KRESSER INSTITUTE
FOR FUNCTIONAL AND EVOLUTIONARY MEDICINE

Thyroid Hypofunction I - Part One

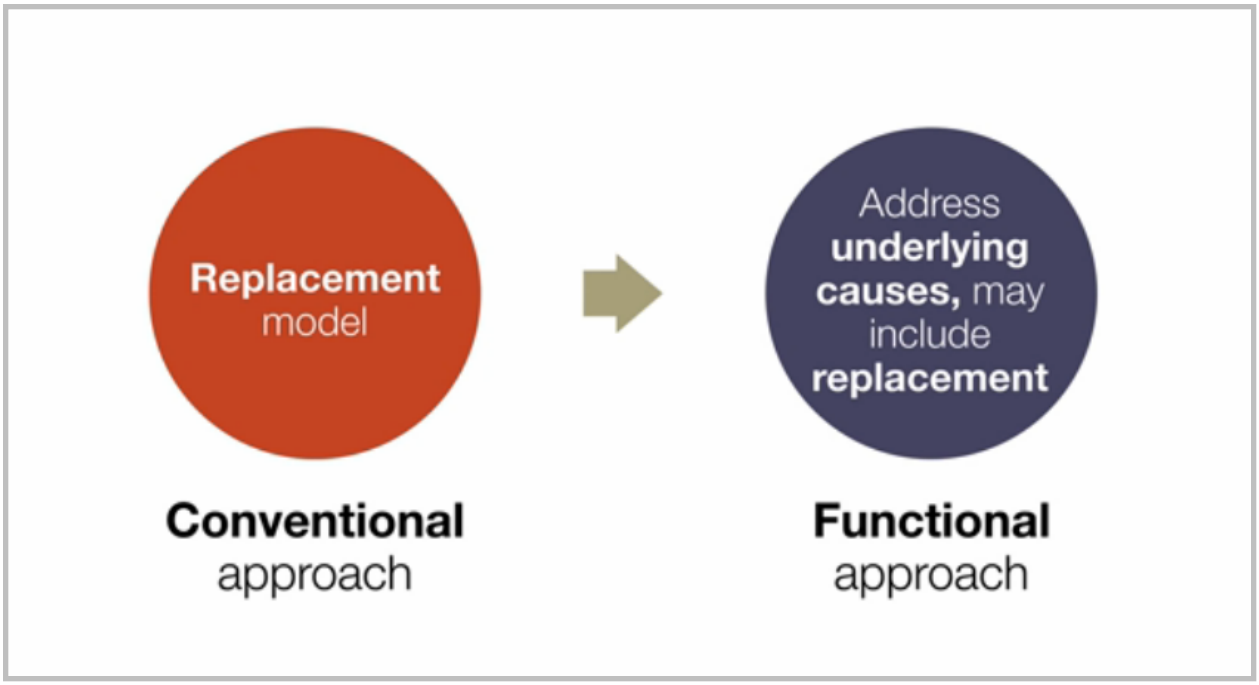
Hey, everyone. In this presentation, we're going to talk about thyroid hypofunction. I've written extensively on this topic on my blog, and rather than rehash that here, I'm going to direct you to those resources so we can focus as much as possible on practical application and other information that is not publically available. If you haven't already, I suggest reading my thyroid e-book. There is good info there on the basics of thyroid physiology and some patterns that you'll see in clinical practice. I've also published a series on low T3 syndrome that I'd like you to read. There is some important information there on low T3 and reverse T3 that is a somewhat different take than the typical integrative or functional medicine viewpoint. Also remember that the focus in ADAPT is on using blood chemistry as a screening tool. I'm going to teach you how to identify imbalances and address them in a basic way, but future courses will dive into more depth on treating particular patterns such as thyroid dysfunction. We could easily spend three months on this alone, but obviously we don't have time to do that within the context of this Level One Framework course.

In this presentation, we're going to start with the basic statistics that are important for you to know, a brief overview of looking at thyroid dysfunction from a functional perspective, what markers for thyroid function I include on the case review blood panel, and then I'll talk briefly about why conventional ranges, for particularly TSH, are not accurate and what the optimal functional range is. Then we're going to dive right into case studies and talk about other aspects of thyroid diagnostics, pathology, and treatment through the functional medicine lens.

