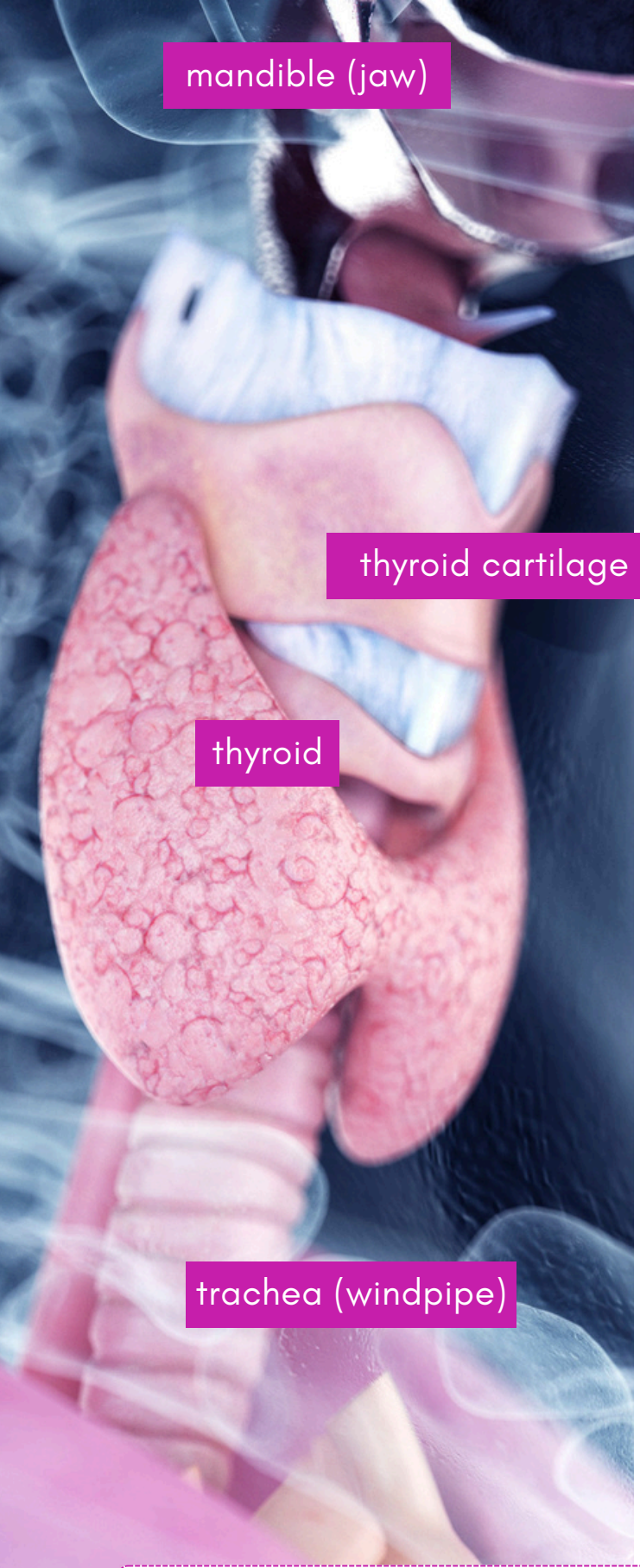

The Thyroid: The Metabolic Gas Pedal of Your Body

The **thyroid gland** itself, represented by this butterfly-shaped figure, is located in your neck between the laryngeal prominence in the top of your sternum. It's about the size of a walnut. And really, it provides and makes hormones that form **the metabolic gas pedal of your body** influencing every organ and system. And that will include of course the gastrointestinal tract.

The relationship between SIBO and the thyroid occurs in the context of the relationship that the gut has with the thyroid. In general, that begins in embryology. For those of you that don't know, embryology would be human development. As the human being is developing as an embryo, the thyroid gland is actually created in the foregut, which is to say that **the thyroid and the GI tract are developing together out of related tissues**. That's pretty interesting considering how related the thyroid is to the gut in the developed human being.

There are relationships between the gut and the thyroid through the liver. The liver is part of the digestive system. The liver is also where the thyroid metabolized significantly. Eighty percent of the thyroid metabolism occurs in the liver itself.

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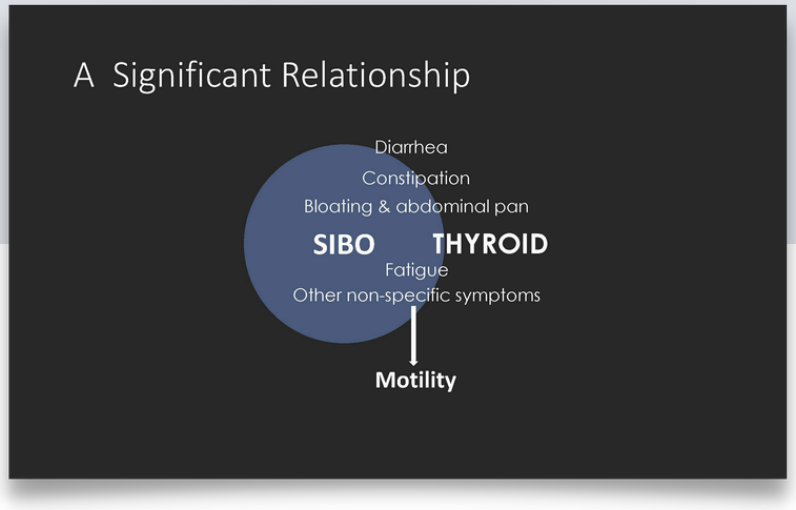
And then, there's something called **enterohepatic circulation** which has to do with how **the thyroid gland appears to secrete the hormones through the bile**. The gland makes the hormones. They circulate. They are absorbed in the liver. The liver creates bile and secrets that into the GI. And then it's reabsorbed. And this is the way—one of the ways—that the thyroid is actually regulated in the body, through the GI.

It's also responsible for that HCl and mucous secretions in the body. And the pancreatic fluids are dependent upon the thyroid hormone.

In fact, so much of thyroid metabolism is actually handled and regulated through the gastrointestinal tract. And the two go hand-in-hand.

The most significant overlap between the gut and the thyroid and SIBO and thyroid is through motility itself. We tend to talk about the migrating motor complex in relation to SIBO. We have to remember that **the thyroid is one of the key regulators of motility**.



And the reason these two circles are overlapped is because thyroid problems and GI problems focused on SIBO are often co-presenting, meaning they can create the same symptoms—diarrhea, constipation, bloating, abdominal pains, fatigue. The things that you associate with SIBO. I'm sure are also exactly, precisely the same type of symptoms that are created by dysthyroidism or abnormal metabolism. This is where there is confusion for patients, and frankly, for doctors who have to sort this out.

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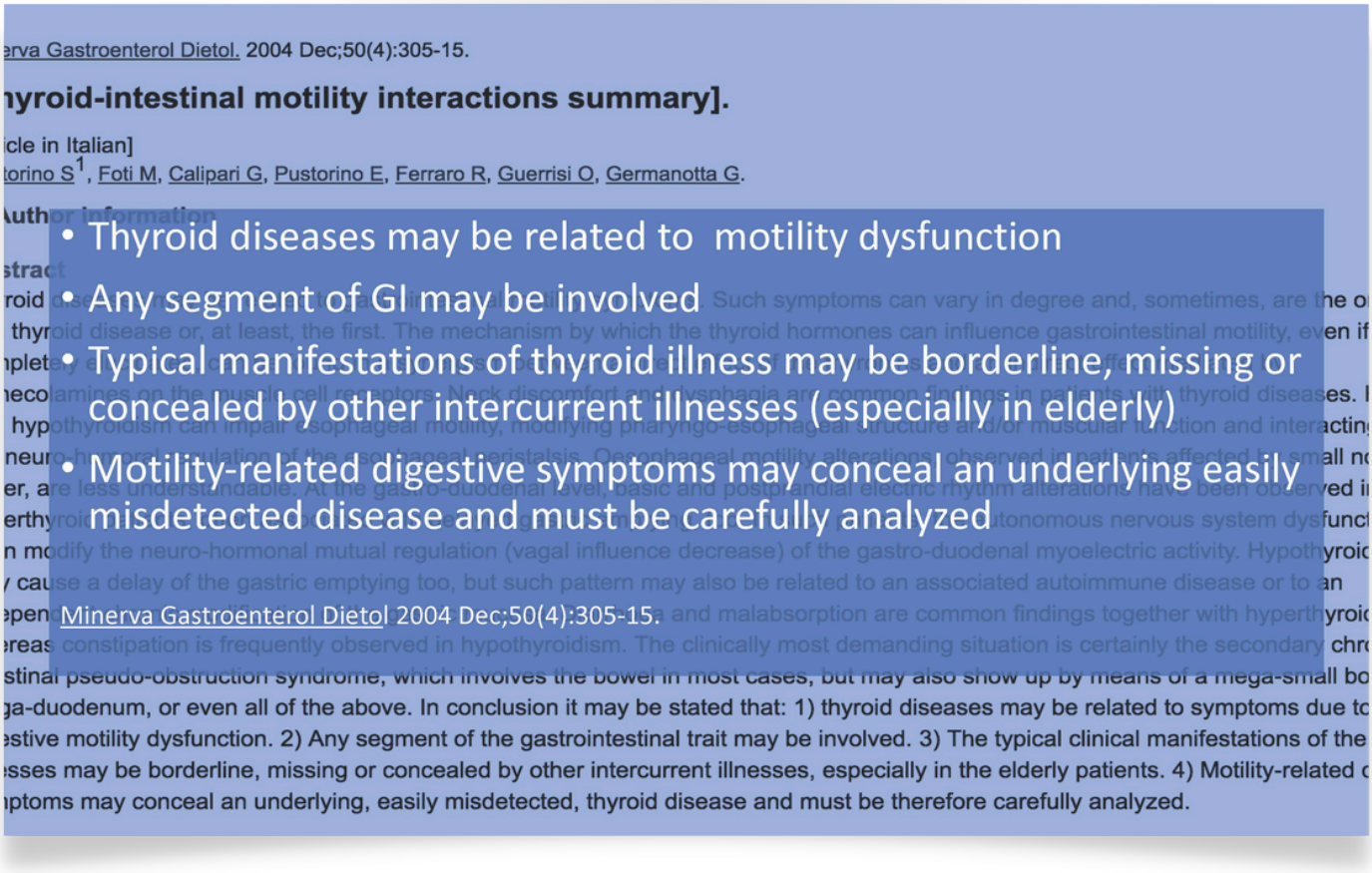
This the relationship is best summarized in this paper from 2004, that diseases may be related to motility dysfunction. Just contemplate that as you think about SIBO cases that don't resolve.



Any segment of the GI can be involved. Typical manifestations of thyroid illness may be just borderline, they may be missing, or they may be concealed by other problems like SIBO. Motility-related digestive symptoms may conceal an underlying, easily mis-detected disease or symptom pattern or meta-phenomenon like SIBO and must be carefully analyzed.

This is really important. I know it's a kind of boring slide, research summary. But the take home here for you is **SIBO and thyroid are often confused**.

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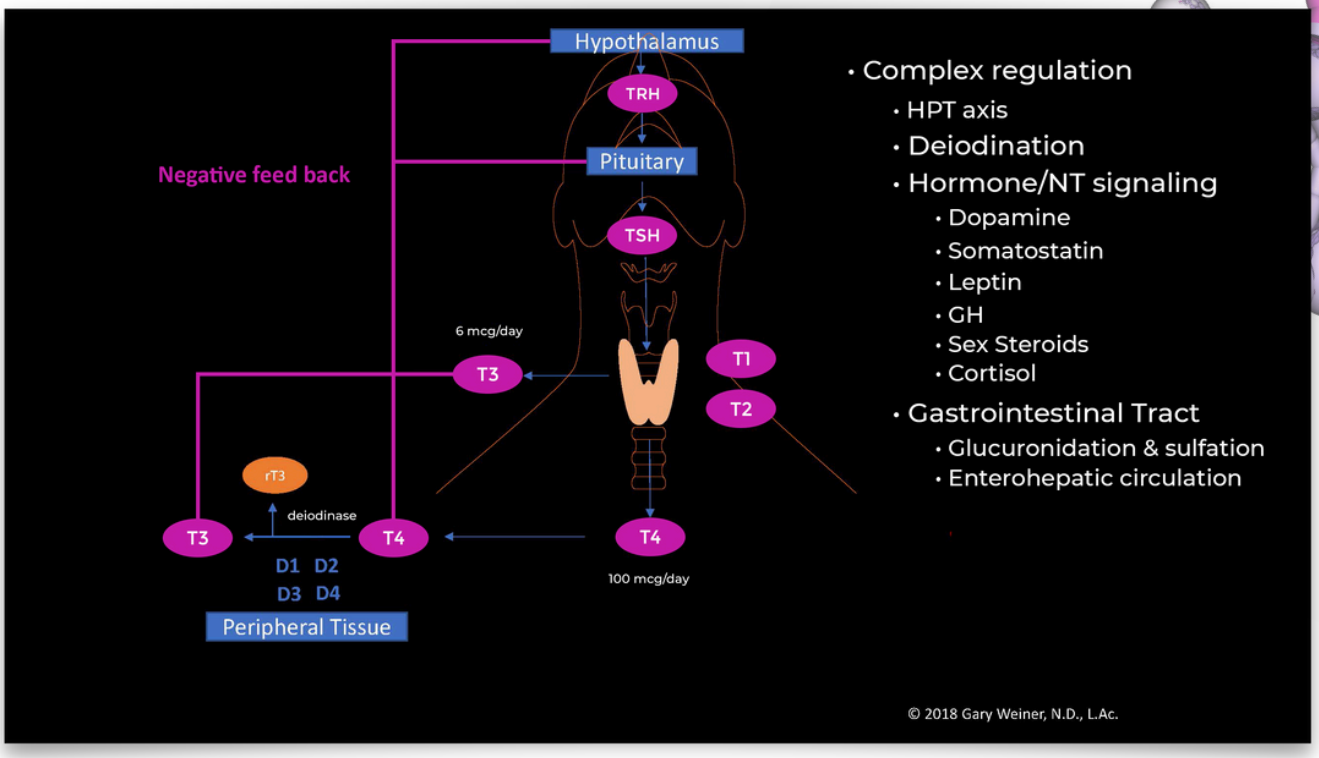
Obstacles to the Regulation of the Thyroid Gland

So, there are many obstacles to the regulation of the thyroid gland.

The thyroid gland, that butterfly, walnut-sized illustration creates basically two hormones: one is **T4 and levothyroxine**, and the other is **T3 which is liothyronine or triiodothyronine**. The **T** stands for **tyrosine**, four molecules of it or three molecules of it. And you can see that we make about 100 mcg. a day of T4 and 6 mcg. a day approximately of T3. Most of the thyroid that we make is T4 or thyroxine. And the reason for that is that we are constantly having to turn thyroxine into liothyronine or turn T4 into T3 as we need it moment by moment, tissue by tissue, everywhere in the body.



It is so fundamental to the metabolism of every tissue of every organ, not just the GI tract that there is a very complex regulatory mechanism in place. Most of this is occurring in peripheral tissues and not in the thyroid itself.

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There's also something called **T1** and **T2** which is referring to one molecule of iodine or two molecules of iodine. And there's not that much known about these. I don't have too much to say about them except they're there. They seem to be part of a cascade of reactions that turn into the more relevant hormones T3 and T4.

When I refer to the "peripheral tissues," it means "outside the thyroid" in different parts of your body. It's in the peripheral tissues where the T4 turns into the T3. It's on demand. It's like, "Uh-oh, I need T3" and boom, I make it.

And this is very important... under stress (which I'm sure many of you have a little of that in your life), something else is made instead of T3. And that's **reverse T3**.

Through complex reactions where iodine molecules are lopped off of T4 to become T3, sometimes—and that happens through something called D1, D2, D3 and D4. D standing for deiodinase, meaning lop off the iodine.

So, in the body, T4 has very little direct action on any tissue. It's called a **prohormone**. It's T3 that has the action. It's the action that's actually going to cause peristalsis in the gut. T4, it does very little. T4, you can consider it like a little transfer molecule that's moving around ready to be deiodinated, ready to have one of its iodines lopped off of it by D1, D2, D3 or D4.

I think in medical school, there's a whole day of lecture on just what I just said to you. So if you don't quite get it, don't feel bad. I think most of us didn't get it for a long time.

When you have a disease, when there's lots of mental-emotional stress, when there's lots of inflammation in the body, that's a stress. When you're eating the stuff that's not right for you, that's a stress. When someone you love talks to you the wrong way, that's a stress.

