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Thyroid Disorders

An e-book adapted by CHRIS KRESSER (USA)

This e-book has been adapted from the Chris Kresser Institute, an esteemed alternative health expert. It is the best information I can find on thyroid health. I have added in some information about Australian statistics in this book and about how the Compounding Lab can help and support thyroid issues. I hope you find this information worthwhile.

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Thyroid Health: Australian Stats

It is thought six to ten percent of women in Australia are affected by hypothyroidism and the risk increases with age. A great deal of these people are unaware of their condition. One in eight women will develop a thyroid disorder during her lifetime. The number of people suffering from thyroid disorders continues to rise each year. It is currently estimated that 850,000 Australians live with a thyroid issue.

Hypothyroidism is one of the most common thyroid disorders. One recent analysis suggested up to 10% of women over 60 have clinical or sub-clinical hypothyroidism. It is characterized by mental slowing, depression, dementia, weight gain, constipation, dry skin, hair loss, cold intolerance, hoarse voice, irregular menstruation, infertility, muscle stiffness and pain, and a wide range of other not-so-fun symptoms.

In fact, every cell in the body has receptors for thyroid hormone. These hormones are responsible for the most basic aspects of body function, impacting all major systems of the body. Thyroid hormone directly acts on the brain, the G.I. tract, the cardiovascular system, bone metabolism, red blood cell metabolism, gall bladder and liver function, steroid hormone production, glucose metabolism, lipid and cholesterol metabolism, protein metabolism and body temperature regulation. For starters. You can think of the thyroid as the central gear in a sophisticated engine. If that gear breaks, the entire engine goes down with it.

One of the biggest challenges facing those with hypothyroidism is that the standard of care for thyroid disorders in both conventional and alternative medicine is hopelessly inadequate.

This eBook explains why thyroid disorders are so often under- and misdiagnosed, why so many patients with thyroid symptoms have normal lab results, why conventional thyroid treatments so often don't work, and how to successfully treat thyroid disorders by addressing the underlying mechanisms involved.

The Most Important Thing You May Not Know About Hypothyroidism

The dream of patients with thyroid disorders and the practitioners who treat them is to find that single substance that will magically reverse the course of the disease. For doctors, this is either synthetic or bio-identical thyroid hormone. For the alternative types, this is iodine.

Unfortunately, in the vast majority of cases neither approach is effective. Patients may get relief for a short period of time, but inevitably symptoms return or the disease progresses.

So what's the problem? Why have replacement hormones and supplemental iodine been such dismal failures? Because hypothyroidism is caused by an autoimmune disease.

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The most common cause of primary hypothyroidism (where the thyroid itself fails) in Australia is an autoimmune disease called Hashimoto's. This is where the immune system to attack and destroy the thyroid, which over time causes a decline in thyroid hormone levels.

While not all people with Hashimoto's have hypothyroid symptoms, thyroid antibodies have been found to be a marker for future thyroid disease.

Most doctors know hypothyroidism is an autoimmune disease. But most patients don't. The reason doctors don't tell their patients is simple: it doesn't affect their treatment plan.

Conventional medicine doesn't have effective treatments for autoimmune disease. They use steroids and other medications to suppress the immune system in certain conditions with more potentially damaging effects, such as multiple sclerosis, rheumatoid arthritis and Crohn's disease.

But in the case of Hashimoto's, the consequences - i.e. side effects and complications - of using immunosuppressive drugs are believed to outweigh the potential benefits. (Thanks to conventional medicine for a relative moment of sanity here.)

So the standard of care for a Hashimoto's patient is to simply wait until the immune system has destroyed enough thyroid tissue to classify them as hypothyroid, and then give them thyroid hormone replacement. If they start to exhibit other symptoms commonly associated with their condition, like depression or insulin resistance, they'll get additional drugs for those problems.

The obvious shortcoming of this approach is that it doesn't address the underlying cause of the problem, which is the immune system attacking the thyroid gland. And if the underlying cause isn't addressed, the treatment isn't going to work very well - or for very long.

If you're in a leaky rowboat, bailing water will only get you so far. If you want to stop the boat from sinking, you've got to plug the leaks.

Extending this metaphor to Hashimoto's disease, thyroid hormones are like bailing water. They may be a necessary part of the treatment. But unless the immune dysregulation is addressed (plugging the leaks), whoever is in that boat will be fighting a losing battle to keep it from sinking.

Hashimoto's often manifests as a "polyendocrine autoimmune pattern". This means that in addition to having antibodies to thyroid tissue, it's not uncommon for Hashimoto's patients to have antibodies to other tissues or enzymes as well. The most common are transglutaminase (Celiac disease), the cerebellum (neurological disorders), intrinsic factor (pernicious anemia), glutamic acid decarboxylase (anxiety/panic attacks and late onset type 1 diabetes).

What the vast majority of hypothyroidism patients need to understand is that they don't have a problem with their thyroid, they have a problem with their immune system attacking the thyroid. This is crucial to understand, because when the immune system is out of control, it's not only the thyroid that will be affected.

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5 Thyroid Patterns That Won't Show Up On Standard Blood Tests

Unfortunately misdiagnosis is common in the management of hypothyroidism. If you go to a doctor with hypothyroid symptoms, you'll simply be given replacement hormones without any further inquiry into the cause of your condition.

Even worse, if you have hypothyroid symptoms but your blood tests are normal, you'll be told you're "fine". If you insist you're not, you might be sent home with an antidepressant, but no further clue about the cause of your symptoms.

The problem with this approach is that thyroid physiology is complex. The production, conversion and uptake of thyroid hormone in the body involves several steps. A malfunction in any of these steps can cause hypothyroid symptoms, but may not show up on standard lab tests. It's incorrect and even negligent to assume that all cases of hypothyroidism share the same cause and require the same treatment. Yet that's exactly what the standard of care for hypothyroidism delivers.