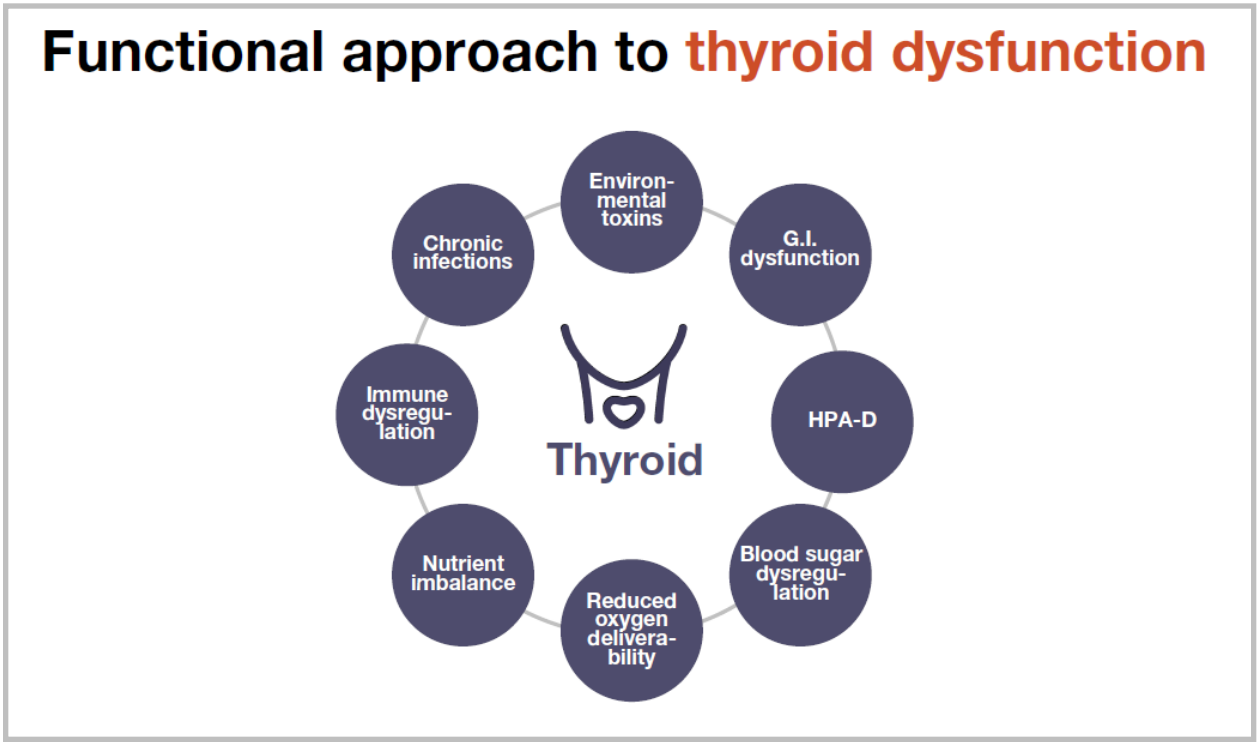
More than 12 percent of people will develop a thyroid condition in their lifetime, so that's over one in ten. Up to 60 percent of them, however, will be unaware of their condition, so in other words, for every two people who know that they have it, there are three who do not. An estimated 20 million Americans currently have thyroid disease. Women are five to eight times more likely than men to have thyroid problems. One woman in eight will develop a thyroid disorder during her lifetime.

The causes of thyroid problems are not fully understood, but they involve genetics, environmental factors, autoimmunity, and dysfunction of other endocrine organs. Undiagnosed thyroid disease

may put patients at risk for certain serious conditions such as cardiovascular disease, osteoporosis, and infertility. Finally, pregnant women with undiagnosed or inadequately treated hypothyroidism have an increased risk of miscarriage, preterm delivery, and severe developmental problems in their children.



There is a fundamental difference between the convention and functional approach to thyroid dysfunction. The conventional approach is the replacement model, so if TSH is high and thyroid hormones are low, you would replace them with exogenous hormone. In the functional model, as with other conditions we've already talked about, we look for the underlying causes of thyroid dysfunction and address those first. We don't exclude the possibility of replacement, and it is sometimes necessary, but that isn't the only way that we address thyroid problems.