Under stress, you often don't make the T3 that you need. You can make this reverse T3 which I guess on some level is **a survival mechanism** to preserve your energy, to preserve what you need. That would be speculation on my part, but that happens.

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Now, most of this is regulated in the pituitary gland which you know is an endocrine gland, a gland in your head. And if the pituitary regulates a lot of hormones—including thyroid, adrenal gland, the sex hormones—the pituitary makes something called **TSH** which is **thyroid stimulating hormone**, and that then contacts the thyroid receptors and makes the T3 and T4—mainly T4 as we said.

Now, that TSH is regulated by a higher order of hormone in the hypothalamus. And that's called **TRH** or **thyroid regulating hormone**.

So, the secretion of your hormones in the thyroid is very, very related to your brain, your pituitary function, and your limbic systems and emotions—which is hard to talk about in terms of how we regulate that medically. But there are relationships between emotional states and the thyroid and the neurotransmitters that are very hard to delineate in terms of medical interventions.

That said, the hypothalamic-pituitary axis (HPA) is regulated through a negative feedback system. That means the more thyroid hormone you make, the less stimulating hormone is created by your brain. The less hormone you make or that is available or that is registered, the more the brain stimulates.

You could think of these stimulating hormones as asking for more hormone, asking the thyroid to make more.

But regulation is more complex than that. I think new doctors and patients reading the Internet think it's all about the HPA axis, it's all about the TSH, it's all about T3 levels and T4 levels and that whole HPA axis relationship—which I've listed as number one. It really goes further than that. And research is not quite clear on the complex mechanism of how all these regulatory systems interact.

They include the deiodination reactions—the D1 through D4—in the peripheral tissues, the dopamine and the neurotransmitters and other hormones of the body, including the sex, steroids, and cortisol. And finally, it includes the gastrointestinal tract itself.

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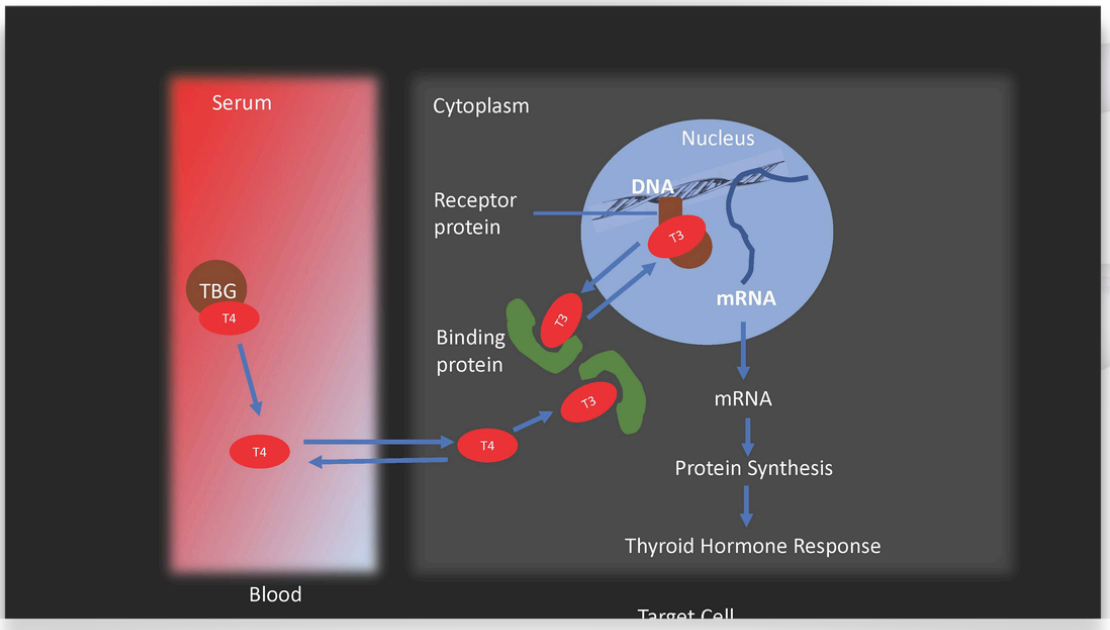
If you are ill in your gastrointestinal tract—and we have to include SIBO, the meta-phenomenon that it is that Dr. Pimentel said it was just recently—it's a sign that the GI tract, which itself regulates thyroid, may not be in order.

And that's a problem with regard to this discussion and probably some of the frank, firm, clear answers that I know many of you are seeking about, how to resolve thyroid problems in relation to SIBO. But it's a conundrum. It's a confounding variable for us. The fact that so much of the thyroid is regulated by the GI itself, so how then do we heal it—which I hope we have some insight into that by the end of our talk today.

I don't know if any of you saw Hamilton. I was lucky enough to have four tickets for my family. And I won't belabor the point. But there's a song in there called The Room Where It Happens. This is the room where it all happens for the thyroid. The red section is the blood, and the gray section is inside every cell in your body.



In the blood, the T4 is carried by a binding globulin, a binding protein, a carrier protein called **TBG**, **thyroid binding globulin**. And that travels through the blood. And it leaves the blood and goes into every cell as T4.

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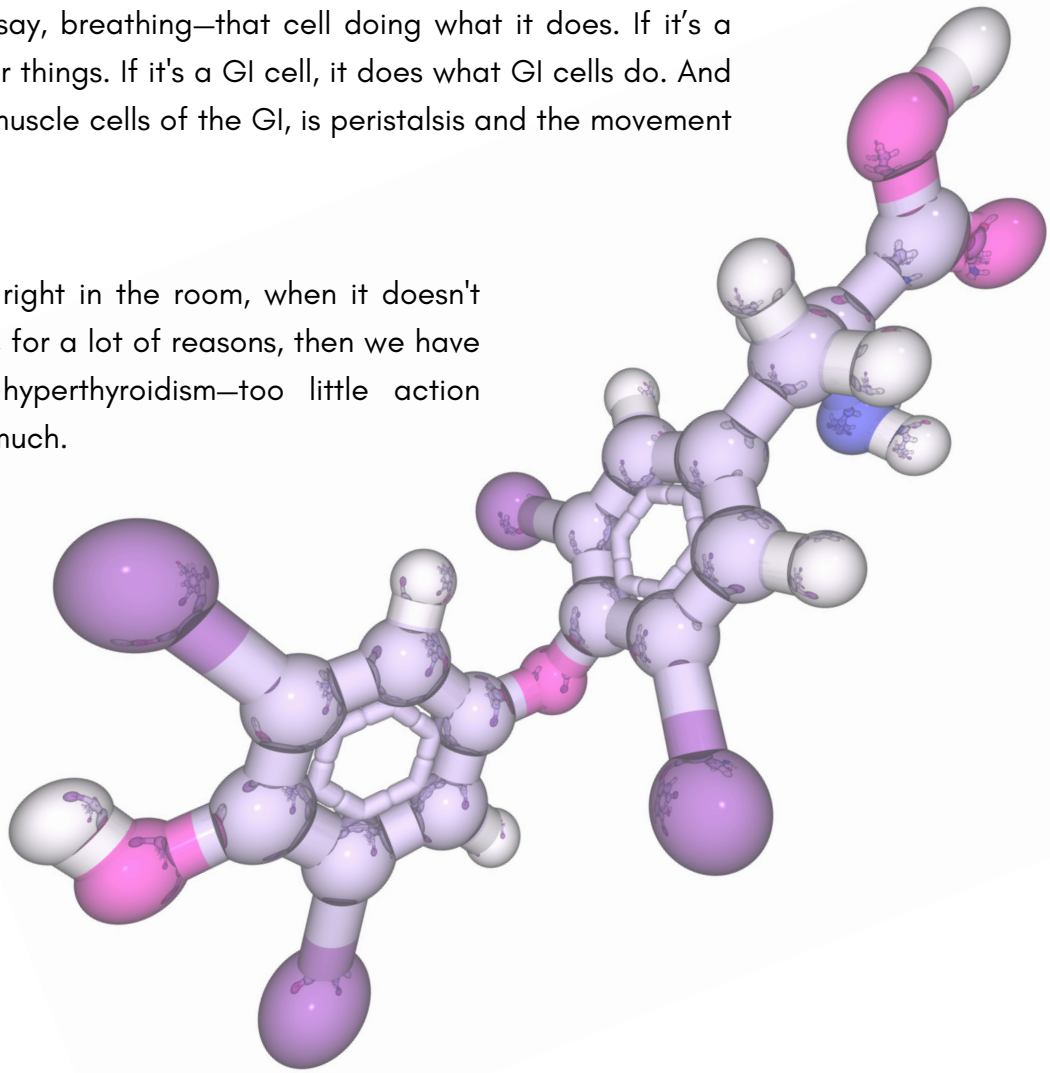
So, you see, you can take thyroid as a T4 supplement, and it has to go through the blood and get into the cell because it doesn't really become T3 until it gets in the cell and binds to a protein and goes into—that blue circle is the nucleus of the cell where the genetic material of every cell is. Inside the nucleus, that T3 binds and creates something in conjunction with the DNA called messenger RNA. And that synthesizes protein and causes the response in that cell.

So, this is happening in every cell. And that response is the action of the cell, you could say, breathing—that cell doing what it does. If it's a liver cell, it does liver things. If it's a GI cell, it does what GI cells do. And part of that, in the muscle cells of the GI, is peristalsis and the movement of the bowel.

When it doesn't go right in the room, when it doesn't happen in the room, for a lot of reasons, then we have hypothyroidism or hyperthyroidism—too little action from thyroid or too much.



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SIBO: Hyper & Hypothyroidism

In SIBO, we're mainly going to be talking about hypothyroidism (and a little bit about hypothyroidism) today. But I want you to just get an understanding of the pictures.

Constipation is mostly associated with hypothyroidism, but not exclusively. And diarrhea, more exclusively with hyperthyroidism, though not exclusively. Hyperthyroids can be constipated for other reasons; and hypothyroid can also have diarrhea for other reasons, one of them being that they are hypothyroid with SIBO.

The basic picture of the hypothyroid is one that you can see is often consistent with many of the things you see in SIBO—fatigue and brain fog, cognitive problems, coldness (not always with SIBO, but in hypothyroidism), low immune function, low neurotransmitter function, depression.

Hypothyroid folks tend to gain weight, have trouble losing weight. There's water accumulation and swelling. There's too little metabolism. And when there's too little metabolism, you have the low's.

And there's goiter. Because of the low thyroid, there is a swelling in the thyroid. That can also occur in hypothyroidism.

In hyperthyroidism, you have the opposite. You have hypermetabolism. Instead of coldness, you have too much heat in terms of the action that the thyroid gland has on muscles. There's too much action. There's too much metabolism. There is heart palpitations, something called tachycardia where the heart beats too quickly. There's too much sweat. Hypothyroid folks tend not to sweat. They often complain "I don't sweat" until menopause.

There's heat intolerance in hypo and hyperthyroidism, coldness in hypothyroidism.

And hypothyroidism, again, were going to be mainly concerned within SIBO. There's mood swings. There are memory problems. There's often a complaint of dry skin—dryness, loss of hair. There are menopausal complaints. There are menstrual complaints in premenopausal females, joint and muscle pain. High cholesterol is common.

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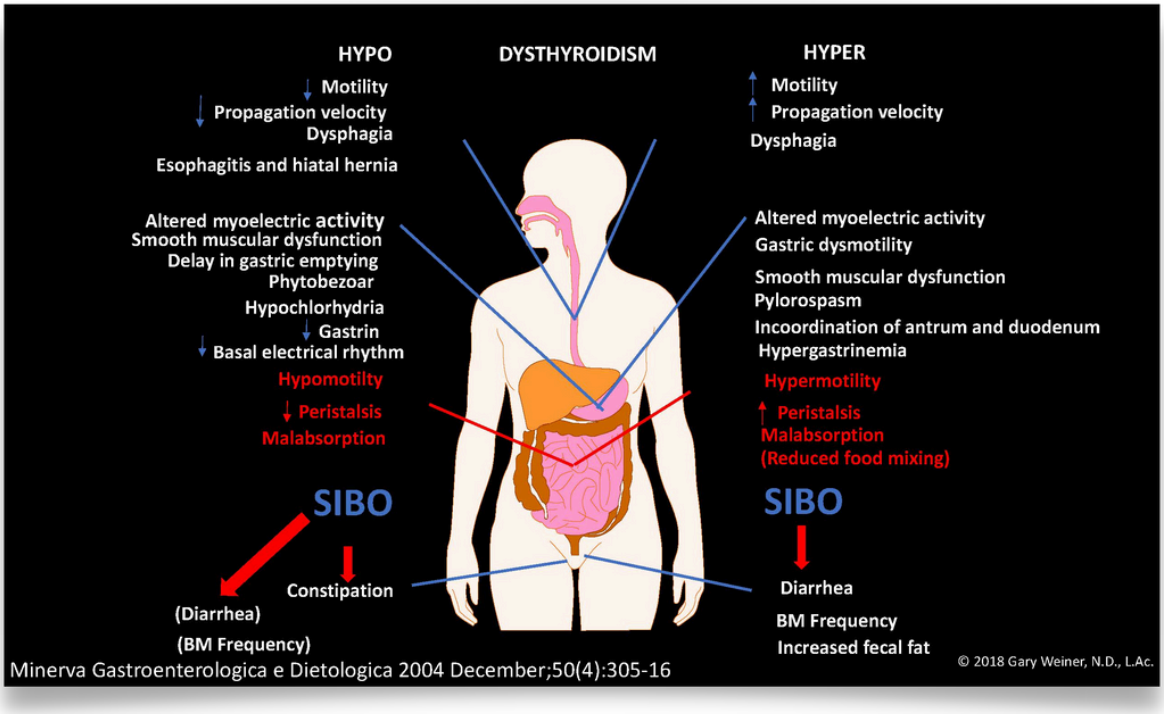
The Thyroid Hormones & the Gastrointestinal Connection

The hormones created by the thyroid gland, as I said, are affecting every system in the body, not just the GI. But the GI is the one that we're going to focus on for now because it's connected to our subject.

We said constipation is mainly associated with hypo and diarrhea with hyper. We said that the thyroid dysfunction can affect every part of the GI. It causes load motility in the esophagus, which is what we're looking at right here, itself and what's called **decreased propagation velocity**. And in hyperthyroidism, it causes **hypermotility** versus hypomotility.



In the stomach, we also see alterations. As my colleague, Dr. Sandberg-Lewis often says when we're talking about the GI, we have to look at all segments, not just the small intestine. We who focus on SIBO can fall into that trap of just looking at the small intestine when, in fact, the problem of SIBO can also often be accompanied by problems in other parts of the GI.

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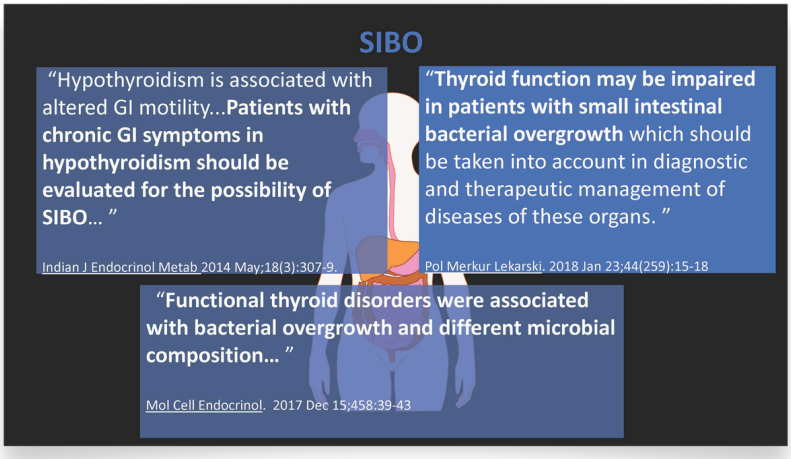
When we come to the small intestine itself, we see hypomotility in hypothyroidism. **We see decreased peristalsis in hypothyroidism. And we see increased hyperthyroidism**.

However, **both of them can cause SIBO for different reasons**. You know from previous lectures that any disturbance in motility where there is a lack of it sets up an environment where SIBO can flourish.

Likewise, in hyperthyroidism where there's too much peristalsis, the stool can move so fast that the food is improperly mixed with the components of digestion that are needed to properly digest. So there's also **malabsorption** there and SIBO.