Now, I'm going to present five patterns of thyroid dysfunction that won't show up on standard blood tests. If you have one of these patterns, your thyroid isn't functioning properly and you will have symptoms. But sometimes if you go to your doctor, you maybe be told there's nothing wrong with your thyroid.

A standard thyroid panel usually includes TSH and T4 only or maybe just the TSH. The ranges for these may vary from lab to lab, which is one of two main problems with standard lab ranges. The other problem is that lab ranges are not based on research that tells us what a healthy range might be, but on a bell curve of values obtained from people who come to the labs for testing.

So it follows that this may not be an ideal method. Who goes to labs to get tested? Sick people. If a lab creates its "normal" range based on test results from sick people, is that really a normal range?

Does that tell us anything about what the range should be for health?

The following are the five major thyroid patterns that don't show up on standard tests.

1. HYPOTHYROIDISM CAUSED BY PITUITARY DYSFUNCTION

This pattern is caused by elevated cortisol, which is in turn caused by active infection, blood sugar imbalances, chronic stress, pregnancy, hypoglycemia or insulin resistance. These stressors fatigue the pituitary gland at the base of the brain so that it can no longer signal the thyroid to release enough thyroid hormone. There may be nothing wrong with the thyroid gland itself. The pituitary isn't sending it the right messages.

With this pattern, you'll have hypothyroid symptoms and a TSH below the functional range (1.8 - 3.0) but within the standard range (0.5 - 5.0). The T4 will be low in the functional range (and possibly the lab range too).

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2. UNDER-CONVERSION OF T4 TO T3

T4 is the inactive form of thyroid hormone. It must be converted to T3 before the body can use it. More than 90% of thyroid hormone produced is T4.

This common pattern is caused by inflammation and elevated cortisol levels. T4 to T3 conversion happens in cell membranes. Inflammatory cytokines damage cell membranes and impair the body's ability to convert T4 to T3. High cortisol also suppresses the conversion of T4 to T3.

With this pattern you'll have hypothyroid symptoms, but your TSH and T4 will be normal. If you have your T3 tested, which it rarely is in conventional settings, it will be low.

3. HYPOTHYROIDISM CAUSED BY ELEVATED TBG

Thyroid binding globulin (TBG) is the protein that transports thyroid hormone through the blood. When thyroid hormone is bound to TBG, it is inactive and unavailable to the tissues. When TBG levels are high, levels of unbound (free) thyroid hormone will be low, leading to hypothyroid symptoms.

With this pattern, TSH and T4 will be normal. If tested, T3 will be low, and T3 uptake and TBG will be high.

Elevated TBG is caused by high estrogen levels, which are often associated with birth control pills or estrogen replacement (i.e. Premarin or estrogen creams). To treat this pattern, excess estrogen must be cleared from the body.

4. HYPOTHYROIDISM CAUSED BY DECREASED TBG

This is the mirror image of the pattern above. When TBG levels are low, levels of free thyroid hormone will be high. You might think this would cause hyperthyroid symptoms. But too much free thyroid hormone in the bloodstream causes the cells to develop resistance to it. So, even though there's more than enough thyroid hormone, the cells can't use it and you'll have hypothyroid - not hyperthyroid - symptoms.

With this pattern, TSH and T4 will be normal. If tested, T3 will be high, and T3 uptake and TBG will be low.

Decreased TBG is caused by high testosterone levels. In women, it is commonly associated with PCOS and insulin resistance. Reversing insulin resistance and restoring blood sugar balance is the key to treating this pattern.

5. THYROID RESISTANCE

In this pattern, both the thyroid and pituitary glands are functioning normally, but the hormones aren't getting into the cells where they're needed. This causes hypothyroid symptoms.

Note that all lab test markers will be normal in this pattern, because we don't have a way to test the function of cellular receptors directly.

Thyroid resistance is usually caused by chronic stress and high cortisol levels. It can also be caused by high homocysteine and genetic factors.

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Thyroid resistance is usually caused by chronic stress and high cortisol levels. It can also be caused by high homocysteine and genetic factors.

The five patterns above are only a partial list. Several others also cause hypothyroid symptoms and don't show up on standard lab tests. If you have hypothyroid symptoms, but your lab tests are normal, it's likely you have one of them.

Not only do these patterns fail to show up on standard lab work, they don't respond well to conventional thyroid hormone replacement. If your body can't convert T4 to T3, or you have too much thyroid binding protein, or your cells are resistant, it doesn't matter how much T4 you take; you won't be able to use it.

Unfortunately, if you have one of these patterns and tell your doctor your medication isn't working, all too often the doctor's response is to simply increase the dose. When that doesn't work, the doctor increases it yet again.

The good news is that, once the correct diagnosis is made, you respond very well to treatment.

The Gluten-Thyroid Connection

Several studies show a strong link between autoimmune thyroid disease (both Hashimoto's and Graves') and gluten intolerance. The Compounding Lab can help with testing for intolerance. The link is so well- established that researchers suggest all people with autoimmune thyroid disease (AITD) be screened for gluten intolerance, and vice versa.

What explains the connection? It's a case of mistaken identity. The molecular structure of gliadin, the protein portion of gluten, closely resembles that of the thyroid gland. When gliadin breaches the protective barrier of the gut, and enters the bloodstream, the immune system tags it for destruction. These antibodies to gliadin also cause the body to attack thyroid tissue. This means if you have AITD and you eat foods containing gluten, your immune system will attack your thyroid.