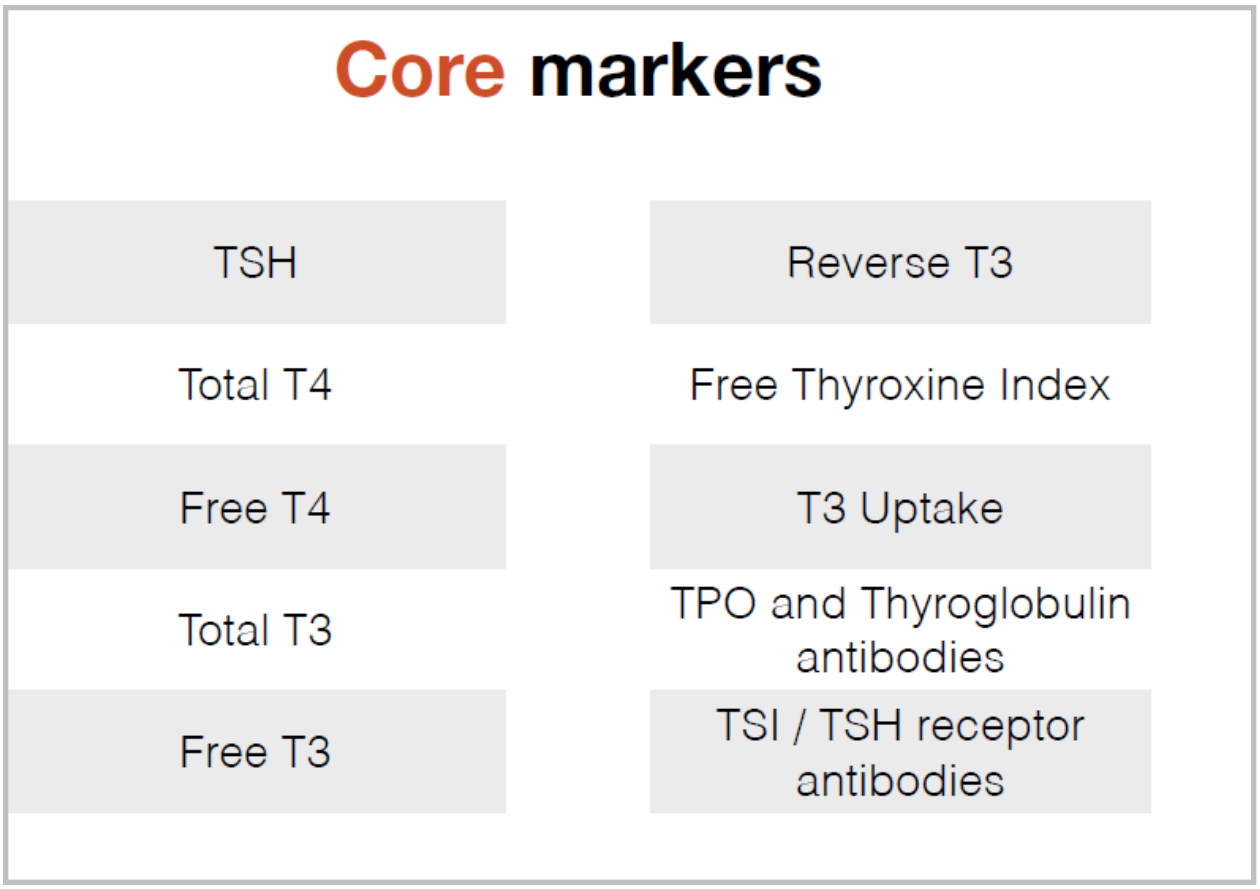


There are numerous interactions between the thyroid gland and the rest of the body, so I've created a chart here on this slide that goes over some of those interactions. The thyroid communicates with the gastrointestinal system. GI dysfunction can contribute to thyroid problems, and thyroid problems can contribute to GI dysfunction. I'm just going to go ahead and say that in almost all of these cases, the relationship is bidirectional, so I don't have to do that every time. HPA axis, the thyroid gland is really part of that axis, and we could more accurately call it the HPTGA axis, G for gonadal. Blood sugar and thyroid have a tight relationship. Reduced oxygen deliverability to the tissue can affect thyroid. Nutrient imbalances can affect thyroid function. Immune dysregulation obviously is one of the major causes of thyroid dysfunction. Chronic infection can decrease thyroid function. Finally, environmental toxins such as mercury can interfere with thyroid function.

The good news is we're covering nearly all of these underlying causes in this ADAPT Framework Level One course with the exception of infections and toxins such as mold and heavy metals. If you do a comprehensive case review with all of the suggested labs we discuss in this course, and you address all of those problems, you're going to be doing 80 to 90 percent of what you need to do to approach thyroid from a functional perspective.

The key point is that thyroid dysfunction is often a symptom or result of a deeper underlying problem, and that is what is so often missed in the conventional paradigm. Replacing thyroid hormone without addressing that problem is not functional medicine, and it won't get you very far.