So, while we say hypothyroidism mainly causes constipation, and hyper mainly causes diarrhea, because SIBO is so frequent in hypothyroidism, once it's there, it can easily cause diarrhea too.

So, I think all cases of SIBO with diarrhea or constipation, whether methane predominant or not, will need to be evaluated for the thyroid being a player here.



Hypothyroidism has been associated with altered GI motility from the very beginning of research on thyroid. However, after 2010, there have been numerous studies showing directly, and rather inconclusively, that GI symptoms in hypothyroidism should be evaluated for the possibility of SIBO—an important study to look at, and we will.

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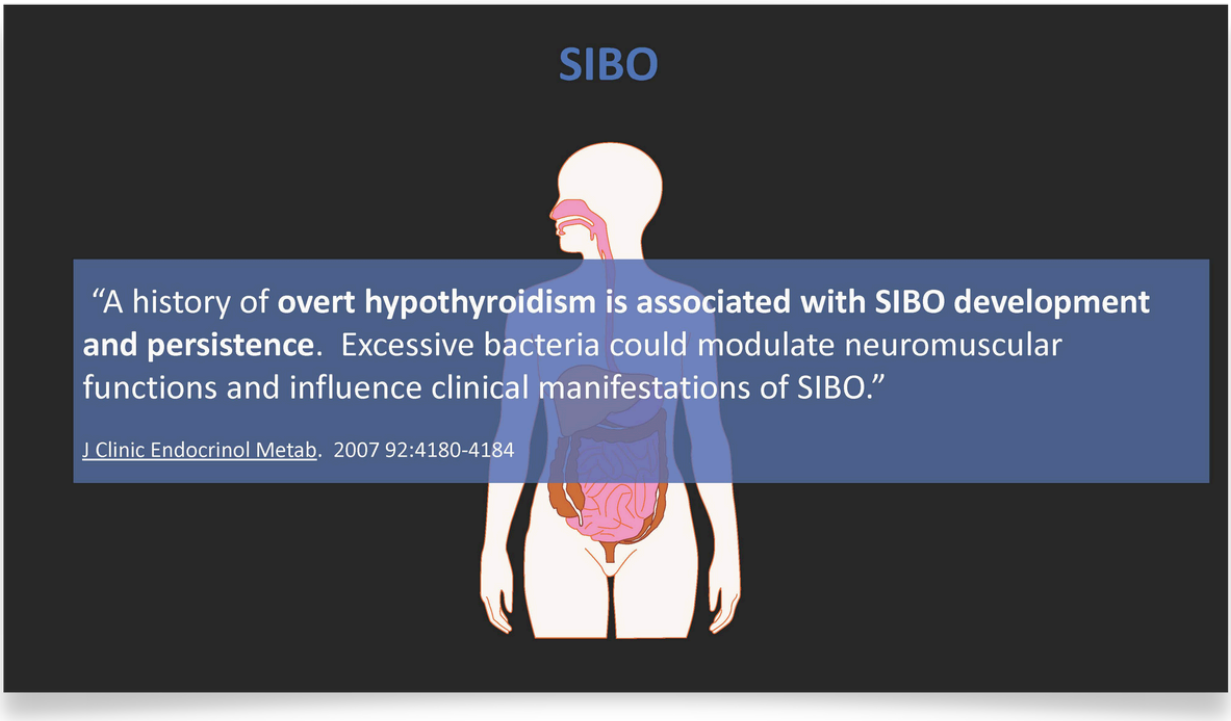
Functional thyroid disorders were associated with bacterial overgrowth and different microbial composition.

And finally, a history of overt hypothyroidism is associated with SIBO development and persistence. I want you to read that again: **"Overt hypothyroidism is associated with SIBO development and persistence."**



Most of you, I'm imagining, are watching and paying close attention to everything that Shivan's guests are saying because of persistence of SIBO. I mean if you solved the problem, you probably would not still be watching.

I shouldn't say that because they're so interesting to hear, all the different speakers. But it's that persistence that gets you doing your own research and advocating for yourself because you're finding clearly that you need different points of view.

And so, that came out as early as 2007. In other words, if I could cut to the chase on this, this research study is saying, "Hey, look at the thyroid if you have persistent SIBO," period. I mean we could end right here. I mean that's really my message to all of you.

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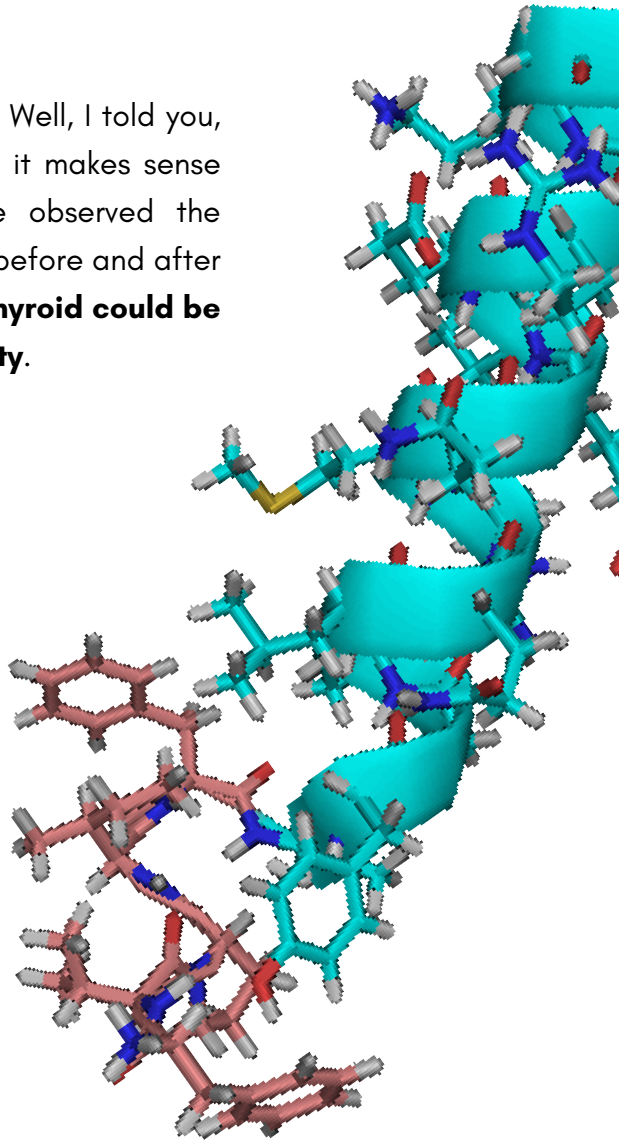
But the plot thickens, doesn't it? You remember this guy, don't you? Okay, this is not medical school, and you're not being quizzed. This is **motilin**, the 22-amino acid polypeptide hormone that plays that crucial role that Dr. Pimentel and Dr. Sandberg-Lewis and Dr. Siebecker and everybody else has been talking about that is so important to cause those cleansing waves, the house cleaning between bowel movements.

Look at this. **Motilin has been found in the human thyroid**. Well, I told you, embryologically, the thyroid was created in the foregut. So, it makes sense that they would share similar chemical components. We observed the concentration of motilin and plasma and also gastric motility before and after thyroidectomy. These results demonstrate that **motilin from thyroid could be secreted into peripheral plasma and affect gastric motility**.



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So, all we really want to say here is that the thyroid appears to affect the migrating motor complex. It's another factor.

Thyroid motilin from this very small study participates in the regulation of the MMC. All I can say to you is, due to this, your thyroid is affecting not just peristalsis.

So, let me clear that up for you in case some of you aren't tracking me on this. **Peristalsis** is the advancing of the bolus, of the food in your intestine. As you have digested it down to the small intestine, it's advancing that forward toward a bowel movement.

Thyroid affects peristalsis deeply. Thyroid also affects the MMC. That's not peristalsis. I know you were at all the other lectures. And you may have forgotten that. The migrating motor complex is the cleaning waves, the cleansing, the housekeeping between bowel movements. That's why you've been told you shouldn't eat between meals, so that housecleaning can occur. The maids won't come in the room unless the sign is out. You know that one.

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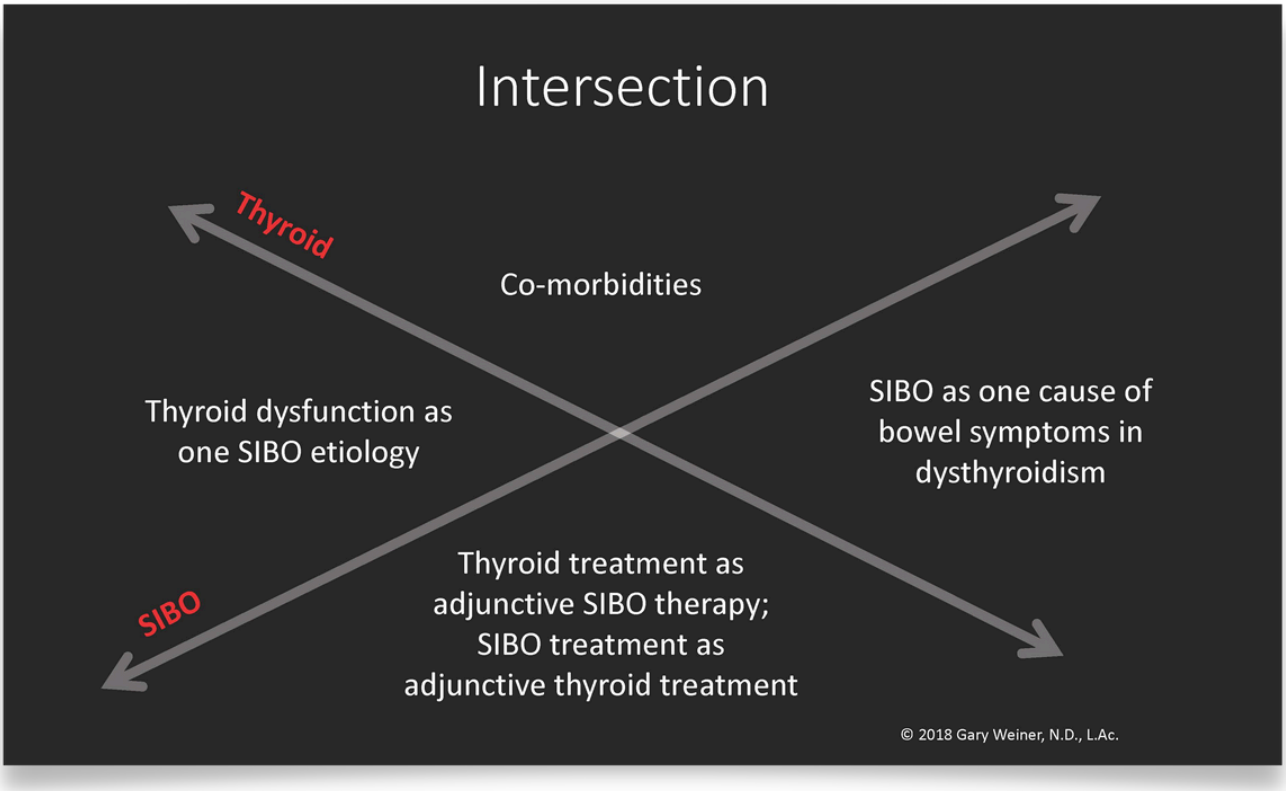
Pulling It All Together: SIBO & the Thyroid

So, pulling together what I've said so far, we have dysthyroidism and SIBO that intersect. That intersection is that they are **comorbidities**. Medical terminology, what do I mean? Thyroid problems and SIBO problems happen in the same patients. The research shows that they often do.



Thyroid dysfunction, i.e. a problem with your thyroid, is one SIBO etiology, one cause of SIBO. It's one of the risk factors. But SIBO is also one cause of the bowels symptoms in thyroid problems.

That's interesting. For those of you who may be watching who don't necessarily have a problem with SIBO anymore, or have a little, have some problems, but have a diagnosed thyroid problem, and you seem to have gotten it all zoned up in terms of your breath test is, really, what I'm saying is... **if you have dysthyroidism and bowel problems, it may be because of SIBO**. You may have gotten the numbers right on your thyroid tests, but you still have diarrhea or constipation. Then it may be your SIBO. We have research on that now. Those studies I showed you are basically demonstrating what you see on the slide right here, these other two messages.

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Shivan Sarna: So, correct me if I'm wrong, you can have the negative breath test from your SIBO, but you still have your symptoms. You feel like your SIBO has cleared, but you still have this underlying cause of the thyroid? So like you had thyroid problems and SIBO. And the thyroid may have caused the SIBO. But now that that's resolved, you still have the thyroid issues, that's just one of the symptoms that gone away?

Dr. Gary Weiner: That's one of the scenarios. In other words, yes, we have those cases. And I may have one in here, or I may not. We take care of the SIBO, and yet we still have bowel problems.

So, we're saying—to answer your question, Shivan—yes, it could be the thyroid. But in fairness to all the topics and speakers and roots of my tree illustration, it also could be something else, yeah.

Finally, thyroid treatment—we were talking before, before I took Shivan's question, about the intersections.

Thyroid treatment as an adjunctive SIBO therapy or SIBO treatment as an adjunctive thyroid treatment should be discussed, should be considered if you're not making progress in your SIBO treatment or your thyroid treatment. We need to look at that as a factor.

Let's remember that **12% of the US population, according to the American Thyroid Association, will develop a thyroid condition**, even more if you start to consider what we call subclinical thyroid, hypothyroidism, which most doctors of a conventional nature will not treat. That's when your TSH test is elevated, but your free T3 and free T4 tests are normal. Most doctors won't treat that.

And then, there are radical interpretations of TSH, which we'll talk about. But by mainstream research, **12% of the population, an estimated 20 million Americans, have some form of thyroid disease**. **Up to 60% are unaware of it** because of a lack of screening.

Women are more likely to have problems. Etiologies defined for you causes largely unknown. It's mainly said that it is autoimmune, but that is not defined as having any specific cause.

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The prevalence of thyroid dysfunction in IBS Is very high. And the study that I looked at showed **19% of IBSD patients had a thyroid problem; 27.8% of IBSC**.

IBSD for new listeners is IBS with a predominance of diarrhea. IBSC is IBS with a predominance of constipation. And IBSM, a predominance that is mixed, usually described as alternating diarrhea and constipation.

Now, what's really fascinating to me—and I hope I can fascinate you with it—is that these statistics came out of studies where SIBO wasn't even considered. And it reminded me of the good old days, the good old days when IBS was defined not by SIBO at all. This is before Dr. Pimentel had his way with the research and brought to our attention with his co-researchers that there was a problem with bacteria in IBS. It was called Rome II.

When I graduated from medical school, and IBS was what we call the purely functional bowel disorder, it reminded me (when I presented recently for the SIBO Symposium in Portland) of the cases I went back to look at before we even know that SIBO was there. But interestingly, where I used a lot of thyroid for the treatment of IBS, and lo, and behold, it often worked. And so I think we were treating SIBO through the thyroid even when we didn't know what we were doing, when were just treating the motility problems that thyroid caused.

Case Study #1

So, we'll do some really quick cases like your medical students. And I'm going to try to make them really clear and simple. For example, one of those early cases from the '90s is this 30-year old female who came to me. I was fresh out of medical school—or '92 or '98, five years out of medical school. She had four to five liquid bowel movements a day, bloating and flatulence, very tired (since she was a teenager). She had lots of ear infections until the age of 18 with a lot of antibiotics and heartburn and was on acid blockers.

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She a standard American diet. She was always cold, depressed and moody. And she was given the kind of things most people are given by other docs for bowel movement problems—Imodium, tricyclic antidepressants, fiber.

And she only had modest improvement. Okay, clear? Alright...

I did a physical exam and some things we do in the office to see if the thyroid's involved. A slow delayed Achilles' reflex is often indicative of low thyroid. The sign of her tug, that's where the eyelash is only filled in halfway where you're losing a lot of what's called the lateral part of the eye brow.

And a low body temperature. These are often nice, little signs in the office that you should be looking at the thyroid.

And she had a TSH—which I'm not showing you the ranges here. It's in the high part of the normal range. She had a normal free T4 level on the labs. And so she got—what I was doing in those days, I gave her a half a grain of **Armour Thyroid** and an **anti-inflammatory diet** which is just a whole foods diet that's non-gluten grains. That's what we sort of used to do in the '90s before we really were too stimulated by this Specific Carbohydrate Diet. The Low FODMAPS really hadn't come to the fore.

And I gave her some well-intentioned **digestive enzymes** and **probiotics** and **fiber formula**.