Even worse, the immune response to gluten can last up to 6 months each time you eat it. This explains why it is critical to eliminate gluten completely from your diet if you have AITD. There's no "80/20" rule when it comes to gluten. Being "mostly" gluten-free isn't going to cut it. If you're gluten intolerant, you have to be 100% gluten-free to prevent immune destruction of your thyroid.

So how do you find out if you're gluten intolerant? Unfortunately, standard lab tests aren't very accurate. They test for antibodies to gluten in the bloodstream. But antibodies in the blood will only be found in cases where the gut has become so permeable that gluten can pass through. This is a relatively advanced stage of disease. Blood tests will miss the many milder cases of gluten intolerance that haven't yet progressed to that stage.

That's why most experts on gluten sensitivity agree that the only reliable test for gluten intolerance is a "gluten challenge." This involves removing gluten from the diet completely for a period of at least 30 days (though preferably three months), and then adding it back in after that. If symptoms improve during the elimination period, and return when gluten is reintroduced, a diagnosis of gluten intolerance can be made.

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However, for many people a gluten-free diet isn't enough. Some grains that don't contain gluten, such as corn, oats and rice, contain proteins that are similar enough in structure to gluten to elicit an immune response in people with celiac disease or gluten intolerance. Moreover, about 50 percent of patients with celiac disease show signs of intolerance to casein (the protein in milk), and up to 30 percent of celiac disease patients continue to have symptoms or clinical signs after adopting a gluten-free diet.

One reason gluten intolerance goes undetected in so many cases is that both doctors and patients mistakenly believe it only causes digestive problems. But gluten intolerance can also present with inflammation in the joints, skin, respiratory tract and brain—without any obvious gut symptoms.

Another problem is that some patients with autoimmune disease, their immune system is so worn out they can no longer produce many antibodies.

Hashimoto's, the most common autoimmune thyroid condition, is primarily a Th1 dominant condition. The basic information to understand is that in Th1-dominant conditions, the Th2 system is suppressed. The Th2 system is the part of the immune system responsible for producing antibodies. When the Th2 system is severely depressed, the body's ability to produce antibodies is impaired. The levels may be so low that they won't show up on a test. So, even if you have gluten intolerance, your test for gluten antibodies may be falsely negative if you have Th1-dominant Hashimoto's.

This is why I recommend that you avoid gluten if you have AITD, regardless of whether tests show an active antibody response. This is especially true if you have one of the genes (HLA DQ1,2, or 3) that predisposes you to developing gluten intolerance. In my opinion continuing to eat gluten when you have a confirmed autoimmune condition simply isn't worth risking the immune destruction it could cause.

The short version: foods that contain gluten (both whole grains and flours) often also contain substances that inhibit nutrient absorption, damage our intestinal lining, and activate a potentially destructive autoimmune response. What's more, there are no nutrients in gluten-containing foods that you can't get more easily and efficiently from foods that don't contain gluten.

The good news is that if you have AITD and are gluten intolerant removing gluten completely from your diet will dramatically improve your health. It's not easy, but it's worth it.

Iodine for Hypothyroidism: Crucial Nutrient or Harmful Toxin?

When used alone, thyroid hormone replacement often fails. One of the reasons can be due to a suboptimal iodine intake, as both too little and too much iodine can be harmful.

Iodine deficiency is the most common cause of hypothyroidism worldwide. Once researchers realized this, health authorities around the world began adding iodine to table salt.

This strategy was effective in correcting iodine deficiency. But it had an unanticipated— and undesired— effect. In countries where iodine has been added to table salt, the rates of autoimmune thyroid disease have risen.

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Why does this happen? Because increased iodine intake, especially in supplement form, can increase the autoimmune attack on the thyroid. Iodine reduces the activity of an enzyme called thyroid peroxidase (TPO). TPO is required for proper thyroid hormone production.

On the other hand, restricting intake of iodine can reverse hypothyroidism. In one study, 78% of patients with Hashimoto's regained normal thyroid function with iodine restriction alone. However—and this is a big "however"— it appears that iodine may only pose a problem for people with Hashimoto's and other autoimmune thyroid diseases in the presence of concurrent selenium deficiency. In the study above where rats developed goiter while receiving excess iodine, when they were given adequate selenium they did not develop the goiter.

Other studies have shown that selenium protects against the effects of iodine toxicity and prevents the triggering and flaring of autoimmune disease that excess iodine without selenium can cause. In my practice I always test for both iodine deficiency and selenium when a patient presents with hypothyroid symptoms. If they are iodine deficient, I will start them on a trial of iodine and selenium together. In most cases, patients see a significant improvement. In a minority of cases, they cannot tolerate supplemental iodine even with adequate selenium intake.

Unfortunately, the blood test for iodine that your doctor might run is not very accurate. The best way to determine iodine status is with a 24 or 16-hour urine loading test. This involves taking a large dose of iodine and collecting your urine for 24 or 16 hours afterward. If you are iodine deficient, you'll retain more of the ingested iodine than you should and the level of iodine excreted in the urine will be lower than expected. Nutripath do this test in Australia.

That said, if your doctor or health care practitioner won't order these tests, you can simply begin an iodine protocol. This involves starting with a low dose of iodine (I start my patients with kelp tablets called Modiflan that contain 325mcg of iodine per capsule) and increasing very slowly over time. As I've described in this article, it's crucial that you also take 200mcg of selenium per day during this protocol to protect against the potentially adverse effects of iodine supplementation, especially if you have autoimmune thyroid disease.

Physicians that specialize in treating hypothyroidism with iodine (such as Dr. Abraham and Dr. Brownstein) suggest doses as high as 50mg per day may be necessary to restore iodine levels in those that are deficient. I have used doses this high in my practice, but it's imperative that patients build up to such high doses very slowly, and I don't recommend doing it without the supervision of a clinician experienced with iodine treatment. Be aware that high doses of iodine can lead to a transient increase in TSH levels, which can be mistakenly interpreted as a sign of hypothyroidism.

