KRESSER INSTITUTE
FOR FUNCTIONAL AND EVOLUTIONARY MEDICINE

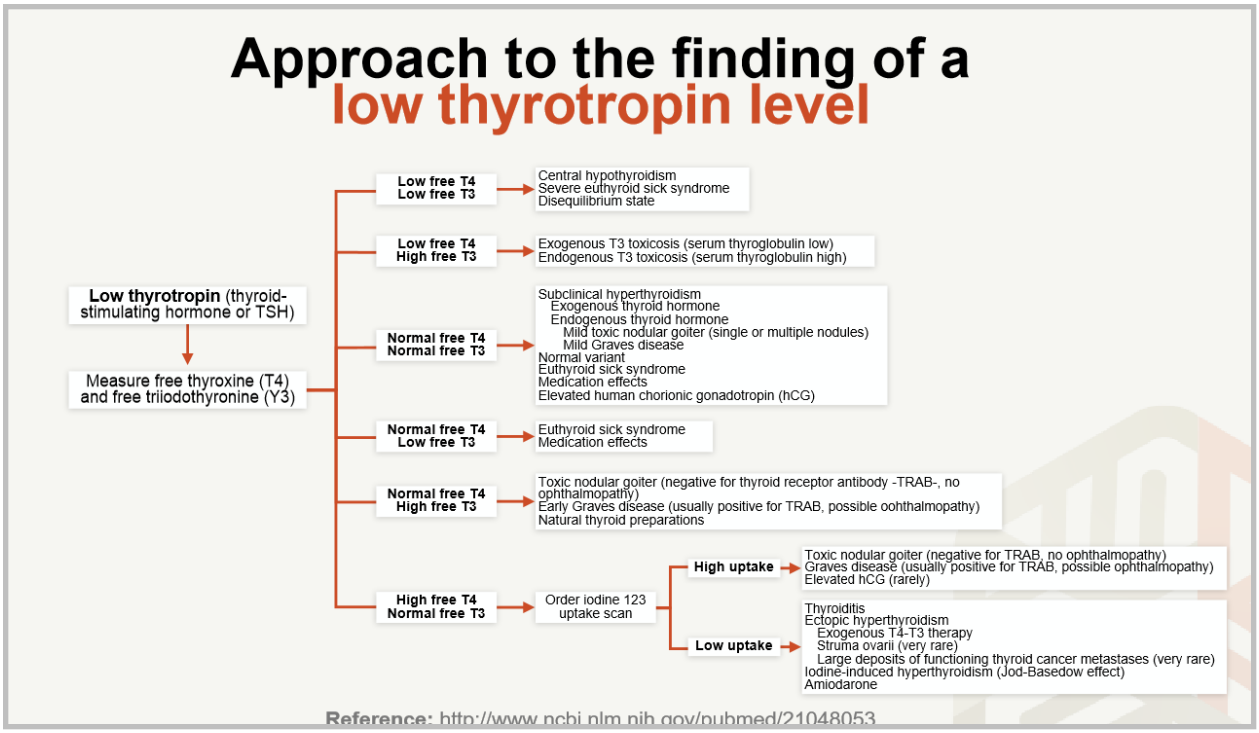
Thyroid Hyperfunction – Part Three

All right, let's move on to a discussion about treatment. The first question, as with hypothyroidism, is whether you should treat the thyroid directly at all or address underlying mechanisms first and then treat. With Graves', it is a little bit different than with Hashimoto's because Graves' can potentially be fatal if T3 levels are significantly elevated. That said, patients are sometimes rushed into aggressive or drastic treatment options such as ablation or thyroidectomy due to concern about the condition worsening and turning into a life-threatening thyroid storm. This is certainly understandable.

Up to 25% of Graves' disease cases spontaneously remit.

However, it's really important to know that spontaneous remission in Graves' disease without treatment occurs at a rate of 10 to 25 percent each year, and most Graves' patients eventually become hypothyroid spontaneously. Also, we need to remember that in Graves', the thyroid isn't the underlying problem. It is the victim. The underlying defect is autoimmunity, so if you can address that, then it is possible that the patient may be able to recover without thyroid-suppressing drugs, ablation, or thyroidectomy.