She came back a month later, and she tells me she had stopped the fiber formula and probiotics because they aggravated her. I didn't really realize that that's a typical reaction if you have SIBO. Many people are aggravated by probiotics and by taking fiber. So, she stopped that.

30-year old female

cc: Irregular Stools/IBS

- 4-5 liquid BM's daily
- bloating and flatulence
- chronic fatigue since adolescence
- ear infections until age 15 (repeated antibiotics)
- heartburn, OTC omeprazole
- Standard American Diet
- always cold, depression
- prescribed Imodium, tricyclics, fibers with only modest improvement

Physical Exam

- delayed Achilles reflex
- signs of Hertoghe
- body temperature 97.3

Treatment

- "Anti-inflammatory" diet
- .5 grain Armour Thyroid
- digestive enzymes
- probiotics
- fiber formula

1-month FU

- stopped the fiber formula & probiotics, d/t aggravation
- "solid stools" 3 days after starting thyroid
- decreased stool frequency
- warmer, improved mood
- TSH 1.23 uIU/mL at 6 weeks

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But after three days of taking the thyroid, she had solid stools and decreased stool frequency. And she was warmer and had an improved mood. And when I retested her TSH, it was in the lower part of the range and she was a happy patient who hadn't really even invested that much in her new diet yet.

So, there's an example for you of a case that probably had SIBO, but we didn't test in those days. And maybe the thyroid was more of a risk factor than anything else. She did very well. She did change her diet. One can really improve by going from a standard American diet to a whole foods diet even without doing, for example, a SIBO Specific Diet. Some smaller cases, SIBO Light, as I like to call it, might respond as that one did.

Case Study #2

This 28-year old female with hyperthyroidism, autoimmune hyperthyroidism, which is called Graves' disease, she had the symptoms of hyperthyroidism, of tachycardia, diarrhea, and excessive perspiration, bloating, flatulence, and heartburn.

No prior history of IBS. And she had just started the anti-thyroid medicine that is given to most hyperthyroid cases to block the thyroid. And after three months of this—and in this case, I gave her this methimazole—she had an improvement in her heart symptoms and in her perspiration, but a persistence in her GI symptoms. In other words, she still had diarrhea. It didn't improve even though everything else did.

So, if we look at her labs, we can see a TSH—and I don't want to bog down this presentation by going too much into the hyperthyroidism labs, but just to say that...

28-year old female

cc: diarrhea/Graves disease

- tachycardia, diarrhea, excessive perspiration
- bloating, flatulence, and heartburn
- no prior history of IBS
- started methimazole
- 3 mos: >tachycardia & perspiration, but persistence of GI sx

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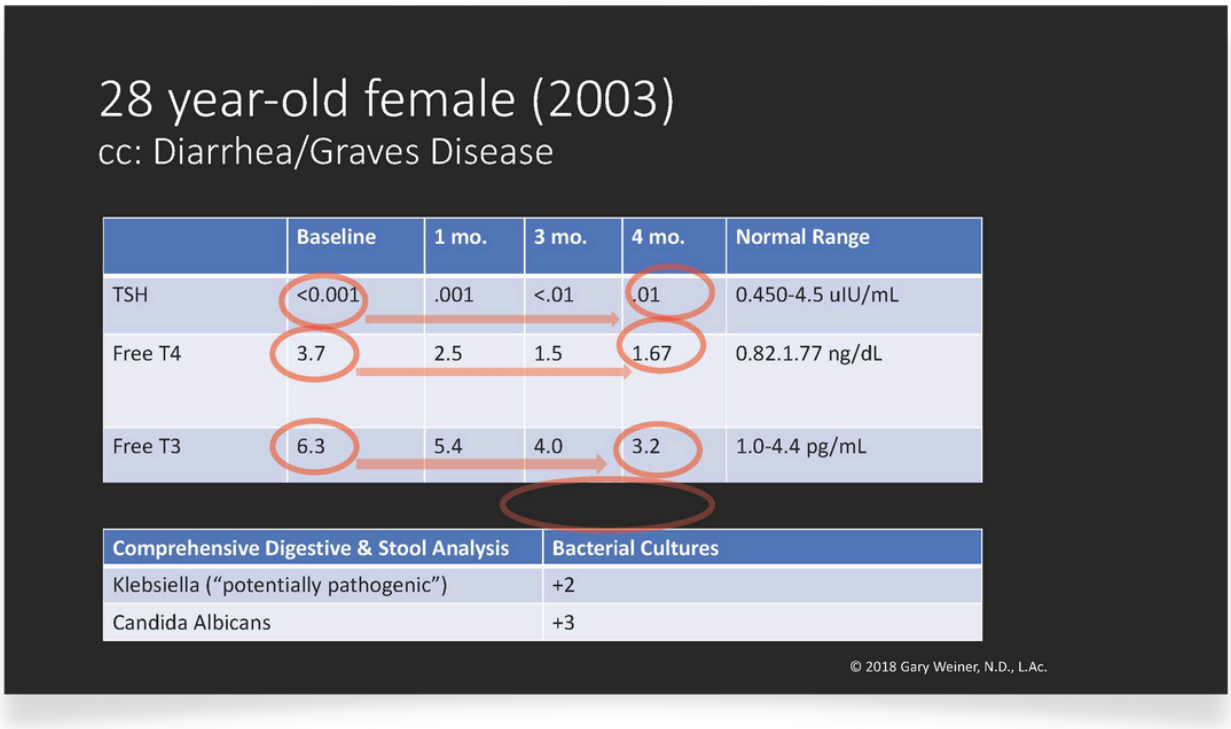

In hyperthyroidism, the TSH is very low. And as the thyroid repairs, it increases. So, we can see that it got better. And the circulating level of T4 went from being high to being normal. And the circulating level of T3 went from high to being normal. And the bowel symptoms did not change.

Now, I hope I'm not losing anybody. The bowel symptoms didn't change, but things were getting better.

And hyperthyroidism, by the way, it takes a long time even with the medication that I prescribed here. It can take months and months. And as long as the patient is getting better with the very, very dangerous symptoms of a heart that's beating out of control, then you're okay too as long as you're moving in the right direction.

What's of interest here for our SIBO audience is that the bowel symptoms didn't change, that hyperthyroidism cause SIBO likely, but treating the thyroid itself was not enough, that SIBO persisted—remember that word persistence—the thyroid problem can lead to SIBO, but the treatment of the thyroid symptom doesn't always resolve the SIBO without SIBO treatment.

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In this case, we weren't doing SIBO yet. But in those days, I was doing stool analysis. And here, we had **Klebsiella** and **Candida albicans** which were two organisms that can show up. I often see those when I do stool testing in cases with SIBO. In other words, many patients with SIBO also have dysbiosis of the large intestine where something can be cultured—not always, but often enough.

So, in the patient's treatment plan, what I want to point out to you is, in addition to doing the regular things I would do for a hyperthyroid case (including reducing stress, doing some acupuncture, getting the patient on a better diet)—

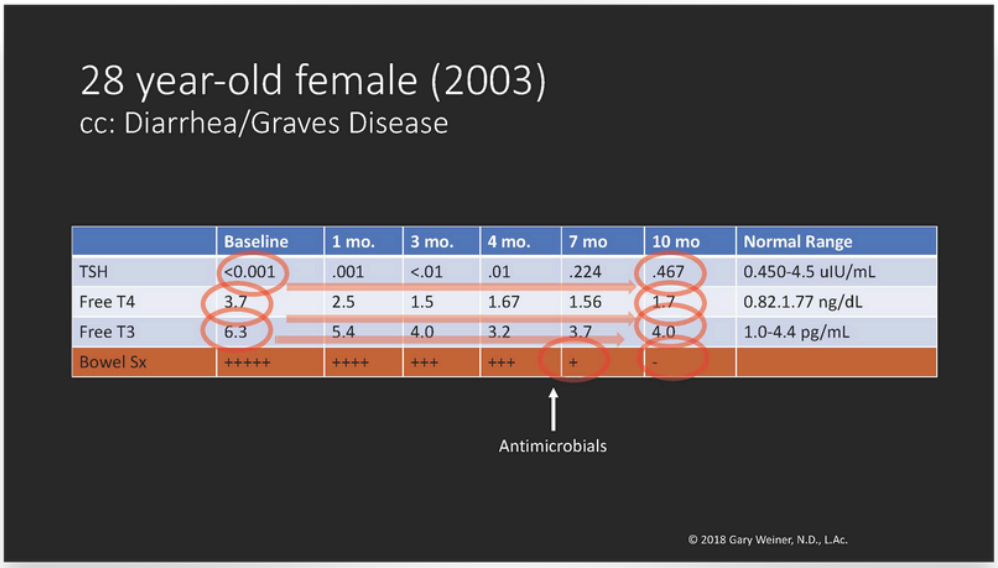
Remember, this is 2003. We're still not really into SIBO yet. I'd give them a couple of tinctures—one for their thyroid to help it regulate, and another for the adrenal gland to help it regulate. And for that bacteria that we found, and yeast, I gave berberine and oregano, two friends you probably know from the herbal treatments of SIBO.

Treatment

- Stress reduction, counseling, acupuncture
- "Anti-inflammatory" diet
- **Tincture #1:** Lycopus, Leonurus, Melissa, Passiflora, Selenicerus, Lavender essential oil
- Tincture #2: holy basil, Ashwagandha, licorice, Cordyceps mushroom
- berberine 500mg 3x/day
- oregano oil 50mg 3x/day

1-month FU

- dramatic improvement in diarrhea and bloating



And lo, and behold, one month later... dramatic improvement in the diarrhea and the bloating.

So, the reason I'm going back, and I'm hoping you're following me in doing these old cases, is to show you the role that this has probably always been playing even before we were measuring SIBO in that when I treated with antibacterials, it probably was treating the patient's SIBO.

You can see here where we look at 10 months of labs that, after continuation of this plan, the lab values all resolved. And the bowel symptoms also resolved. And that big change came when we gave antimicrobials.

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Research Studies

So, if we now move to the research on SIBO and thyroid and what we now know, we start with a 2017 study with 50 hypothyroid recruits and 40 healthy controls. Hypothyroid patients were given T4 only medication which is known as **levothyroxine** or **Synthroid**, and they became normal—they had normal thyroid tests—before they're tested SIBO with GBT's which means "glucose breath test" (which were used, and still are, by some doctors to test for SIBO).

Twenty seven of the 50 patients were positive for SIBO by glucose breath tests. I'm sure, with lactulose, it would be a higher number.

They got seven days of rifaximin and had a decontamination rate of 78.4%.

The conclusions of the study were that once SIBO is established in hypothyroidism, or in the hypothyroidism phase, it does not clear spontaneously even when euthyroidism is achieved.

The conclusions of the study were that once SIBO is established in hypothyroidism, or in the hypothyroidism phase, it does not clear spontaneously even when euthyroidism is achieved.

SIBO development and persistence of excessive bacteria might modulate neuromuscular function and clinical manifestations. And overt hypothyroidism is indeed a risk factor for SIBO.

This is big stuff for, I think, the SIBO community.

If we go to the 2018 Polish study, researchers recruited 34 SIBO diarrhea patients, 30 constipation patients, and 30 controls.

The thyroid hormone levels were similar in both SIBO-D and the control patients. But patients with constipation-predominant SIBO had thyroid panels characteristic of hypothyroidism.

The TSH was measured, the free T3 and the free T4. And an antibody to thyroid which is

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often measured to determine if Hashimoto's thyroiditis which is the main cause of hypothyroidism was present. The antibodies to TPO or thyroid peroxidase (which is one of several measures for Hashimoto's disease), the main cause of hypothyroidism, was significantly higher in SIBO groups compared to the control group.

So, this really is to say that SIBO patients have a susceptibility or a co-morbidity, they are likely to have, or more likely to have, autoimmune thyroid illness than non-SIBO patients.

Read: SIBO patients with problems with your SIBO, be tested and understand if you are a Hashimoto's patient, if you need the attention and care to an autoimmune disease, as it were—though a very survivable, I must say

Conclusion of this study—and mine, and I hope yours—SIBO should be taken into account in the management of thyroid diseases.

Okay, another study, 2017, this was a retrospective German study with 1809 patients—the purpose of which, to assess for risk factors for SIBO. This is fascinating, really fascinating. In fact, Shivan got wind of this when I said I was going to talk about SIBO. And she said—this is how ahead of the game she is—she said,. "Are you going to talk about that study with levothyroxine?" and I said, "You bet!"

They studied four categories of risk factors—impaired gastric acid barrier (that's not enough HCl. And by the way, thyroid is one of the co-regulators as I said in the beginning of my lecture when you were probably just getting comfortable and might not have really heard that), they studied impaired gastric barrier, impaired intestinal clearance, immunosuppression, and miscellaneous factors including thyroid gland variables.

Conclusion: the most important contributor out of all of those to SIBO were levothyroxine therapy and hypothyroidism

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So, hypothyroidism in this study, I actually don't have an intrinsic feeling for my own practice that thyroid is bigger than any other factor that other docs are talking about. But in this study, hypothyroidism was bigger than any of these other factors and bigger than hypothyroidism was a patient who was on the main therapy for hypothyroidism already. That's pretty strange.

In other words, if you're on T4 medication only, you are at risk for SIBO.

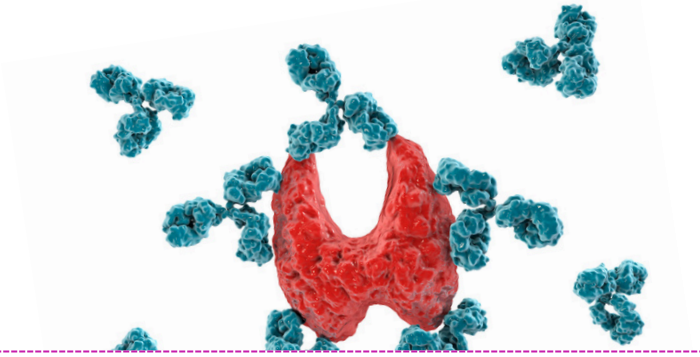
Of course, unfortunately, the study had no insight into why that was.

This is an abstract of that study: "Levothyroxine therapy and impaired clearance are the strongest contributors to SIBO and the results. The most important contributors for the development of SIBO, in ascending order, are immunosuppression, impairment of intestinal clearance, and levothyroxine use."

So, the most common cause of hypothyroidism is indeed autoimmune factors, which get called **Hashimoto's disease**. And the cause of Hashimoto's disease in traditional literature is quite unknown, quite just an object of speculation, of what those factors are.

Unfortunately, in my community of integrative physicians who work the gray areas of the research, this comes down to a lot of detoxification of the body and looking at where there is toxicity in the body to try to get those factors down and to get the body balanced and get the inflammatory cycle balanced. And then, we can start to make some headway with treating Hashimoto's in a fundamental way.

But back to this study where levothyroxine is a risk factor, this really raises the big question in our last section of the presentation, before we sum up, which is how do we treat the thyroid.



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Thyroid Therapy as an Adjunctive SIBO Treatment

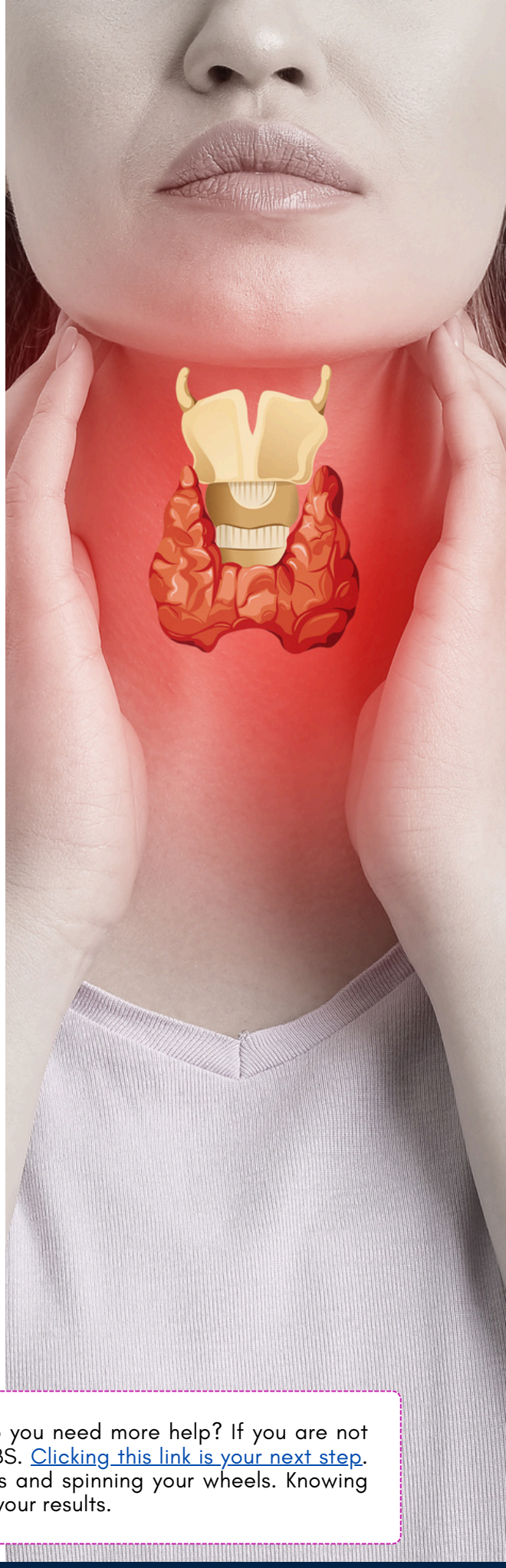
How do we use thyroid treatment as an adjunctive SIBO therapy? You're all here, what's the hook of you being here in addition to just working on matters of health is our common cause in SIBO. **And what role does thyroid treatment play if the very treatment that most people are on is a risk factor for SIBO?**

I mean, to me, that's a mind blower and a very difficult question. So, it behooves us to go over what the questions really are for you—and that is **1)** is the thyroid one of the causes of your SIBO? **2)** If you have been diagnosed with hypothyroidism, is it being treated optimally? Does that T3 medication need to be considered? There are T4 medications. There are medications that are mixtures of T4 and T3.