Finally, it's important to keep in mind that a minority of patients with Hashimoto's confirmed by biopsy (the gold standard) never test positive for thyroid antibodies. This is probably because their immune systems are so depressed they can no longer produce antibodies. If you have a combination of hyper- and hypothyroid symptoms, I would still suspect Hashimoto's even if your thyroid antibody tests are normal. It's wise to be cautious with iodine if you have any signs of autoimmune thyroid disease, even without a confirmed diagnosis.

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Selenium - The missing link for treating hypothyroidism?

Selenium deficiency is not thought to be common in healthy adults, but is more likely to be found in those with digestive health issues causing poor absorption of nutrients, such as Crohn's or celiac disease, or those with serious inflammation due to chronic infection. (4, 5) It is thought that selenium deficiency does not specifically cause illness by itself, but that it makes the body more susceptible to illnesses caused by other nutritional, biochemical or infectious stresses, due to its role in immune function. Adequate selenium nutrition supports efficient thyroid hormone synthesis and metabolism and protects the thyroid gland from damage from excessive iodine exposure.

Several research studies have demonstrated the benefits of selenium supplementation in treating autoimmune thyroid conditions. One study found that selenium supplementation had a significant impact on inflammatory activity in thyroid-specific autoimmune disease, and reducing inflammation may limit damage to thyroid tissue. This may be due to the increase in glutathione peroxidase and thioredoxin reductase activity, as well as the decrease in toxic concentrations of hydrogen peroxide and lipid hydroperoxides which result from thyroid hormone synthesis.

Another study followed patients for 9 months, and found that selenium supplementation reduced thyroid peroxidase antibody levels in the blood, even in selenium sufficient patients. While these studies show promise for the use of selenium supplementation in preventing thyroid tissue damage, further research is needed to determine the long-term clinical effects of selenium treatment on inflammatory autoimmune thyroiditis.

Additionally, selenium is also essential for the conversion of T4 to T3, as deiodinase enzymes (those enzymes that remove iodine atoms from T4 during conversion) are selenium-dependent. As I've explained before, T3 is the active form of thyroid hormone, and low T3 can cause hypothyroid symptoms. A double-blind intervention study found that selenium supplementation in selenium deficient subjects modulated T4 levels, theoretically by improving peripheral conversion to T3.

In cases of severe selenium deficiency, conversion of T4 to T3 may be impaired, leading to hypothyroid symptoms. As T3 conversion is not performed by the thyroid, the dependence on selenoproteins for this conversion demonstrates how significant selenium deficiency could lead to hypothyroid symptoms.

So the question is, should you start supplementing with selenium if you have hypothyroidism, Hashimoto's thyroiditis, or low T3 levels?

As the answer often is, it depends. These preliminary studies show the positive effects of selenium supplementation on inflammatory activity in autoimmune thyroid conditions, but the long term effects of supplementation on thyroid health are still unknown. And we know that selenium is an essential component of the enzymes that convert T4 to T3, but whether supplementation will increase serum T3 levels is unclear.

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For now, the best option for most people may be to include selenium-rich foods in the context of a healthy Paleo diet. Great sources of selenium include: brazil nuts, crimini mushrooms, cod, shrimp, tuna, halibut, salmon, scallops, chicken, eggs, shiitake mushrooms, lamb, and turkey. For those concerned with the high level of omega-6 fats in brazil nuts, it may be worth considering the fact that it only takes one or two brazil nuts per day to improve your selenium status and boost immune function.

For those who choose to supplement, I consider 100-200 micrograms of selenium to be a safe supplemental dose for people with thyroid issues. We compound this in a liquid or capsule often with other important Thyroid minerals.

Making sure your selenium intake is optimal may give your immune system and thyroid the boost it needs to help it function better. Whether through selenium-rich foods or supplements, it is especially important for those managing thyroid conditions to ensure their selenium status is adequate.

Thyroid, Blood Sugar, and Metabolic Syndrome

Metabolic syndrome (MetS), also affects 3 million Australians, and insulin resistance, one of the components of metabolic syndrome. That's a large 7% of the population. Metabolic syndrome has become so common that it's predicted to eventually bankrupt our healthcare system. Both metabolic syndrome and insulin resistance are risk factors for heart disease and diabetes, two of the leading causes of death in the developed world.

With such a high prevalence of both thyroid dysfunction and metabolic syndrome, you might suspect there's a connection between the two. And you'd be right.

Studies show an increased frequency of thyroid disorders in diabetics, and a higher prevalence of obesity and metabolic syndrome in people with thyroid disorders.

That's because healthy thyroid function depends on keeping your blood sugar in a normal range, and keeping your blood sugar in a normal range depends on healthy thyroid function.

HOW HIGH BLOOD SUGAR AFFECTS THE THYROID

Metabolic syndrome is defined as a group of metabolic risk factors appearing together, including:

- abdominal obesity
- high cholesterol and triglycerides
- high blood pressure
- insulin resistance
- tendency to form blood clots
- inflammation

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Metabolic syndrome is caused by chronic hyperglycemia (high blood sugar). Chronic hyperglycemia is caused by eating too many carbohydrates. Therefore, metabolic syndrome could more simply be called "excess carbohydrate disease". In fact, some researchers have gone as far as defining metabolic syndrome as "those physiologic markers that respond to reduction in dietary carbohydrate."

When you eat too many carbs, the pancreas secretes insulin to move excess glucose from the blood into the cells where glucose is used to produce energy. But over time, the cells lose the ability to respond to insulin. It's as if insulin is knocking on the door, but the cells can't hear it. The pancreas responds by pumping out even more insulin (knocking louder) in an effort to get glucose into the cells, and this eventually causes insulin resistance.