If only TSH is low, and free T4 and T3 are normal, there is a strong argument for addressing the underlying causes and retesting every two to three months, or sooner if hyperthyroid symptoms increase, to make sure that they are not worsening. If TSH is low, and free T4 or free T3 are very high, you may need to take a more aggressive approach in order to make sure that the patient is safe.

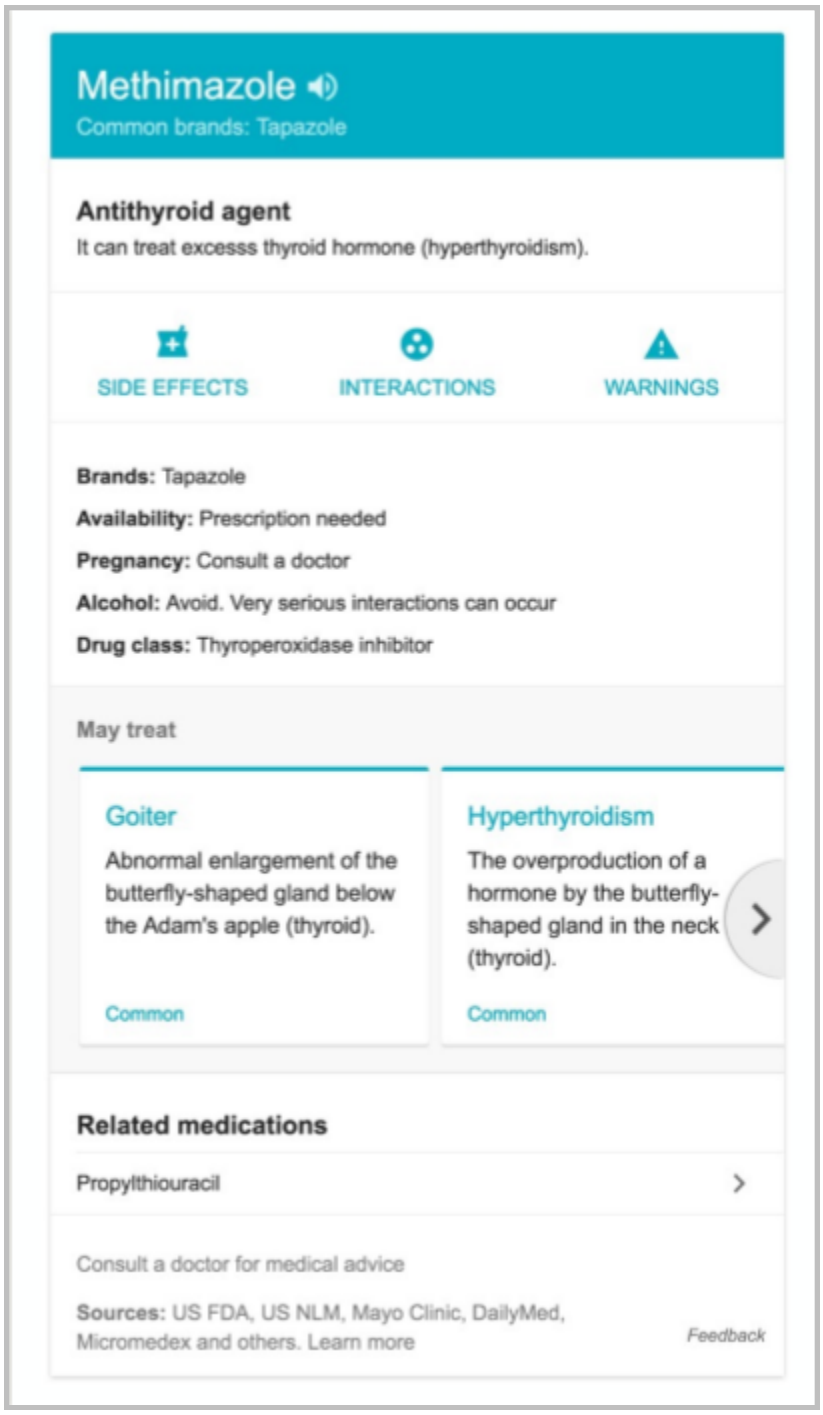


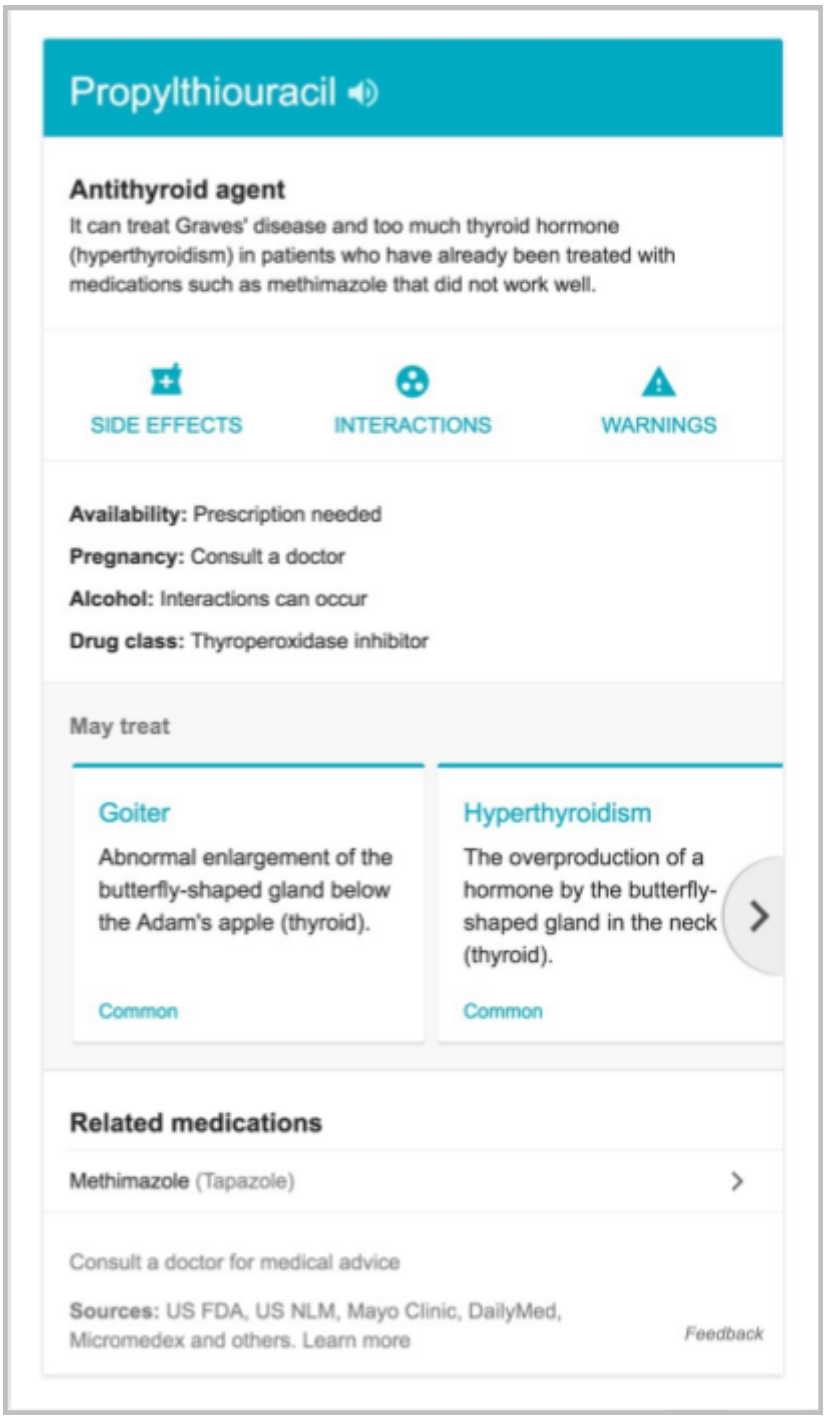
Here is an algorithm you can use to make sense of the various combinations of markers when TSH is low, and we can put this into a handout for you that you can refer to. If free T4 and free T3 are also low, in addition to TSH being low, you're looking at euthyroid sick syndrome, some kind of chronic or acute illness that is affecting both the pituitary output of TSH and also the output of T4 and T3, possibly central hypothyroidism, which is insufficient stimulation by TSH, which we talked about in the last unit, or a disequilibrium state, which is the period between or during the hypothyroid phase of thyroiditis in which the thyroid-stimulating hormone level transiently remains low or inappropriately normal in the setting of low levels of free thyroid hormones.