3) Could you have a subclinical or sub-laboratory hypothyroidism, meaning could you require treatment even though, clinically, you don't have hypothyroidism?

4) Could subclinical hyperthyroidism be related to your SIBO? **5)** Could the thyroid problem be an obstacle to the effective treatment that you have received already or the well-intentioned treatment that you've received that has been only partially effective or were not effective?

So, when we talk about the standard of care—and this is complex material, I warn you—the main controversies in thyroid care are around these areas:



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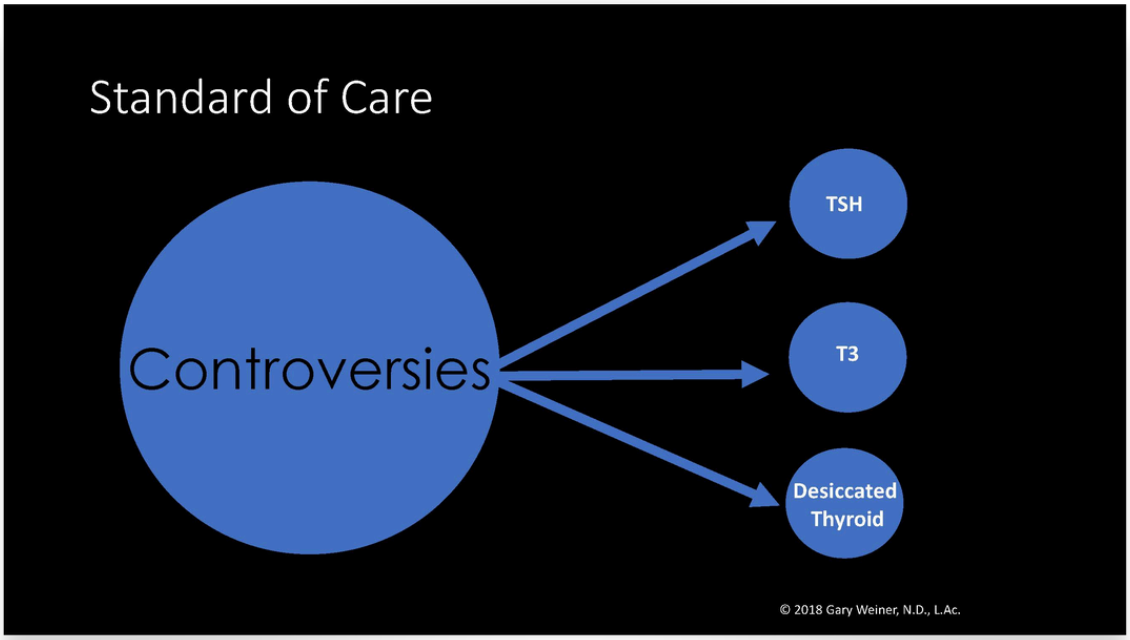
Thyroid Therapy: Why the Standard of Care Doesn't Work

So, when we talk about the standard of care—and this is complex material, I warn you—the main controversies in thyroid care are around these areas:



One, how to read the TSH? Do we use the range? Do we use subclinical? Or do we go rogue and not look at labs at all?

T3, should it be given? Should it be included? Should it not be included? What kind?

The use of desiccated thyroid, this pig thyroid, the glandular medicine that has been with us since the 20th century, early 20th century? Dessicated thyroid, it goes under brand names like Armour Thyroid, Nature Throid, WP Thyroid.

So, the treatment of choice—and now I'm talking standard of care. I'm a physician, I am talking from the largest number of physicians' point of view. But because I am an integrative physician and a naturopathic physician, we will also talk about what we do outside of the conventional standard of care. So you've got to keep two heads on here.

The standard of care in traditional conventional medicine is the very risk factor for SIBO. I smile when I say it because it's a real surprise when we look at that study, **Levothyroxine T4 therapy**.

And the routine use of combined T3 and T4 is ill-advised in standard literature and in standard parlance through the American Thyroid Association publications.

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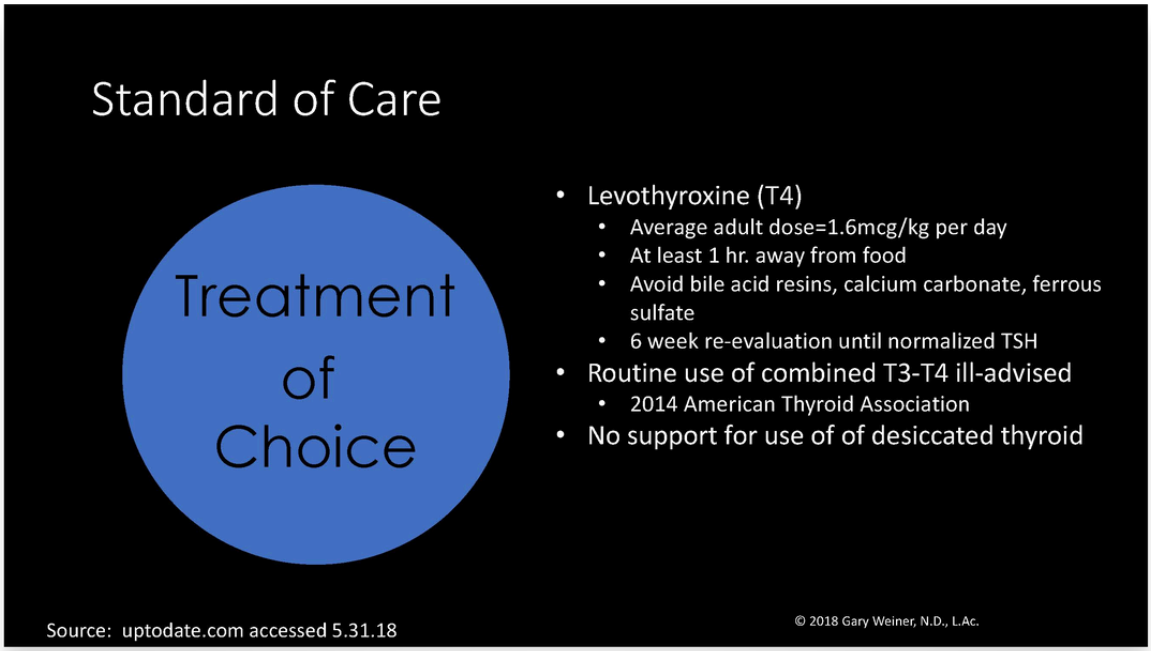
No support at all for the use of desiccated thyroid, which most naturopathic and holistic physicians tend to be open to, if they do not prefer desiccated thyroid—or at least a combination of T3 and T4 where indicated.



Therapeutic goal... we're back now to the standard of care, the way T4 is used. It is used until the patient has an amelioration—that means improvement in medical parlance—and a normalization of the TSH, a reduction of goiter when there is goiter, and an avoidance of overdosing of thyroid, thyrotoxicosis (the medical term).

So, really, let me break this down for you. **Standard of care is to reduce your symptoms, but most of us see patients who are on standard of care and still do have symptoms.** And I'm sure from the look of many of the questions I saw in my advanced peek (that Shivan was gracious enough to share with me), I could see that feeling that T4 wasn't enough in many questionnaires.

Normalization of TSH is often reached, but it's not a normalization of symptoms.

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The historical context of this is that, in the 20th century, desiccated thyroid was given. And desiccated thyroid contains both T3 and T4. It used to be dosed not by labs at all in the 20th century, early 20th century through mid, even through the '70s, but by basal metabolic rates and a serum iodine level and normalization of symptoms. But in truth, a lot of people were overdosed and thyrotoxicosis was common, meaning doctors made many patients hypothyroid temporarily in order to try to cure their low thyroid function by dosing by symptoms. I hope everybody is following me there.

It was in the 1970s that TSH was invented—not invented, was developed. The physiology and biochemistry and laboratory diagnostics were evolved enough to find this and use it. And the entire research profession was involved in identifying these deiodinase-mediated conversion reactions. And through that, justification for a T4 monotherapy (or just using T4 or levothyroxine) became the standard of care as the best to stabilize and normalize a TSH. It became the way it's done. And **T3 and T4 combinations were excluded entirely**. And that still, to this day, dominates (although there is more openness in some environments).

If we look at the research on T4 monotherapy, limited studies in it, but this one from the New England Journal of Medicine in 1971, **T4 monotherapy fails to restore hypothyroidism markers at TSH-normalizing doses**.

So, in many cases, it did restore hypothyroidism. But there were enough cases worth publishing where it didn't. And frankly, these are probably many folks watching today and the people that come to the doctors often featured on SIBO SOS®, cases where T4 monotherapy is failing to restore the symptoms of hypothyroidism.

T4-treated patients with normal TSH, in those patients, **the basal metabolic rate remained 10% less than controls even after three months of treatment of levothyroxine**.

I hope you're starting to get the hint of where I'm going with this, which is that, sometimes, it may be that T3 is what is needed in some of the cases where the bowel is still having symptoms and where the patient is still having symptoms of hypothyroidism.

In another New England Journal article study in 1965, doses of T4 that normalized the BMR actually suppress the TSH and cause iatrogenic thyrotoxicosis, meaning they caused hyperthyroidism—which is dangerous.

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Higher serum T4 levels will impair—and this is the one I think sheds the most light on the levothyroxine study. I'm surprised that this was not mentioned. Higher serum T4 levels will impair systemic T3 production via downregulation of a deiodinase pathway—I could spend an hour on this study alone, meaning...

If you just dose T4, you can impair T3, which is the active form, which is needed for peristalsis and for stimulating the migrating motor complex to sort of move that into the SIBO arguments, to our argument for SIBO.

Nonetheless, doctors are using free T3 and free T4 as I do (and as I'm sure many of the doctors in our panel does). And the American literature has kind of co-opted or imported the European Thyroid Association standards which have outlined a way in which T3 therapy should be used.

And so candidates for combined therapy are those who've had a thyroidectomy (meaning who had their thyroid glands removed. And many people with hyperthyroidism have their thyroid glands removed, and then become hypothyroid), post-ablation therapy (ablation is where part of the thyroid gland in hypothyroidism is destroyed to decrease the thyroid levels), or a serum T3 level is below the lower end of the T3 reference range.

So, this is worthy of you noting for those of you who are on T3 therapies already or on T4 therapies. Many doctors, including myself, will look at the T3 reference range and try to get the thyroid function where the T3 is in the upper part of the range.

Dosing T4 and T3 should attempt to mimic normal physiology. And that is a ratio of 13:1 to 16:1 while maintaining a normal TSH.

This is pretty strict. And I would say, for most doctors who use desiccated thyroid, this does not meet this requirement. In other words, dessicated thyroid has a ratio of about 4:1 and not 13:1. And if we were to meet these standards, we would not be helping the tremendous number of people that we do help with desiccated thyroid.

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Dose of T3 should be divided into morning and afternoon doses. And that's because T3 doesn't last very long in the body.

So, those are the European standards, in addition to monitoring the TSH every six weeks. But monitoring T3 levels is not recommended.

As we move forward here, desiccated thyroid which contains both T3 and T4 in a single grain of Armour or NatureThroid, we have 38 mcg. of T4 and 9 mcg. of T3.

In the conventional research, if we look in PubMed or any of the online resources, we see that **desiccated thyroid extract did not result in significant improvement in the quality of life by the standards of the study**. **But more than half the patients expressed a preference for it**. Most of my patients do prefer it. It does cause significant improvement. But in this study, of which there were other variables to consider, it didn't show. This study is used as an argument against the desiccated thyroid rather than for it.

This is the range of TSH. You can see, it's about five points. Many doctors will not treat the thyroid if the TSH is in this range. Many doctors will still not treat the thyroid even if it's five points above this range if the free T3 and free T4 levels are normal.

Is this right? Well, there's conflict in the research. **More than 95% of normal individuals have TSH below 2.5** according to this study. The remainder with higher values are outliers, most of whom are likely to have underlying Hashimoto's thyroiditis or other causes.

This is a mainstream journal. And there are many, many studies like this which are used to refute the standard of care, such as this one arguing that therapeutic interventions should not be considered when levels are below the long-accepted threshold.

Yet even though various large surveys defined a much narrower range of TSH between 0.3 and 2.5, I tend to look at that range. I tend to look when I look at a patient's TSH that **if they are below 2.5, and they have symptoms of hypothyroidism and various medical problems and depending on their history of using thyroid, I will consider treatment** despite the fact that they are outside of the standard levels.

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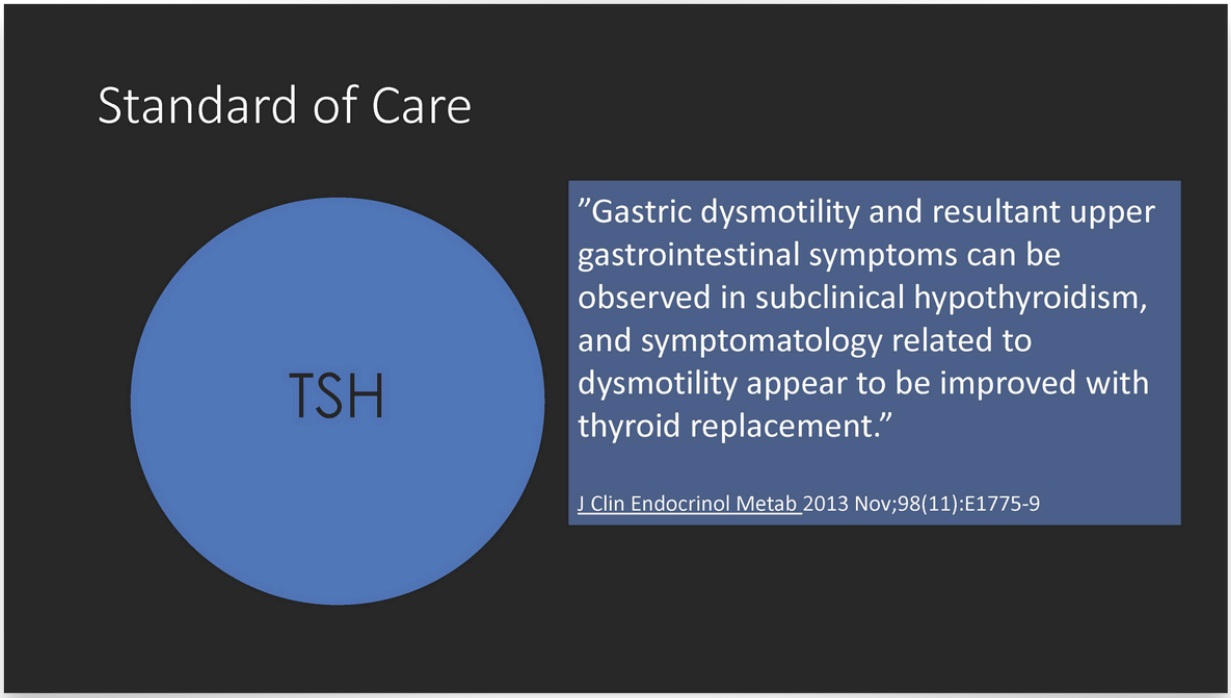

Dysmotility symptoms were observed in subclinical hypothyroidism and symptomatology related to dysmotility, i.e. SIBO, appeared to be improved with thyroid replacement. This was 2013.