Studies have shown that the repeated insulin surges common in insulin resistance increase the destruction of the thyroid gland in people with autoimmune thyroid disease. As the thyroid gland is destroyed, thyroid hormone production falls. .

HOW LOW BLOOD SUGAR AFFECTS THE THYROID

But just as high blood sugar can weaken thyroid function, chronically low blood sugar can also cause problems.

Your body is genetically programmed to recognize low blood sugar as a threat to survival. Severe or prolonged hypoglycemia can cause seizures, coma, and death. When your blood sugar levels drop below normal, your adrenal glands respond by secreting a hormone called cortisol. Cortisol then tells the liver to produce more glucose, bringing blood sugar levels back to normal.

The problem is that cortisol (along with epinephrine) is also a sympathetic nervous system hormone involved in the "flight or fight" response. This response includes an increase in heart rate and lung action and an increase in blood flow to skeletal muscles to help us defend against or flee from danger. Cortisol's role is to increase the amount of glucose available to the brain, enhance tissue repair, and curb functions - like digestion,

growth and reproduction - that are non-essential or even detrimental in a fight or flight situation.

Unfortunately for hypoglycemics, repeated cortisol release caused by episodes of low blood sugar suppresses pituitary function. And as I showed in a previous article, without proper pituitary function, your thyroid can't function properly.

Together, hyperglycemia and hypoglycemia are referred to as dysglycemia. Dysglycemia weakens and inflames the gut, lungs and brain, imbalances hormone levels, exhausts the adrenal glands, disrupts detoxification pathways, and impairs overall metabolism. Each of these effects significantly weakens thyroid function. As long as you have dysglycemia, whatever you do to fix your thyroid isn't going to work.

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HOW LOW THYROID FUNCTION AFFECTS BLOOD SUGAR

We've seen now how both high and low blood sugar cause thyroid dysfunction. On the other hand, low thyroid function can cause dysglycemia and metabolic syndrome through a variety of mechanisms:

- it slows the rate of glucose uptake by cells
- it decreases rate of glucose absorption in the gut
- it slows response of insulin to elevated blood sugar
- it slows the clearance of insulin from the blood

These mechanisms present clinically as hypoglycemia. When you're hypothyroid, your cells aren't very sensitive to glucose. So although you may have normal levels of glucose in your blood, you'll have the symptoms of hypoglycemia (fatigue, headache, hunger, irritability, etc.). And since your cells aren't getting the glucose they need, your adrenals will release cortisol to increase the amount of glucose available to them. This causes a chronic stress response, as I described above, that suppresses thyroid function.

HOW TO KEEP YOUR BLOOD SUGAR IN A HEALTHY RANGE

It's important to understand that whether you have high or low blood sugar, you probably have some degree of insulin resistance. I described how high blood sugar causes insulin resistance above. But insulin resistance can also cause low blood sugar. This condition, called reactive hypoglycemia, occurs when the body secretes excess insulin in response to a high carbohydrate meal - causing blood sugar levels to drop below normal.

In either case, the solution is to make sure your blood sugar stays within a healthy range. There are two targets to consider. The first is fasting blood glucose, which is a measure of your blood sugar first thing in the morning before eating or drinking anything. I define the normal range for fasting blood glucose as 75 - 95mg/dL.

Although 100 is often considered the cut-off for normal, studies have shown that fasting blood sugar levels in the mid-90s were predictive of future diabetes a decade later. And although 80mg/dL is often defined as the cut-off on the low end, plenty of healthy people have fasting blood sugar in the mid-to-high 70s (especially if they follow a low-carb diet).

The second, and much more important, target is post-prandial blood glucose. This is a measure of your blood sugar 1-2 hours after a meal. Several studies have shown that post-prandial blood glucose is the most accurate predictor of future diabetic complications and is the first marker (before fasting blood glucose and Hb1Ac) to indicate dysglycemia.

I highly recommend you pick up a blood glucose meter if you have a thyroid and/or blood sugar problem. It's the simplest and most cost-effective way to figure out how much carbohydrate is safe for you to eat. There are tons of meters out there,

Finally, if you have poor thyroid function it's important that you take steps to normalize it. As I've described in this article, the cycle works in both direction. Dysglycemia can depress thyroid function, but thyroid disorders can cause dysglycemia and predispose you to insulin resistance and metabolic syndrome.

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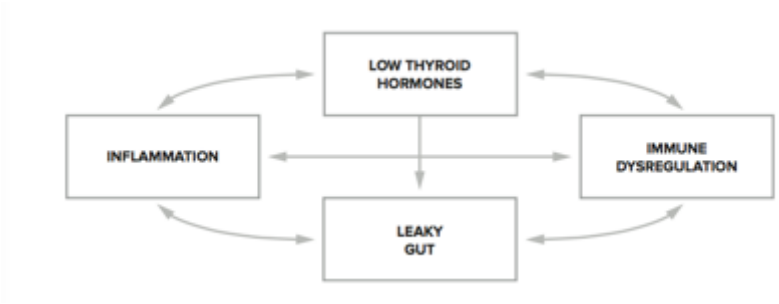
The Thyroid-Gut Connection

Hippocrates said: "All disease begins in the gut." 2,500 years later we're just beginning to understand how right he was. Hypothyroidism is no exception to this rule. Poor gut health can suppress thyroid function and trigger Hashimoto's disease, and low thyroid function can lead to an inflamed and leaky gut - as illustrated in the following diagram:



Have you ever considered the fact that the contents of the gut are outside the body? The gut is a hollow tube that passes from the mouth to the anus. Anything that goes in the mouth and isn't digested will pass right out the other end. This is, in fact, one of the most important functions of the gut: to prevent foreign substances from entering the body.