If free T4 is low but free T3 is high, the patient is probably taking too much natural desiccated thyroid, which suppressed endogenous T4 production. If free T4 and free T3 are normal, you're looking at subclinical hyperthyroidism. They may have hypothyroidism and are taking thyroid replacement. They may have mild toxic goiter or mild Graves'. They may be taking growth hormone, and or it could be nonpathological.

If free T4 is normal, and free T3 is low, it could be again euthyroid sick syndrome, chronic inflammation reducing the conversion of free T4 to free T3, or medication effects. If free T4 is normal, and free T3 is high, that's not uncommon for just standard Graves' disease. It also can happen with toxic multinodular goiter if they are negative for TSI and don't have ophthalmopathy, and it could happen if they are taking natural desiccated thyroid.

If you see high free T4 and/or normal or high free T3, that could be Hashitoxicosis, especially if the free T3 is very high. You want to refer out for additional workup.


Methimazole
Common brands: Tapazole
Antithyroid agent
It can treat excesss thyroid hormone (hyperthyroidism).
SIDE EFFECTS
INTERACTIONS
WARNINGS
Brands: Tapazole
Availability: Prescription needed
Pregnancy: Consult a doctor
Alcohol: Avoid. Very serious interactions can occur
Drug class: Thyroperoxidase inhibitor
May treat
Goiter
Abnormal enlargement of the butterfly-shaped gland below the Adam's apple (thyroid).
Common
Hyperthyroidism
The overproduction of a hormone by the butterfly-shaped gland in the neck (thyroid).
Common
Related medications
Propylthiouracil
Consult a doctor for medical advice
Sources: US FDA, US NLM, Mayo Clinic, DailyMed, Micromedex and others. Learn more
Feedback



If free T4 and/or free T3 are significantly elevated, and the patient has tachycardia and other concerning symptoms, how should they evaluate the various treatment options? Before Graves' was recognized as an autoimmune condition, radioactive ablation or thyroidectomy seemed like the best options. Now, however, using antithyroid drugs such as PTU or methimazole is favored because they can actually reduce TSI production and help the immune system to recover.

Surgery removes some of the TSI because they are stored in the thyroid gland. However, removing the thyroid gland is a little bit like removing gum from the sole of a shoe. It's hard to get all of it. There are sensitive structures in the neck, and surgeons don't want to damage them, so they are cautious about removing all of the tissue. This means that TSI production can continue, especially if patients don't remove the underlying autoimmune triggers.