So, we really should be looking at subclinical hypothyroidism. In this 2015 retrospective study of 399 subjects, it was concluded re-evaluation of the reference systems on a broader scale is necessary and that subclinical thyroid functions are being missed in a tremendous number of patients.

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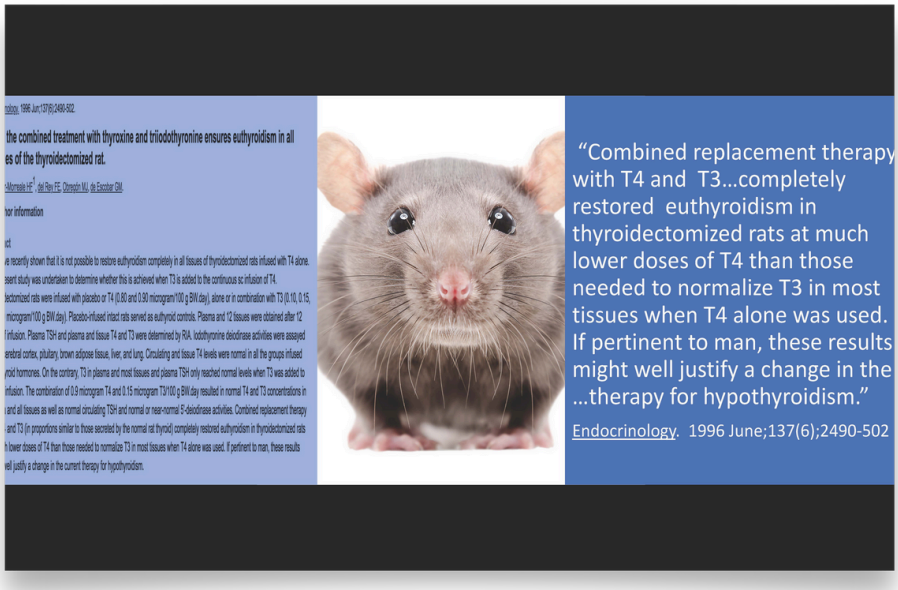
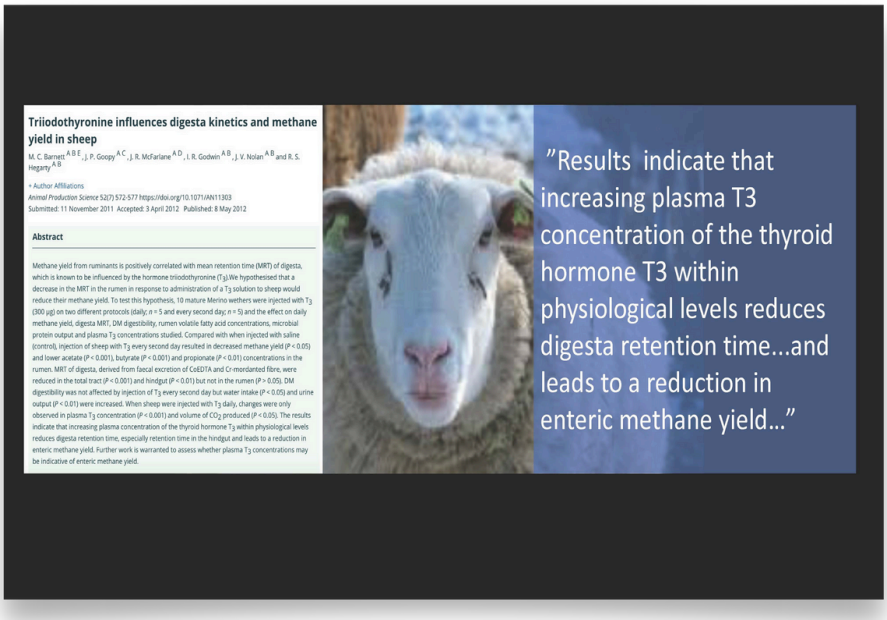
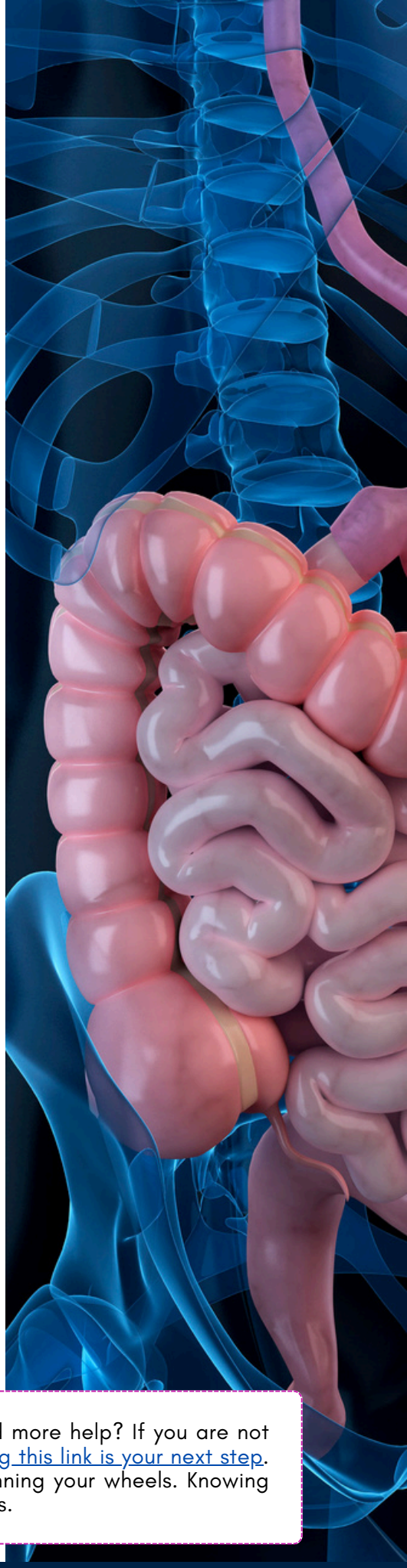


This is a rat. A lot of our studies are done with rats. Okay! What does this mean? More optimal achievement of normal thyroid when T3 and T4 were used in the rats.



Here's a sheep. As you know, these animals make a lot of methane. This is a veterinary study showing that the use of T3 influenced digestive kinetics—prokinetics, are you getting the connection—in the methane yield in sheep. Results indicate that increasing plasma T3 concentration of the thyroid hormone within physiological levels reduces digestive retention time and leads to a reduction in enteric methane.



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I like this study because so many of our difficult cases are methane cases, like this 63-year old female with constipation, bloating, abdominal pain in a setting of Hashimoto's. She came to me on Levothyroxine, 50 mcg. For 10 years, she was on 50 mcg. a day of Levothyroxine.

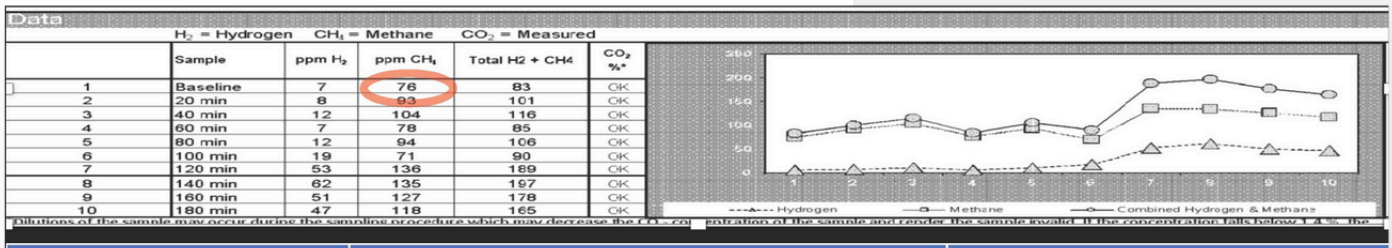
Look at her methane in my first SIBO test, 76 (image below). And look at the methane downstream from that. It's a carpet of methane throughout the entire small intestine.

If you look at her thyroid function tests, they're all within normal limits. What do you do with this?

63-year old female
cc: Constipation

Symptoms

- constipation
- bloating
- abdominal pain in setting of Hashimoto's
- on Levothyroxine 50mcg/day for 10 years



Data

H_2 = Hydrogen CH_4 = Methane CO_2 = Measured

	Sample	ppm H_2	ppm CH_4	Total H2 + CH4	CO_2 %
1	Baseline	7	76	83	OK
2	20 min	8	93	101	OK
3	40 min	12	104	116	OK
4	60 min	7	78	85	OK
5	80 min	12	94	106	OK
6	100 min	19	71	90	OK
7	120 min	53	136	189	OK
8	140 min	62	135	197	OK
9	160 min	51	127	178	OK
10	180 min	47	118	165	OK

Thyroid Lab	Finding	Normal Range
TSH	1.83	0.450-4.5 uIU/mL
FT4	1.0 WNL	0.82-1.77 ng/dL
FT3	2.0	1.0-4.4 pg/mL

So, she got **rifaximin** and **neomycin**, followed by **low dose erythromycin**.

Treatment

- rifaximin 550mg TID
- neomycin 500bid
- followed by LDE 50mg. hs

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Summary Report of Hydrogen & Methane Breath Analysis with Carbon Dioxide Correction

Gasses Analyzed:	Patient Result	Expected (Small Intestine only)
Increase in Hydrogen (H_2) Level:	83 ppm (high)	< 20 ppm
Increase in Methane (CH_4) Level:	36 ppm	< 12 ppm (< 3 ppm[2])
Increase in Combined H_2 & CH_4 Levels:	119 ppm (high)	< 15 ppm
Analysis of the above data suggests:	Data suggests small intestinal bacterial overgrowth[2]	

Number	Expected Location	Collection Interval	ppm H_2	ppm CH_4	Combined	Sample Normalization[1] ppm CO_2	f CO_2
1	Small Intestine	Baseline	6	20	26	3.9	1.41
2		20 Min.	7	37	44	3.7	1.48
3		40 Min.	10	43	53	3.2	1.71
4		60 Min.	12	43	55	3.2	1.71
5		80 Min.	56	50	106	3.2	1.71
6		100 Min.	89	56	145	3.9	1.41
7	Transition	120 Min.	32	17	49	2.9	1.89
8	Large Intestine	140 Min.	84	29	113	1.5	3.66
9		160 Min.	107	44	151	3.9	1.41
10		180 Min.	85	30	115	2.4	2.29

And you see the high methane baseline (image above), which is a sign on a lactulose breath test that it's going to possibly be a difficult case. You can also see the methane is carpeting her small intestine downstream from that 20. If you look at that 37, and that 43, and that 43, and that 50 at 80 minutes, and a high rise in hydrogen and methane.

So, she was given, for various reasons, herbs and low dose erythromycin and low dose naltrexone. And six weeks later, she still had a 20. Her results were better in terms of rises. So progress was made. But the baseline methane didn't budge.

We continued the herbs, but added to the **levothyroxine T3** as **liothyronine**, 5 mcg., one tablet twice a day. Liothyronine, that is a generic form of T3 which I essentially added to her T4 prescription. And we got a normalized methane (image below).

6-week FU

- rifaximin 550mg TID
- neomycin 500bid
- followed by LDE 50mg. hs
- plus, levothyroxine 50mcg/day: Liothyronin 5mcg, 1 tablet BID

Thyroid Lab	Finding	Normal Range
TSH	1.63	0.450-4.5 uIU/mL
FT4	1.0	0.82-1.77 ng/dL
FT3	3.8	1.0-4.4 pg/mL

lysis with Carbon Dioxide Correction

Number	Expected Location	Collection Interval	ppm H_2	ppm CH_4	Combined	Sample Normalization[1] ppm CO_2	f CO_2
1	Small Intestine	Baseline	7	0	7	4.2	1.30
2		20 Min.	9	1	10	4.2	1.30
3		40 Min.	9	0	9	4.1	1.34
4		60 Min.	26	1	27	4	1.37
5		80 Min.	48	3	51	4.1	1.34
6		100 Min.	55	4	59	4	1.37
7	Transition	120 Min.	62	4	66	4	1.37
8	Large Intestine	140 Min.	125	7	132	4.2	1.30
9		160 Min.	117	7	124	4.2	1.30
10		180 Min.	61	5	66	4.4	1.25

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Now, this is not going to happen in every case this well in full disclosure to my colleagues. And I have many cases like this. But I have other cases where there are other factors. And so we don't want to walk away with a false impression that adding liothyronine will be the answer in every case with high methane. But I am seeing this enough to pay a lot of attention to it.

You can see that her free T3 level—all her levels improved. But her free T3 level went from being in the lower part of the range to a higher part of the range, meaning that European standards.

Another case similar or related, a 31-year old female, also with constipation. She has a bowel movement every three to four days, was diagnosed with slow transit constipation. No response to fiber laxatives, magnesium, various pharmaceutical laxatives like linaclotide or two tricyclic antidepressants. Bloating, flatulence, belching, abdominal discomfort, hair loss, chronic fatigue, lightheadedness, years of thyroid screens with a normal TSH.

31-year old female
cc: Constipation

- BM q 3-4 days since adolescence
- Dx slow transit constipation
- no response to fiber, laxatives, magnesium, linaclotide, tricyclic anti-depressants
- bloating, flatulence, belching, abdominal discomfort
- hair loss, chronic fatigue, lightheadedness
- years of thyroid screens with "normal" TSH

Lactulose Breath Test

Summary Report of Hydrogen & Methane Breath Analysis with Carbon Dioxide Correction

Gasses Analyzed:	Patient Result	Expected (Small Intestine only)
Increase in Hydrogen (H_2) Level:	43 ppm (high)	< 20 ppm
Increase in Methane (CH_4) Level:	52 ppm (high)	< 12 ppm (< 3 ppm[2])
Increase in Combined H_2 & CH_4 Levels:	95 ppm (high)	< 15 ppm
Analysis of the above data suggests:	Data suggests small intestinal bacterial overgrowth[2]	

Number	Expected Location	Collection Interval	ppm H_2	ppm CH_4	Combined	Sample Normalization[1] ppm CO_2	f CO_2
1		Baseline		25	27	4.7	1.17
2		20 Min.	7	46	53	4.5	1.22
3	Small Intestine	40 Min.	23	67	89	4	1.37
4		60 Min.	43	71	114	3.7	1.48
5		80 Min.	37	77	114	4	1.37
6		100 Min.	37	65	102	3.8	1.44
7	Transition	120 Min.	45	70	115	4	1.37
8		140 Min.	44	70	114	4.1	1.34
9	Large Intestine	160 Min.	58	101	159	4	1.37
10		180 Min.	56	104	160	4.2	1.30

Thyroid Lab	Finding	Normal Range
TSH	2.73	0.450-4.5 uIU/mL

And on her lactulose breath test, high methane again and high peak rises of hydrogen and methane. You can see the carpeted methane throughout the small intestine as it goes into the large intestine.

And the TSH, that is frankly normal. But with an interpretation that is somewhat radical in the

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community of it being closer up to 3.0. But with free T3 and free T4 levels that aren't usually even measured in the average conventional office, we have low values and thyroid antibodies.

So, with these thyroid antibodies, we can diagnose autoimmune thyroid disease. And this is a typical picture that I often see in early Hashimoto's where it's not manifesting very strongly in the TSH. If we cover up the rest of the values and just see what an average doctor sees, they don't touch the thyroid. But by doing the more advanced testing, more complete testing, you see that we have uncovered a thyroid problem.

So, this patient is treated with thyroid through a compound thyroid that I chose to make for various reasons instead of using a manufactured product. She took that for a week, and then she doubled it in the second week.

And in three weeks, she had a stool every day, improved energy, and decreased bloating and flatulence.

I continued her on herbs. And at the same time, I added prokinetics. And we see an improvement in her methane and a SIBO test, a lactulose breath test that is largely improved. And we also see improvement in her thyroid tests, at least her function tests, her TSH and her free T3.

A follow-up on this patient is that her constipation is consistently improved. And she has one formed stool a day, but still feels unfinished. She still bloats often. And it's painful.