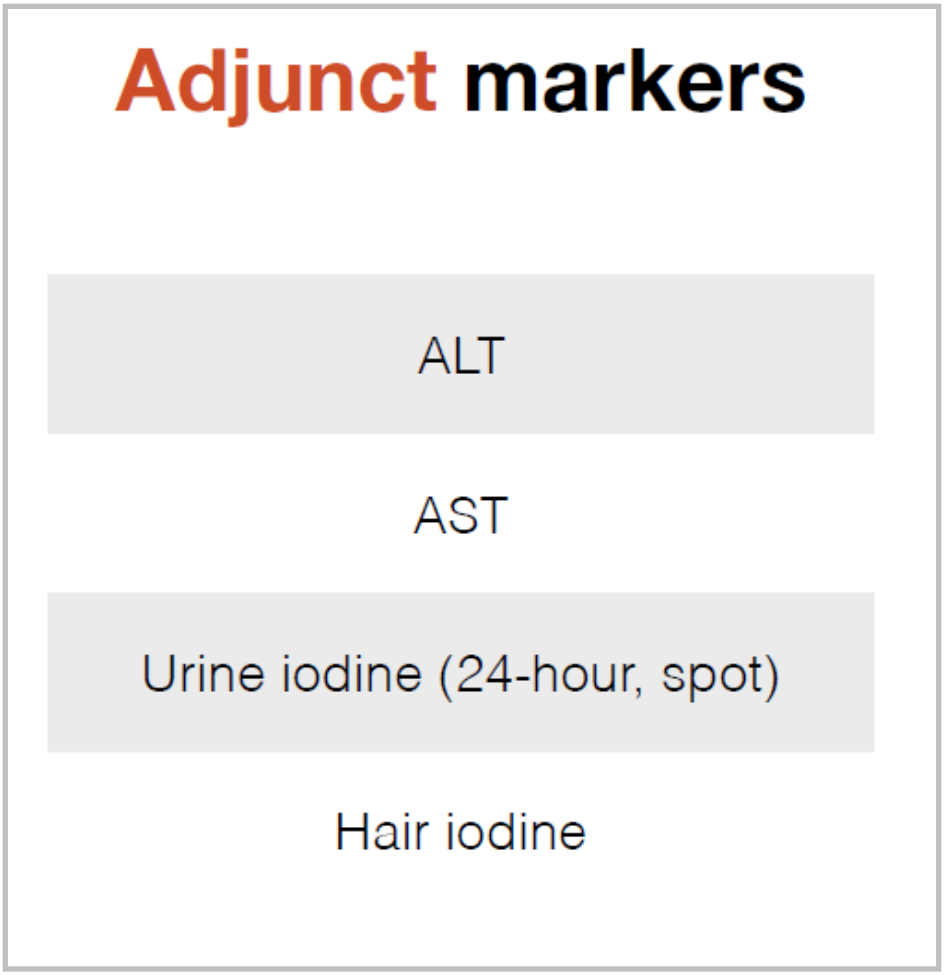
For example, I often see patients, and I'm sure you do too, who have been diagnosed with hypothyroidism many years ago. The physician may have prescribed levothyroxine without any investigation into the underlying causes. Initially the treatment might have been successful in terms of returning TSH and other thyroid hormone markers, if they were even measured, which they are often not, into the normal range, but after a while, the symptoms return, and labs go out of range again. Maybe the doctor increases the dose, and this continues over many years until the patient is at a dose so high that their TSH is effectively zero, and their T3 is high, but they still have tons of symptoms. Now they may be at increased risk of osteoporosis and other issues due to factitious hyperthyroidism, which is the name for hyperthyroidism that is induced by excess medication, and have lost most of their endogenous thyroid function because the autoimmune element wasn't detected or addressed. I really think we can do better as functional medicine clinicians here.



On the case review panel, I include a thyroid panel, which has TSH, total T4, free T4, total T3, free T3, reverse T3, free thyroxine index, T3 uptake, and TPO and thyroglobulin antibodies. T3 uptake is more of a marker of estrogen-testosterone levels and pregnancy, and it's affected by medication use. It's not that relevant in thyroid diagnosis, though it can be useful as part of the overall picture. Free thyroxine index is often out of range in hypothyroid or hyperthyroid states, but it's not a reliable marker on its own. It's possible to save patients money by not including free T4, free T3, and thyroid antibodies on the initial panel. Earlier on I didn't include those on the initial workup,

but now I do because the advantage is that you gather more information upfront. It's certainly helpful to know right away if they have autoimmune thyroid disease, and free T4 and free T3 are better indicators of what's happening at the cellular level than total T4 and total T3.

We also know that antibody production precedes the development of clinical thyroid disease by many years, if not decades, so identifying patients with positive antibodies and normal TSH and thyroid hormones can prevent future problems. The only disadvantage to including all these markers is that it, of course, increases the baseline cost of the case review panel, and in this case, it is somewhat significant. It's about a $40 to $50 increase, so something like 20 percent increase over the basic costs of the panel without those markers.



There are some other markers that can be useful in establishing the diagnosis of hypothyroidism. None of these can be used on their own, but they will often be part of the constellation, so these are alkaline phosphatase; MCV, or mean corpuscular volume; urine iodine, 24-hour or spot; and hair iodine. Alkaline phosphatase and MCV are part of the Case Review blood panel. Urine iodine and hair iodine can be ordered separately.