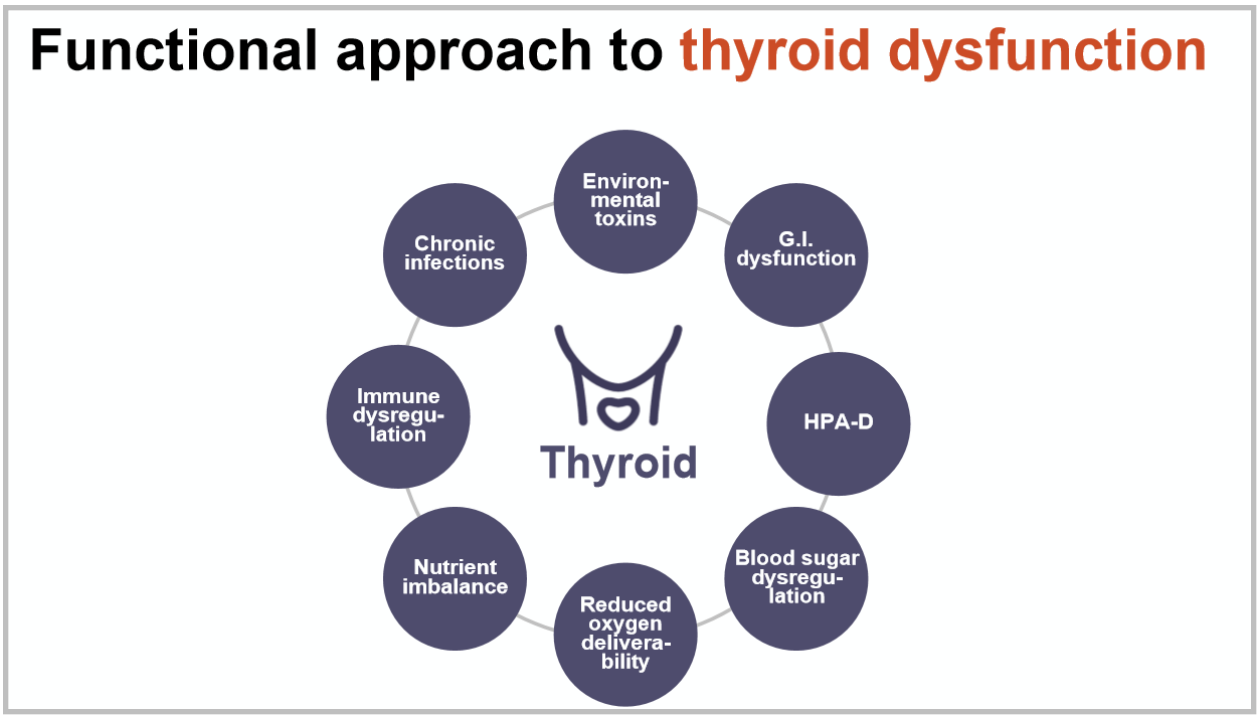
When radioiodine is used to ablate or destroy the thyroid gland, the immune cells within the gland perceive this as a foreign attack. TSI production dramatically rises, which causes an increase in hyperthyroidism that persists until most of the thyroid tissue is destroyed, which can take a long time. Studies have shown that after radioactive ablation, TSI production persists, peaks within one year, and remains elevated for many years. It's also established that radioactive ablation causes a slight but significant increase in thyroid and small intestine cancer.

Finally, several years after radioactive ablation, the body produces blocking TSI. These not only block TSI but also TSH from stimulating the thyroid gland, which leads to atrophic hypothyroidism, and blocking TSI can also contribute to eye disease and skin complications.