Treatment
Started thyroid treatment before SIBO treatment

- Compound, per cap: T3/T4 2.25 mcg/9.5mcg
  - 1 cap q am x 1 week
  - then 2 caps q am x 2nd week

3-week f/u:
- stool QD or QOD
- improved energy
- decreased bloating, flatulence and belching

Treatment
- allicin 550mg caps: 1 cap TID
- oregano oil 50mg tabs: 1 tab TID
- proprietary blend per 200mg cap (Emblica officianlis, Terminalia chebula, Terminalia belerica, Tinospora cordifolia, Rubia cordifolia, 300 mg. Azadirachta indica), 1 cap TID
- blend per cap (Berberis vulgaris 400mg, Berberis aquafolium 400mg, Hydrastis canadensis 200mg), 1 cap BID
- 2 weeks later: +LDE 50mg/mL

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Lactulose Breath Test

Summary Report of Hydrogen & Methane Breath Analysis with Carbon Dioxide Correction

Gasses Analyzed:	Patient Result	Expected (Small Intestine only)
Increase in Hydrogen (H_2) Level:	24 ppm (high)	< 20 ppm
Increase in Methane (CH_4) Level:	8 ppm	< 12 ppm (< 3 ppm[2])
Increase in Combined H_2 & CH_4 Levels:	32 ppm (high)	< 15 ppm
Analysis of the above data suggests:	Data suggests small intestinal bacterial overgrowth[2]	

Number	Expected Location	Collection Interval	ppm H_2	ppm CH_4	Combined	Sample Normalization[1] ppm CO_2	f CO_2
1	Small Intestine	Baseline	3	9	12	3.6	1.52
2		20 Min.	6	9	15	3.8	1.44
3		40 Min.	8	11	19	3.6	1.52
4		60 Min.	9	9	18	3.6	1.52
5		80 Min.	12	14	26	3.6	1.52
6		100 Min.	20	16	36	3.5	1.57
7	Transition	120 Min.	27	17	44	3.5	1.57
8	Large Intestine	140 Min.	34	16	50	3.7	1.48
9		160 Min.	56	23	79	3.4	1.61
10		180 Min.	46	17	63	3.3	1.66

Thyroid Lab	Finding	Normal Range
TSH	1.26	0.450-4.5 uIU/mL
FT4	.79 L	0.82-1.77 ng/dL
FT3	2.0	2.0-4.4 pg/mL
Thyroblobulin	158.6	0-60 U/mL
Anti TPO ab	99.8	0-60 U/mL

So, in addition to continuing the botanical protocol and continuing the low dose erythromycin, we added additional prokinetics support and adjust the thyroid with an increased dosage to find improvement in her bowels one month later and an absence of the fatigue, getting better thyroid treatment which is affecting her bowel and her SIBO treatment considerably with a nice concluding lactulose breath test from this period of time, including thyroid function tests.

We earlier said that constipation is the main bowel pattern associated with hypothyroidism. But when there's SIBO (bacterial overgrowth), that can cause

diarrhea in hypothyroidism. But it's not the usual pattern we see.

Follow-Up

- continued botanical protocol at 2/3 dose x 2 weeks, then 1/3 dose x 2 weeks
- continued LDE 50mg hs
- + low dose naltrexone - titration from 1.5mg to 3mg hs
- + thyroid support adjustment
  - from T3/T4 2.25mcg/9.5mcg, 2 caps q am to 3 caps q am

J Clin Gastroenterol. 1990 Feb;12(1):98-9.

Diarrhea in hypothyroidism: bacterial overgrowth as a possible etiology.

Goldin E[1], Wengrower D.

Author information

Abstract

Constipation is common but diarrhea is very rare in myxedematous patients. We report a young woman with myxedema in whom chronic diarrhea was the dominant symptom. A fasting breath hydrogen test was positive, indicating bacterial overgrowth as the cause of the diarrhea. Antibiotic treatment was successful. We conclude that bacterial overgrowth due to hypomotility may be the etiology of the diarrhea in such patients.

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Treating an Underactive Thyroid

So, in going to this slide, when we treat an underactive thyroid, we are often using thyroid medicine. But there are also other pieces of the picture that are detailed here for you.

Nutrition is an important component of treating the thyroid. The **immune system** is important.

The **adrenal gland** function is like a brother or sister to the thyroid gland in terms of completing the endocrine balancing that needs to occur because the thyroid is interacting with adrenal glands and sex steroids in ways known and unknown. And the adrenals is especially important.

And then, **the microbiome** as a whole, SIBO is a piece of. It's representative and part of what's going on with the microbiota, but it's not the whole picture. And Hashimoto's disease and Graves' disease autoimmune thyroid problems are associated very strongly with microbiome issues, of which SIBO is only one piece.

Nutrition

For those of you looking for easy answers, it's hard to say what those are when it comes to this. The nutrition involved and the thyroid metabolism is complicated. It involves adequate **iodine**. Too much iodine by taking lots of supplements of iodine can actually cause a worsening of hypothyroidism. But it can also cause hypothyroidism. So making sure your iodine is right is important.

Selenium, zinc, important minerals. The thyroid needs them to properly function—copper, tyrosine, B12, vitamin E, vitamin A, DHEA, pregnenolone. I'm sorry, the last two are hormones.

The iron is extremely important. I know Dr. Gurevich talked to you about iron tests and lab tests. And it's very important in getting thyroid treatment that the iron is correct, is balanced, that you're not anemic, that you don't have too much or too little.

These are the pieces that need to be looked at.

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The Immune System

The immune system, most thyroid illness is autoimmune. And the treatment of autoimmune diseases, of which these cases get lumped into that category, is a complex discussion about how we detoxify the body, how we use various supplements and pharmaceuticals to help regulate the immune system, how we detoxify the body and what we look at. That has to be taken into consideration in some cases.

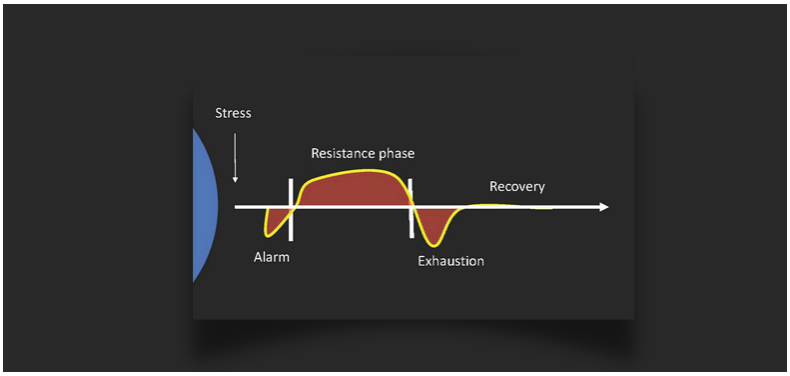
We want to reduce the negative influences on the immune system.

The Adrenals

The adrenal gland makes the hormones cortisol, DHEA, and others. If the cortisol is not balanced in a human being, the thyroid will not work well.

We must at least test and assess adrenal function when we're working with a problematic thyroid gland.

This is a picture of what is called the **reaction of alarm resistance exhaustion and recovery due to a stress stimuli**. This is what's supposed to happen in most of us. You get stressed, and actually, at first, the stress causes a decrease in your adrenal cortisol, which then rebounds in resistance and increases. And you get an increase in this, which is healthy, while you manage the stress. And then, once that is over, you're exhausted, but it recovers. That's the way it's supposed to happen.



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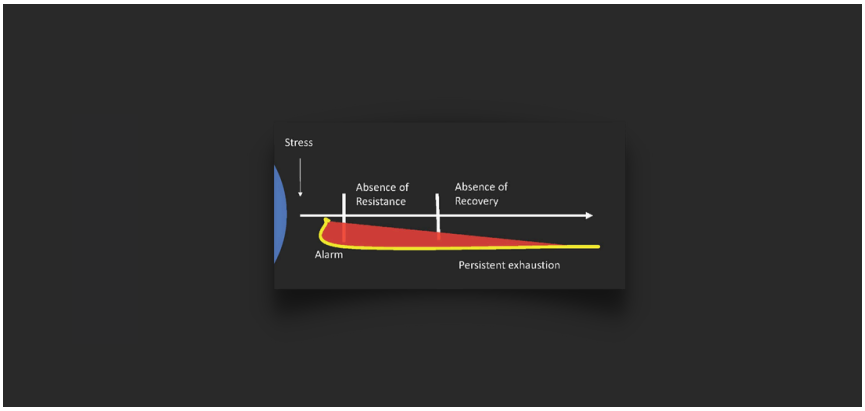
In people who are chronically stressed, it doesn't happen. You have an alarm reaction, and you don't have the cortisol to manage that. You have an absence of resistance. You have an absence of recovery. You have a persistent exhaustion.

I drew this myself. I did this to make the point that it is impossible to have good thyroid regulation if you have this kind of an adrenal function.



We have to address the adrenal gland function with treatment and stress reduction and a change in behavior—which is the hardest things for any of the doctors to do with any of the patients just in the clinical half an hour or hour in addition to prescribing medicine taking care of the SIBO prescriptions. How do we also make an effective change toward changing the things that are so difficult to influence?

So, we have to strengthen our gland. We have to take various supplements and pay attention to that.

Chronic stress impacts thyroid negatively. It reduces T4 to T3 conversion. It suppresses the immune system. It decreases inflammatory control and increases cytokines. It makes thyroid receptors more resistant to thyroid hormones. The prolonged cortisol elevation then increases estrogen and increases levels of thyroid binding globulin. High or low cortisol weaken the immune to system and its primary barriers including the gut barrier.

So, a lot of overlap in low thyroid function and low adrenal function that a good, knowledgeable doctor can help you sort out.

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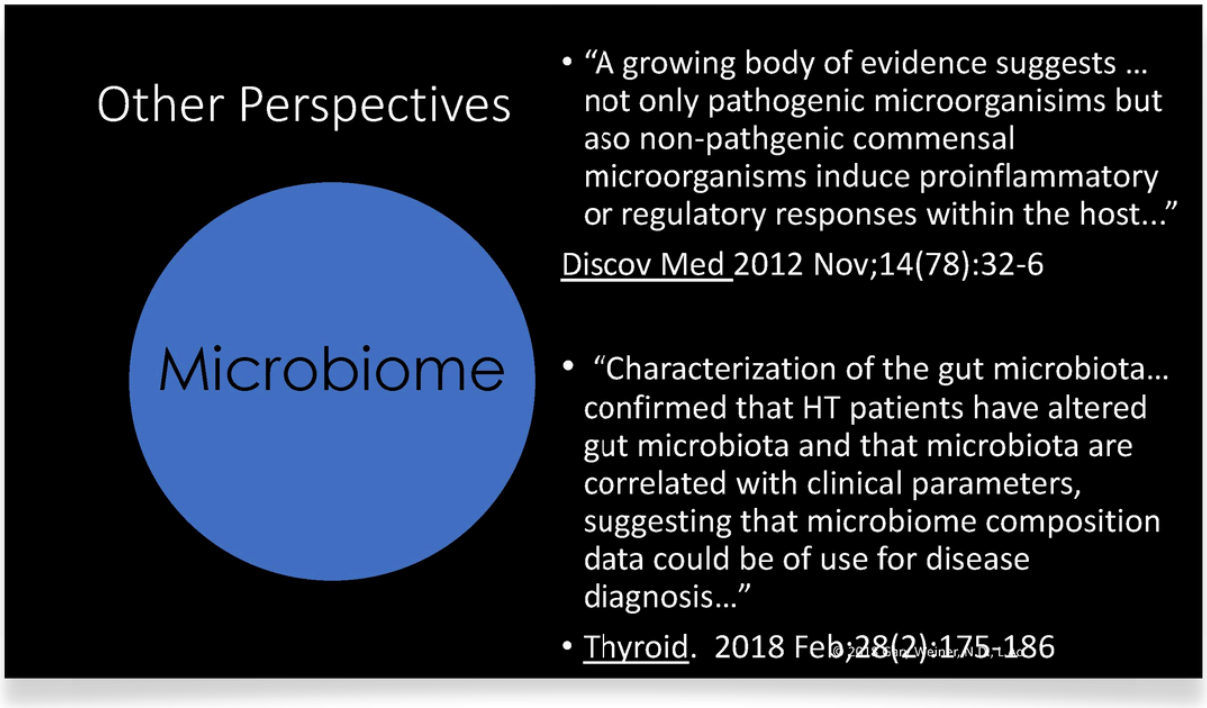
The Microbiome

And the microbiome has to be looked at as a whole in thyroid cases to some degree because the characterization of the gut microbiota was confirmed in Hashimoto's patients. They have altered gut microbiota.



So, when we treat the thyroid, we can use meds. We have to decide T4, T3, natural, synthetic. But we also should look at nutrition, immune system, adrenal function, microbiome. We should look at the whole health picture of a patient.

Between you and me, often, there is a move—and I do this as well—toward medication at first because of its immediate assistance that it can provide to making a patient feel better and having an influence on the case. But getting to the bottom of the low thyroid function often involves these other areas in addition to treating the whole gut itself and influencing the SIBO (which is what we're all here for).

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Conclusions

So, the take home to wrap this presentation up, SIBO docs and patients really need to recognize the thyroid problems are related to SIBO and IBS. If you take home nothing else, take home that.

SIBO docs and patients should understand progressive medical perspectives on the interpretation of TSH, the use of combinations of T3 and T4, and diagnosis of subclinical hypothyroidism.

SIBO docs and patients must consider that abnormal thyroid function is most commonly hypothyroidism, but it can be others. And it can be a big obstacle to the resolution of SIBO.

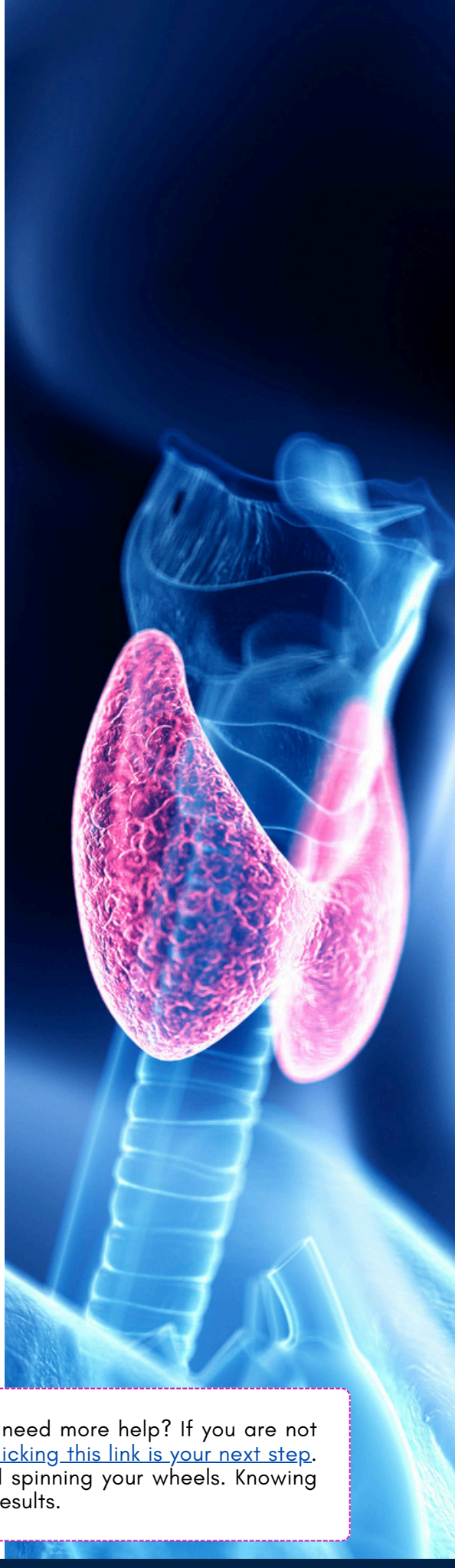
A complete thyroid workup should be performed on SIBO and IBS patients.

That work up for the thyroid: the **TSH**, the **free T3** and the **free T4** (make sure that you don't get the total T3. I mean, they're useful, but these are the ones that are most useful).

We have the **reverse T3** which is very helpful. I don't always require it. And I haven't talked about it very much.

And the antibody test most commonly associated with thyroid problems, the **thyroglobulin antibodies** and the **thyroid peroxidase antibodies**.

Appropriate thyroid medications can be considered for motility support. That's the big take home message. This can be **levothyroxine**. But I am tending not to use that because of the study. I certainly do have cases where people are just fine with levothyroxine.



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I really should take it off the list because since encountering that study, I'm really not using it alone very much at all. But there are some outliers that are still remaining on it who are doing just fine.

