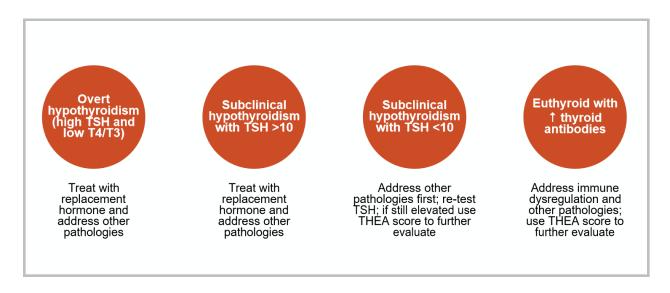


Thyroid Hypofunction II - Part One

Hey, everybody. In this presentation, we're going to cover Part II of thyroid hypofunction. Let's move on to discussing treatment.

The first question is whether to address the thyroid directly. As we talked about previously, thyroid dysfunction can be the result of other underlying pathologies such as autoimmune conditions, nutrient deficiency, or GI disorders; an underlying pathology itself of the thyroid system; or both. In cases where it's the result of another pathology, it is probably better to address that pathology first without treating the thyroid directly. In other cases, you'll have to address the thyroid directly along with the other pathologies in order to get full resolution.



I've listed four different scenarios on this slide along with the recommended course of action for each. First, in overt hypothyroidism with high TSH and low T4 and T3, you'd probably want to treat with replacement hormone, whether natural desiccated thyroid, either over the counter or prescription depending on your scope of practice and your preference, and also address underlying pathologies, but if their numbers are as far off as they are here, they are going to benefit from some thyroid support while you're addressing those other issues.

In subclinical hypothyroidism where TSH is above 10 but T4 and T3 are normal, you may also want to treat with replacement hormone while addressing other pathologies. Even though their T4 and T3 are normal, with a TSH above 10, it's a pretty clear sign that their thyroid is malfunctioning, and most studies suggest that treatment is warranted when TSH is above 10.

Then we have subclinical hypothyroidism with a TSH somewhere between 2 or 2.5 and 10, so it's elevated, but it's not significantly elevated. In that case, I would probably address other



pathologies first and then retest TSH. If it's still elevated, use the THEA score to further evaluate the case and decide whether to treat.

Then finally, we have a patient who is euthyroid with elevated thyroid antibodies, so their TSH, T4, and T3 are normal, but they have high thyroid antibodies. In that situation, I would definitely address immune dysregulation and other pathologies and then use the THEA score to further evaluate.

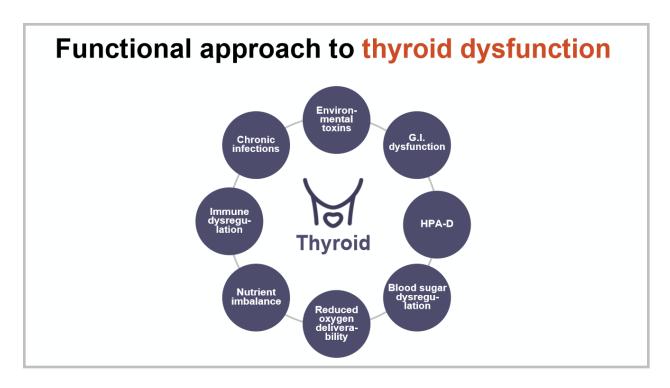
| Calculation of the THEA Score* | | | | | |
|--------------------------------|----------------------|-----------------------|-----------|---|-------------|
| Characteristic | Hypothyroid Event | Hyperthyroid Event | Any Event | | |
| TSH, mIU/L | | | | | |
| <0.4 | 0 | 2 | 2 | THEA Score | 5-year Risk |
| 0.4-2.0 | 0 | 0 | 0 | 0-7 | Low |
| >2.0-4.0 | 3 | -1 | 2 | 8-10 | Medium |
| >4.0-5.7 | 6 | -2 | 4 | 11-15 | High |
| >5.7 | 9 | -3 | 6 | | |
| TPO antibodies, IU/mL | | | | 16-21 | Very High |
| ≤100 | 0 | 0 | 0 | | |
| >100-1000 | 3 | 1 | 4 | | |
| >1000-10000 | 6 | 2 | 8 | | |
| >10000 | 9 | 3 | 12 | | |
| Family Background | | | | + A D - 1' 1' - O | |
| 2 Relatives with Graves | 0 | 1 | 1 | * A Predictive Score for the Occurrence of Events in a Euthyroid Cohort of 790 Women with first- or second-degree Relatives with proven AITD | |
| 2 Relatives with Hashimoto | 3 | 0 | 3 | | |
| Maximum THEA score | 21 | 6 | 21 | | |

I mentioned the THEA score on the last slide. If the patient has only high TSH and/or high thyroid antibodies, you can use this scoring system called THEA, which stands for Thyroid Events Amsterdam, to predict the progression of overt hypothyroidism. Now this was based on the study called Thyroid Events Amsterdam that looked at or tried to predict the progression to overt hypothyroidism or hyperthyroidism in female relatives of patients with autoimmune thyroid disease. We know that siblings of patients with autoimmune thyroid disease have an almost sixfold higher risk of developing hypothyroidism or hyperthyroidism than the general population, which makes sense because 70 percent of the risk of autoimmune thyroid disease is genetic.

THEA is based on TSH, TPO antibodies, and a family history of autoimmune thyroid disease. A score of 0 to 7 indicates a low likelihood of progression to overt hypothyroidism or hyperthyroidism. A score above 7 suggests that the patient should be screened annually with a full thyroid panel, including antibodies. If TSH is above 10, most studies suggest that treatment is warranted regardless of what the THEA score is, or at the very least identifying and addressing the underlying cause. We have **an online calculator** that we've created that makes it very easy for you to calculate the patient's THEA score, and that will help you make decisions on treatment.