Another important function of the gut is to host 70% of the immune tissue in the body. This portion of the immune system is collectively referred to as GALT, or gut-associated lymphoid tissue. The GALT comprises several types of lymphoid tissues that store immune cells, such as T & B lymphocytes, that carry out attacks and produce antibodies against antigens, molecules recognized by the immune system as potential threats.

Problems occur when either of these protective functions of the gut are compromised. When the intestinal barrier becomes permeable (i.e. "leaky gut syndrome"), large protein molecules escape into the bloodstream. Since these proteins don't belong outside of the gut, the body mounts an immune response and attacks them. Studies show that these attacks play a role in the development of autoimmune diseases like Hashimoto's.

We also know that thyroid hormones strongly influence the tight junctions in the stomach and small intestine. These tight junctions are closely associated areas of two cells whose membranes join together to form the impermeable barrier of the gut. T3 and T4 have been shown to protect gut mucosal lining from stress induced ulcer formation. In another study, endoscopic examination of gastric ulcers found low T3, low T4 and abnormal levels of reverse T3.

Likewise, thyrotropin releasing hormone (TRH) and thyroid stimulating hormone (TSH) both influence the development of the GALT. T4 prevents over-expression of intestinal intraepithelial lymphocytes (IEL), which in turn causes inflammation in the gut.

One little known role of the gut bacteria is to assist in converting inactive T4 into the active form of thyroid hormone, T3. About 20 percent of T4 is converted to T3 in the GI tract, in the forms of T3 sulfate (T3S) and triidothyroacetic acid (T3AC). The conversion of T3S and T3AC into active T3 requires an enzyme called intestinal sulfatase.

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Where does intestinal sulfatase come from? You guessed it: healthy gut bacteria. Intestinal dysbiosis, an imbalance between pathogenic and beneficial bacteria in the gut, significantly reduces the conversion of T3S and T3AC to T3. This is one reason why people with poor gut function may have thyroid symptoms but normal lab results.

Inflammation in the gut also reduces T3 by raising cortisol. Cortisol decreases active T3 levels while increasing levels of inactive T3.

Studies have also shown that cell walls of intestinal bacteria, called lipopolysaccharides (LPS), negatively effect thyroid metabolism in several ways. LPS:

- reduce thyroid hormone levels
- dull thyroid hormone receptor sites
- increase amounts of inactive T3
- decrease TSH
- promote autoimmune thyroid disease (AITD)

Hypochlorhydria, or low stomach acid, increases intestinal permeability, inflammation and infection (for more on this, see the free eBook on heartburn and GERD, which you can access in the membership area of my website). Studies have shown a strong association between atrophic body gastritis, a condition related to hypochlorhydria, and autoimmune thyroid disease. Constipation can impair hormone clearance and cause elevations in estrogen, which in turn raises thyroid-binding globulin (TBG) levels and decreases the amount of free thyroid hormones available to the body. On the other hand, low thyroid function slows transit time, causing constipation and increasing inflammation, infections and malabsorption. Finally, a sluggish gall bladder interferes with proper liver detoxification and prevents hormones from being cleared from the body, and hypothyroidism impairs GB function by reducing bile flow. All of these connections make it clear that you can't have a healthy gut without a healthy thyroid, and you can't have a healthy thyroid without a healthy gut. To restore proper function of the gut-thyroid axis, both must be addressed simultaneously. Healing the gut is a huge topic that can't be covered adequately in a few short sentences. But I will say this: the first step is always to figure out what's causing the gut dysfunction. Low thyroid is one possible cause, but often hypochlorhydria, infections, dysbiosis, food intolerances (especially gluten), stress and other factors play an even more significant role. The second step is to address these factors and remove any potential triggers. The third step is to restore the integrity of the gut barrier. My preferred approach for this last step is the GAPS diet.

The influence of thyroid hormones on the gut is one of many reasons why I recommend that people with persistently high TSH and low T4 and T3 take replacement hormones. Low thyroid hormones make it difficult to heal the gut, and an inflamed and leaky gut contributes to just about every disease there is, including hypothyroidism. Fixing the gut is often the first - and most important - step I take with my patients.

THYROID DISORDERS

5 Ways That Stress Causes Hypothyroid Symptoms

We've already talked about how blood sugar imbalances and poor gut health can lead to hypothyroidism and Hashimoto's. The harmful effects of adrenal stress complete the triad.

The adrenals are two walnut-shaped glands that sit atop the kidneys. They secrete hormones - such as cortisol, epinephrine and norepinephrine - that regulate the stress response. But these hormones play other crucial roles, many of which are directly related to thyroid health. In fact, proper thyroid function depends on healthy adrenal glands.

Most people are aware of the obvious forms of stress that affect the adrenal glands: impossibly full schedules, driving in traffic, financial problems, arguments with a spouse, losing a job and the many other emotional and psychological challenges of modern life.

But other factors not commonly considered when people think of "stress" place just as much of a burden on the adrenal glands. These include blood sugar swings, gut dysfunction, food intolerances (especially gluten), chronic infections, environmental toxins, autoimmune problems and inflammation. All of these conditions sound the alarm bells and cause the adrenals to pump out more stress hormones. In this context, stress is broadly defined as anything that disturbs the body's natural balance (homeostasis).

Adrenal stress is probably the most common problem we encounter in functional medicine, because nearly everyone is dealing with at least one of the factors listed above. Symptoms of adrenal stress are diverse and nonspecific, because the adrenals affect every system in the body. But some of the more common symptoms are:

- Fatigue
- Headaches
- Decreased immunity
- Difficulty falling asleep, staying asleep and waking up
- Mood swings
- Sugar and caffeine cravings
- Irritability or light headedness between meals
- Eating to relieve fatigue
- Dizziness when moving from sitting or lying to standing
- Gastric ulcers

Weak adrenals can cause hypothyroid symptoms without any problem in the thyroid gland itself. In such cases, treating the thyroid is both unnecessary and ineffective, and addressing the adrenals themselves is the key to improving thyroid function.