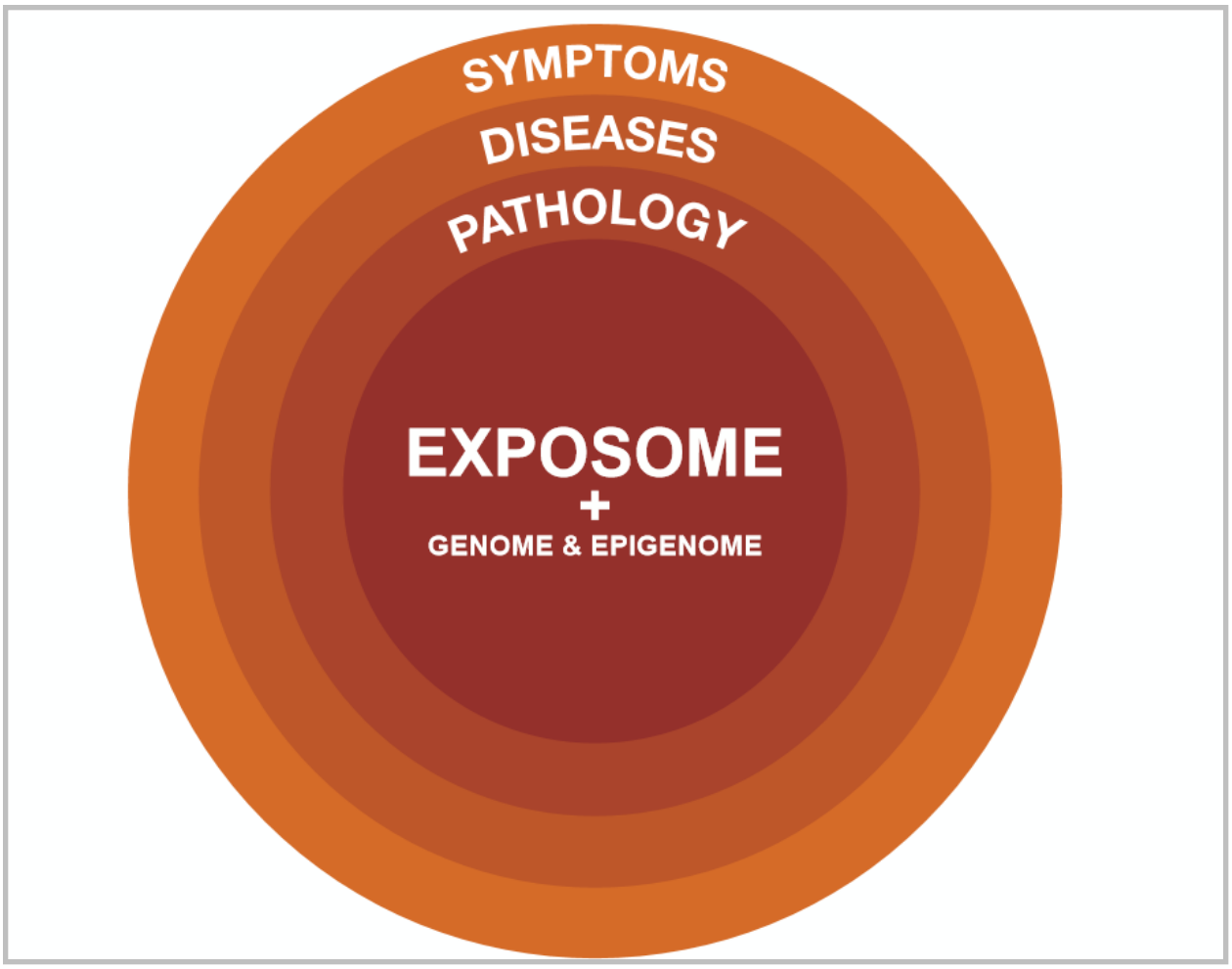
As you can see, surgery and radioactive ablation are typically not good choices. If the patient needs conventional intervention while you are working on underlying causes, either methimazole or PTU would be far better options.

There are, however, many considerations with methimazole and PTU, and the choice of which to take depends on several factors. In pregnancy and nursing, PTU is preferred, since it is less likely to cross the placental barrier and affect the fetus. If there are signs of fetal thyrotoxicosis, however, methimazole is used because it can simultaneously treat fetal symptoms of hyperthyroidism.

PTU has a shorter half-life, and it has to be taken more frequently. At the onset of hyperthyroidism, patients usually take 100 mg three times a day every six to eight hours. Methimazole has a longer half-life and can be taken once daily. Methimazole typically leads to euthyroidism or normalizing thyroid numbers more quickly. It takes about four weeks, while PTU may take as long as 12 weeks. Patients on methimazole go into remission sooner than patients on PTU and are thought to have less trouble with side effects or eye complications. Unless you have experience with these antithyroid drugs, I would highly recommend you refer to an endocrinologist, and you can continue helping the patient with the underlying conditions.



Okay, so in terms of a functional approach to hyperthyroidism, again, you've seen this diagram. These are all the things that you need to be thinking about.



The mechanisms on the last slide are crucial, but don't underestimate the importance of the exposome in healing autoimmune disorders. There is, of course, 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 has been shown to lead to improvement. I've said this before, but I'll say it again. I think these factors may actually be the most important things to address for people with autoimmunity along with diet. It is one of the perils of having all of this fantastic lab testing available and knowing how to address and identify these underlying mechanisms that we can lose sight of how important addressing the basics really is. That's because the basics are what lead to the pathological mechanisms in the first place, and that's again the importance of this diagram and really keeping this in mind as you go. I continually refer to it mentally despite the fact that I've been in practice now for many years because it is just so crucial to understanding what the crux of the problem is with our patients.

Dietary nutrients for thyroid health

Nutrient	Sources
Iodine	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

Okay, so these are the dietary nutrients for thyroid health. This slide is from the hypothyroidism presentation, but the nutrients required for thyroid health are the same. The two considerations are ensuring nutrient balance and avoiding substances that interfere with proper thyroid function. I'm not going to go through this again, since we already went through it. You can refer to that presentation for details.