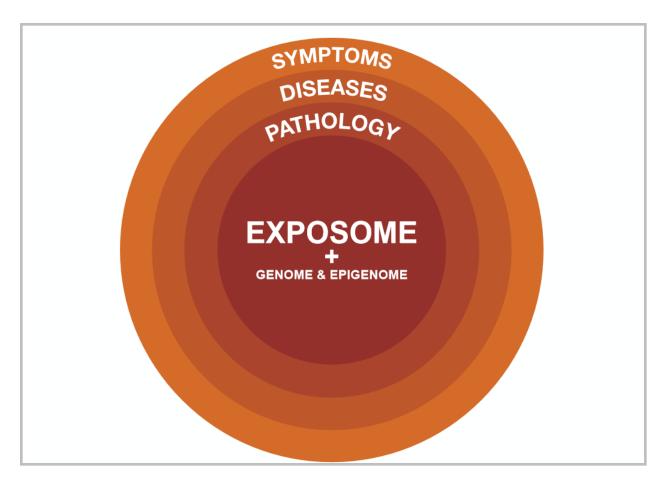


Regardless of the presentation, whether you're starting with direct thyroid treatment or not, you'll always get best results by addressing underlying pathologies. For example, even if the patient has high TSH and low T4 and T3, giving them thyroid hormone alone is often not adequate. We've seen that Hashimoto's is the underlying cause of thyroid dysfunction in up to 90 percent of cases, which means that immune dysregulation must be addressed in the majority of your patients to get a successful outcome. This explains why so many patients who just take thyroid hormone don't get better and/or they need higher and higher doses over time.



But what is the underlying cause of Hashimoto's? This is what often gets overlooked, even in the functional and integrative medicine communities. This is the diagram we saw in Part One of the presentation. All of these mechanisms should be considered in treatment, so environmental toxins, GI dysfunction, HPA axis dysregulation, blood sugar dysregulation, reduced oxygen deliverability or anemia, nutrient imbalance, immune dysregulation, and chronic infections. If a patient has, for example, gut issues such as SIBO, parasites, H. pylori, iron deficiency, mercury toxicity, and dysglycemia, this will provoke or exacerbate Hashimoto's, and you won't really be successful unless you deal with those problems. This is a crucial concept to understand, and it is what differentiates functional endocrinology from the replacement model.





The mechanisms on the last slide are crucial, but don't underestimate the importance of the exposome in healing autoimmune disorders. There is a huge body of evidence connecting sleep deprivation, chronic stress, inappropriate physical activity, lack of social connection, and not enough pleasure or play with autoimmunity, and addressing these factors leads to improvement in autoimmune conditions. In fact, I think these factors may be the most important things for people with autoimmunity to address along with diet.

One of the perils of having all this great lab testing available and knowing how to address and identify underlying mechanisms is that we can lose sight of how important addressing the basics is, and sometimes this can have a bigger effect than addressing those underlying mechanisms themselves because the basics underlie those underlying mechanisms. On this slide here, I'm showing this systems model of functional medicine that we started with way back in the introduction to the course because it is so important to continue to return to this and remember that at the very core, even before the pathologies that we've talked so much about in this course, is the interaction between the exposome and our genome and epigenomes. As clinicians, we always have to remember this. Even if we're addressing things such as SIBO, GI infections, iron deficiency, and things like that, if we're not dealing with stress, lack of physical activity, social connections, or things like that, then we're not going to be successful with the treatment, so please keep that in mind.



We're discussing most of the pathologies listed on the last slide in other parts of ADAPT. We aren't talking about heavy metals, chronic infections, or biotoxin illness in detail in the ADAPT Framework Level One course, although I do intend to teach modules on those subjects later, but I do want to briefly discuss diet and nutrition for people with thyroid dysfunction in particular in a little more detail here.

Dietary nutrients for thyroid health

| Nutrient | Sources | | |
|-----------|--|--|--|
| lodine | Sea vegetables, dairy products, iodized salt | | |
| Selenium | Ocean fish, Brazil nuts, ham | | |
| Iron | Oysters, clams, liver, venison, beef | | |
| Zinc | Oysters, liver, crab, lobster, beef | | |
| B12 | Clam, liver, oyster, mackerel, sardine | | |
| B2 | Liver, mushrooms, seaweed, spinach | | |
| Vitamin C | Red pepper, kiwi, broccoli, citrus | | |
| Vitamin A | Organ meats, CLO, seafood, grass-fed dairy | | |
| Vitamin D | CLO, cold-water fatty fish, UV exposure | | |
| Magnesium | Clams, Swiss chard, spinach, beet greens, kelp | | |

There are two considerations. One is ensuring nutrient balance, and the second is avoiding substances that interfere with proper thyroid function. Nutrients that are important for thyroid health, again, are iodine, selenium, iron, zinc, B12, B2 or riboflavin, vitamin C, vitamin A, vitamin D, and magnesium. With the exception of B12, vitamin C, and magnesium, which have a very high toxicity threshold, we want just the right amount of each of the others. In those cases, too much can be a problem just as too little can be a problem. For this reason, the best option is to eat a nutrient-dense diet with organ meats, shellfish, egg yolks, sea vegetables, and full-fat dairy if it is tolerated. Now remember the cautions that we talked about for iodine, even from sea vegetables, if the patient has elevated thyroid antibodies. It's often worthwhile in these cases to work with a registered dietitian, if you have a relationship with one, who can look at the patient's diet, identify nutrients that the patient may be deficient in, and then prescribe dietary therapy accordingly. We have a handout with all of these nutrients that you'll be able to generate in the handout generator for your patients.