The most significant indirect effect the adrenals have on thyroid function is via their influence on blood sugar. High or low cortisol - caused by any of the chronic stressors listed above - can cause hypoglycemica, hyperglycemia or both. And blood sugar imbalances can actually cause hypothyroid symptoms in a variety of ways.

But adrenal stress also has more direct impacts on thyroid function. The following five mechanisms are the most important.

THYROID DISORDERS

1. ADRENAL STRESS DISRUPTS THE HPA AXIS

By now many people have heard of the hypothalamic-pituitary-adrenal (HPA) axis. It's a complex network of interactions between the hypothalamus, the pituitary and the adrenal glands that regulates things such as temperature, digestion, immune system, mood, sexuality and energy usage - in addition to controlling the body's reaction to stress and trauma.

Countless studies show that chronic adrenal stress depresses hypothalamic and pituitary function. And since these two organs direct thyroid hormone production, anything that disrupts the HPA axis will also suppress thyroid function.

Studies have shown that the inflammatory cytokines IL-1 beta, IL-6 and TNF-alpha, which are released during the stress response, down-regulate the HPA axis and reduce levels of thyroid stimulating hormone (TSH). Another study showed that one single injection of tumor necrosis factor alpha (TNF-alpha), an inflammatory peptide, reduced serum TSH, T3, free T4, free T3 and hypothalamic TRH for 5 days. TNF-alpha was also found to decrease the conversion of T4 to T3, reduce thyroid hormone uptake, and decrease the sensitivity of the thyroid to TSH.

2. ADRENAL STRESS REDUCES CONVERSION OF T4 TO T3

We discussed under-conversion of T4 to T3 before. Remember that although 93% of the hormone produced by the thyroid gland is T4, it is inactive in that form and must be converted into T3 before it can be used by the cells. The inflammatory cytokines I listed above not only disrupt the HPA axis, they also interfere with the conversion of T4 to T3.

The enzyme 5 alpha-deiodinase catalyzes the conversion of T4 into T3 in peripheral tissues such as the liver and the gut. Both Th1 and Th2 inflammatory cytokines - IL-6, TNF-alpha, IFN-gamma and IL-1 beta - have been shown to suppress the conversion of T4 to T3. In patients without thyroid illness, as levels of IL-6 (a marker for inflammation) rise, levels of serum T3 fall. And injections of inflammatory cytokines into healthy human subjects resulted in a rapid reduction of serum T3 and TSH levels, and an increase in the inactive reverse T3 (rT3) form, while T4 and free T4 levels were only minimally changed.

3. ADRENAL STRESS PROMOTES AUTOIMMUNITY BY WEAKENING IMMUNE BARRIERS

The GI tract, lungs and the blood-brain barrier are the primary immune barriers in the body. They prevent foreign substances from entering the bloodstream and the brain. Adrenal stress weakens these barriers, weakens the immune system in general, and promotes poor immune system regulation.

As we discussed earlier, when these immune barriers are breached large proteins and other antigens are able to pass into the bloodstream or brain where they don't belong. If this happens repeatedly, the immune system gets thrown out of whack and we become more prone to autoimmune diseases - such as Hashimoto's.

THYROID DISORDERS

4. ADRENAL STRESS CAUSES THYROID HORMONE RESISTANCE

In order for thyroid hormone circulating in blood to have a physiological effect, it must first activate receptors on cells. Inflammatory cytokines have been shown to suppress thyroid receptor site sensitivity.

If you're familiar with insulin resistance, where the cells gradually lose their sensitivity to insulin, this is a similar pattern. It's as if the thyroid hormone is knocking on the cell's door, but the cells don't answer.

While there's no practical way to measure receptor site sensitivity in a clinical setting, the research above suggests it is decreased in autoimmune and other inflammatory conditions. A perfect example of this in practice is the Hashimoto's patient who is taking replacement hormones but still suffers from hypothyroid symptoms - often in spite of repeated changes in the dose and type of medication. In these patients, inflammation is depressing thyroid receptor site sensitivity and producing hypothyroid symptoms, even though lab markers like TSH, T4 and T3 may be normal

5. ADRENAL STRESS CAUSES HORMONAL IMBALANCES

Cortisol is one of the hormones released by the adrenals during the stress response. Prolonged cortisol elevations, caused by chronic stress, decrease the liver's ability to clear excess estrogens from the blood. Excess estrogen increases levels of thyroid binding globulin (TBG), the proteins that thyroid hormone is attached to as it's transported through the body.

When thyroid hormone is bound to TBG, it is inactive. It must be cleaved from TBG to become "free-fraction" before it can activate cellular receptors. (These free-fraction thyroid hormones are represented on lab tests as "free T4 [FT4]" and "free T3 [FT3]".)

When TBG levels are high, the percentage of free thyroid hormones drops. This shows up on labs as low T3 uptake and low free T4/T3.

Aside from adrenal stress, the most common causes of elevated TBG secondary to excess estrogen are birth control pills and estrogen replacement (i.e. Premarin).

Here's the tricky thing about adrenal stress: it's almost always caused - at least in part - by something else. These causes include anemia, blood sugar swings, gut inflammation, food intolerances (especially gluten), essential fatty acid deficiencies, environmental toxins, and of course, chronic emotional and psychological stress.