T3 medications which are available: desiccated thyroid, and combination thyroid products, including those that are compound by the doctor.

Other forms of treatment can and should be considered. Nutritional treatments, immune enhancements, detox, treatment of the whole GI, and treatment of the whole body, boy, did I get away with murder on that slide.

Action Items

Action items for you in closing:

- If you have SIBO, evaluate the role thyroid problems may be playing.
- Determine if you should pursue treatment of dysthyroidism or refine your current treatment. I realize many of you may be on thyroid treatment already.
- Get the help you need to review thyroid tests in relation to your SIBO.
- And if undiagnosed, determine if the thyroid should be considered as a risk factor for SIBO. I'm now doing a thyroid work-up with most new SIBO patients.
- If already diagnosed, determine if you're on the optimal medication to support motility.
- If you are on the optimal medication, determine if you're on the optimal dose of the optimal medication.
- If you have not been tested for SIBO and have IBS symptoms and thyroid problems, get properly evaluated for SIBO. I don't think I have to tell you folks that. But you never know! There may be somebody who just tuned in and didn't see all the other wonderful presentations.

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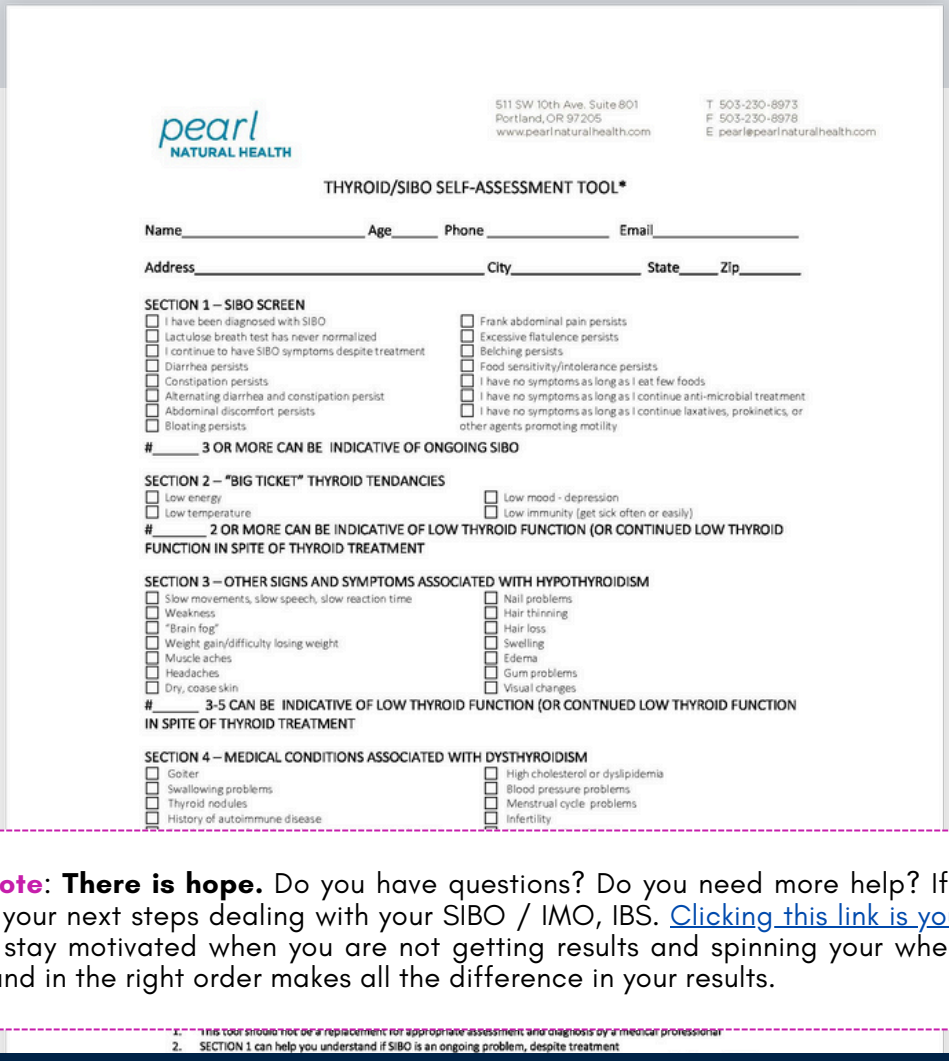
If thyroid problems are present and difficult to address, determine the role of additional factors that may be contributing to dysfunction.

For sure, because of the enormity of the subject, I talk to you less about nutrition, adrenal health, toxic influences, the microbiome, and other subjects related peripherally to thyroid. There's only so much one can do. I think we have had a good introduction to this today.

I've also included a survey to help guide you in your self-help to see if you need to consult further. The first part of it is just for any outliers who are not sure if they're still suffering from SIBO. It's just a helping tool basically for ongoing SIBO.

DOWNLOAD THE THYROID SURVEY (PDF)



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THYROID/SIBO SELF-ASSESSMENT TOOL*

Name_____ Age_____ Phone _____ Email_____

Address_____ City_____ State_____ Zip_____

SECTION 1 – SIBO SCREEN

- ☐ I have been diagnosed with SIBO
- ☐ Lactulose breath test has never normalized
- ☐ I continue to have SIBO symptoms despite treatment
- ☐ Diarrhea persists
- ☐ Constipation persists
- ☐ Alternating diarrhea and constipation persist
- ☐ Abdominal discomfort persists
- ☐ Bloating persists
- ☐ Frank abdominal pain persists
- ☐ Excessive flatulence persists
- ☐ Belching persists
- ☐ Food sensitivity/intolerance persists
- ☐ I have no symptoms as long as I eat few foods
- ☐ I have no symptoms as long as I continue anti-microbial treatment
- ☐ I have no symptoms as long as I continue laxatives, prokinetics, or other agents promoting motility

#_____ 3 OR MORE CAN BE INDICATIVE OF ONGOING SIBO

SECTION 2 – "BIG TICKET" THYROID TENDANCIES

- ☐ Low energy
- ☐ Low temperature
- ☐ Low mood - depression
- ☐ Low immunity (get sick often or easily)

#_____ 2 OR MORE CAN BE INDICATIVE OF LOW THYROID FUNCTION (OR CONTINUED LOW THYROID FUNCTION IN SPITE OF THYROID TREATMENT

SECTION 3 – OTHER SIGNS AND SYMPTOMS ASSOCIATED WITH HYPOTHYROIDISM

- ☐ Slow movements, slow speech, slow reaction time
- ☐ Weakness
- ☐ "Brain fog"
- ☐ Weight gain/difficulty losing weight
- ☐ Muscle aches
- ☐ Headaches
- ☐ Dry, coase skin
- ☐ Nail problems
- ☐ Hair thinning
- ☐ Hair loss
- ☐ Swelling
- ☐ Edema
- ☐ Gum problems
- ☐ Visual changes

#_____ 3-5 CAN BE INDICATIVE OF LOW THYROID FUNCTION (OR CONTNUED LOW THYROID FUNCTION IN SPITE OF THYROID TREATMENT

SECTION 4 – MEDICAL CONDITIONS ASSOCIATED WITH DYSTHYROIDISM

- ☐ Goiter
- ☐ Swallowing problems
- ☐ Thyroid nodules
- ☐ History of autoimmune disease
- ☐ High cholesterol or dyslipidemia
- ☐ Blood pressure problems
- ☐ Menstrual cycle problems
- ☐ Infertility

1. This tool should not be a replacement for appropriate assessment and diagnosis by a medical professional
2. SECTION 1 can help you understand if SIBO is an ongoing problem, despite treatment

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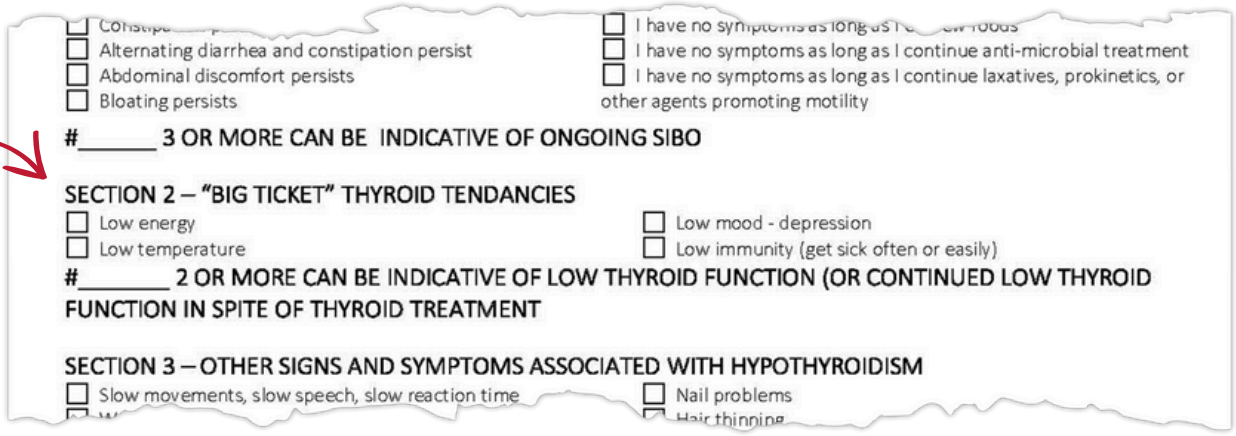
The big ticket thyroid tendencies, the big thing with hypothyroidism, is low energy, low temperature, low neurotransmitter function for mood, and low immunity. If you've got those big ticket items, you really should look at it—other signs and symptoms that can be indicative.



- [ ] Alternating diarrhea and constipation persist
- [ ] Abdominal discomfort persists
- [ ] Bloating persists
- [ ] I have no symptoms as long as I continue anti-microbial treatment
- [ ] I have no symptoms as long as I continue laxatives, prokinetics, or other agents promoting motility

#_____ 3 OR MORE CAN BE INDICATIVE OF ONGOING SIBO

SECTION 2 – "BIG TICKET" THYROID TENDANCIES

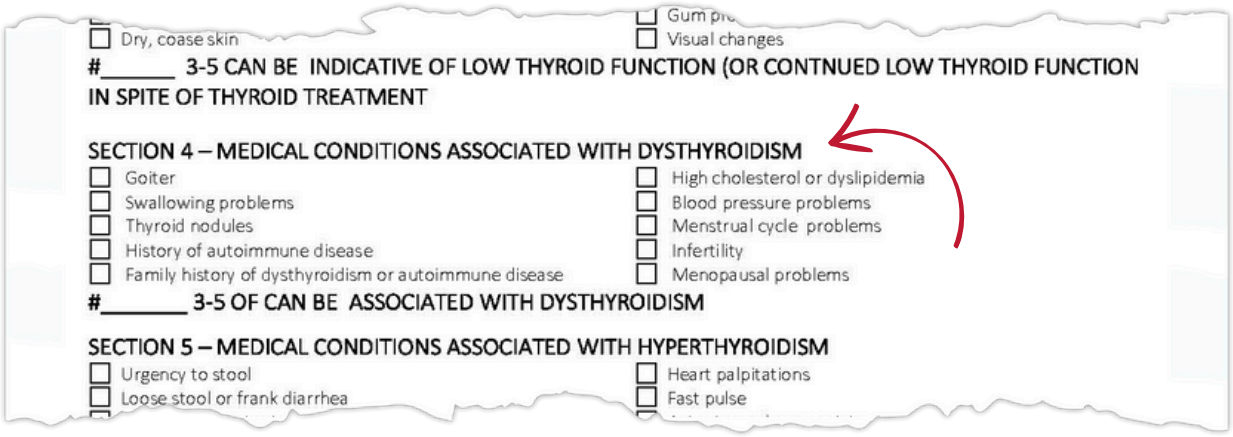
- [ ] Low energy
- [ ] Low temperature
- [ ] Low mood - depression
- [ ] Low immunity (get sick often or easily)

#_____ 2 OR MORE CAN BE INDICATIVE OF LOW THYROID FUNCTION (OR CONTINUED LOW THYROID FUNCTION IN SPITE OF THYROID TREATMENT

SECTION 3 – OTHER SIGNS AND SYMPTOMS ASSOCIATED WITH HYPOTHYROIDISM

- [ ] Slow movements, slow speech, slow reaction time
- [ ] Nail problems

And then, finally, medical conditions, other conditions, that are associated besides SIBO.



- [ ] Dry, coase skin
- [ ] Visual changes

#_____ 3-5 CAN BE INDICATIVE OF LOW THYROID FUNCTION (OR CONTNUED LOW THYROID FUNCTION IN SPITE OF THYROID TREATMENT

SECTION 4 – MEDICAL CONDITIONS ASSOCIATED WITH DYSTHYROIDISM

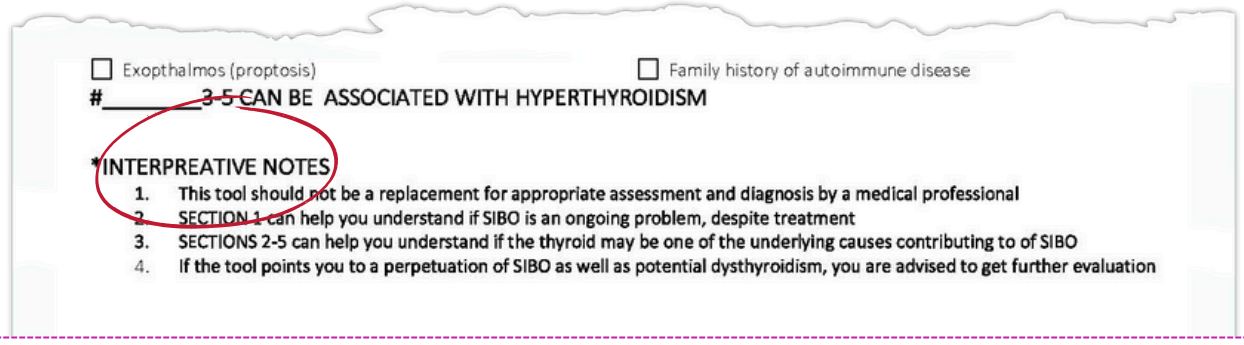
- [ ] Goiter
- [ ] Swallowing problems
- [ ] Thyroid nodules
- [ ] History of autoimmune disease
- [ ] Family history of dysthyroidism or autoimmune disease
- [ ] High cholesterol or dyslipidemia
- [ ] Blood pressure problems
- [ ] Menstrual cycle problems
- [ ] Infertility
- [ ] Menopausal problems

#_____ 3-5 OF CAN BE ASSOCIATED WITH DYSTHYROIDISM

SECTION 5 – MEDICAL CONDITIONS ASSOCIATED WITH HYPERTHYROIDISM

- [ ] Urgency to stool
- [ ] Loose stool or frank diarrhea
- [ ] Heart palpitations
- [ ] Fast pulse

And then, some interpretive notes to help you.



- [ ] Exopthalmos (proptosis)
- [ ] Family history of autoimmune disease

#_____ 3-5 CAN BE ASSOCIATED WITH HYPERTHYROIDISM

*INTERPREATIVE NOTES

1. This tool should not be a replacement for appropriate assessment and diagnosis by a medical professional
2. SECTION 1 can help you understand if SIBO is an ongoing problem, despite treatment
3. SECTIONS 2-5 can help you understand if the thyroid may be one of the underlying causes contributing to of SIBO
4. If the tool points you to a perpetuation of SIBO as well as potential dysthyroidism, you are advised to get further evaluation

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About Dr. Gary Weiner, ND, LAc

Gary Weiner, N.D., L.Ac. was co-founder and clinical director of Pearl Natural Health in downtown Portland Oregon. Dr. Weiner graduated from the National University of Natural Medicine (NUNM) in 1997, and holds degrees in both Naturopathic Medicine and Classical Chinese Medicine. He is a member of the American Association of Naturopathic Medicine, the Oregon Association of Naturopathic Medicine, and a member of the Advisory Committee of the Northwest Crohn's and Colitis Foundation of America. He has been a clinical supervisor at NUNM, and supervises an NUNM certified residency in his clinic, supported by the National Education and Research Consortium (NERC). Dr. Weiner specializes in gastrointestinal and endocrine disorders, with a particular focus on Inflammatory Bowel Disease (IBD), Irritable Bowel Syndrome (IBS) and SIBO. He is a frequent presenter with most recent lectures at the 2017 American Association of Naturopathic Physicians (AANP) convention, the 2017 California Naturopathic Doctors Association (CNDA), and the 2018 SIBO Symposium.

Dr. Gary Weiner, ND, LAc