When they exist, these conditions must be addressed or any attempt to support the adrenals directly will either fail or be only partially successful. With that in mind, here are some general guidelines for adrenal health:

- Avoid or at least greatly minimize stimulants
- Stabilize blood sugar (via a moderate or low-carb diet)
- Practice stress management and relaxation techniques
- Have fun, laugh and make pleasure a regular part of your life
- Avoid dietary causes of inflammation (refined flours, high-fructose corn syrup and industrial seed oils in particular)
- Ensure adequate intake of DHA & EPA

THYROID DISORDERS

Specific nutrients such as phosphatidyl serine and adaptogenic botanicals like Panax ginseng, Siberian ginseng, Ashwagandha and Holy basil leaf extract are also helpful in modulating the stress response and supporting the adrenals. However, these are potent medicines and should be taken under the supervision of a trained practitioner.

Why Thyroid Medication is Often Necessary

I've been critical of pharmaceutical approaches in the past, and in general, I recommend avoiding the use of medication whenever possible. However, I have no problem with pharmaceuticals if:

1. they work,
2. they do more good than harm, and;
3. there are no non-drug alternatives with the same effect.

It turns out that thyroid medication meets these criteria in cases of hypothyroidism with chronically elevated TSH. Elevated TSH indicates that the body is not producing enough thyroid hormone to meet metabolic needs. And thyroid hormone is so important to the proper function of the body that the benefits of replacing it far outweigh any potential side effects of the medication.

Remember that every cell in the body has receptor sites for thyroid hormone. Thyroid hormones are responsible for the most basic and fundamental aspect of physiology: the basal metabolic rate. Since the basal metabolic rate affects every system of the body, low thyroid hormone causes a global decline in cellular function.

Here's a list of things that can go wrong when thyroid hormones are low. It's not complete, but it should give you some idea of how important the thyroid is to proper function.

- Decreased energy production and metabolism in all cells of the body
- Decreased bone quality and increase in fractures
- Elevated cholesterol
- Impaired phase II detoxification
- Anemia
- Decreased stomach acid production
- Constipation, intestinal dysbiosis, malabsorption
- Intestinal inflammation
- Blood sugar imbalances
- Gallstone formation
- Vascular and arterial plaquing
- Neurodegeneration, cognitive problems, depression
- Weight gain
- Hair loss
- Dry skin
- Cold hands and feet
- Infertility and reproductive dysfunction
- Weakened immune system

THYROID DISORDERS

I could go on, but I think you get the point. If your thyroid hormones are low, you can't be healthy. Period. 90% of people with hypothyroidism in the U.S. have Hashimoto's disease. As I explained before, Hashimoto's is an autoimmune condition that causes destruction of the thyroid gland over time. As this destruction progresses, the thyroid gland becomes less and less able to produce enough hormones to meet metabolic needs. This is reflected in an increase in thyroid-stimulating hormone (TSH). Persistently elevated TSH is a sign that the body needs more thyroid hormone than it can produce on its own. This is one clear sign that it's time for replacement medication. But it isn't the only one. Some people with TSH in the normal lab range still find that they benefit from replacement.

Note that I'm not saying everyone with hypothyroid symptoms should be on medication. Earlier, I discussed 5 different patterns of low thyroid function that present with normal TSH levels. These include underconversion of T4 to T3, problems with thyroid binding proteins, pituitary dysfunction and thyroid receptor-site resistance. In these cases, the problem isn't with the thyroid gland itself or its ability to produce enough hormones, but is either "upstream" (in the case of pituitary dysfunction) or "downstream" (in the case of conversion problems, binding protein issues or resistance.) For these patterns, replacement hormones are often unnecessary.

There are many in my profession (natural healthcare) that vehemently oppose the use of medication under any circumstances. I think that's foolish. I'm more concerned about the dangers of Big Pharma than most. But that doesn't mean we should ignore the important role drugs play in treating certain conditions.

In fact, my philosophy on healthcare can be simply stated as: whatever works best and causes the least harm. It' not often that a drug fits the bill. But in the case of hypothyroidism with elevated TSH, I believe replacement medication is a necessary part of a larger strategy that includes balancing blood sugar, adrenals and the immune system and fixing the gut.

Three Reasons Why Your Thyroid Medication Isn't Working

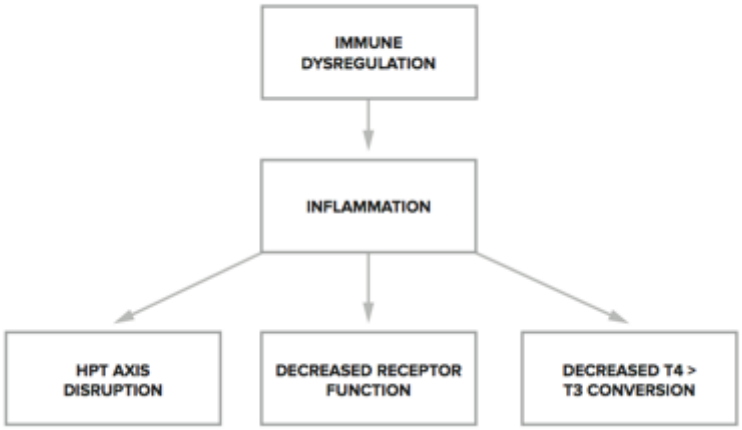
The ultimate effect of hypothyroidism, whether it's caused by iodine deficiency or autoimmunity, is to decrease the amount of thyroid hormone available to the body. The conventional approach is to simply replace these hormones with either synthetic or bio-identical forms.

On the surface it seems like a reasonable approach. Patient doesn't have enough hormones? Give more hormones. Simple, right?

Not so much. Once again the conventional approach falls short because it ignores the underlying cause of the problem. It's like taking Advil when you've got a pebble stuck in your shoe. It might work for a little while, and might even be necessary to dull the pain. But you'd be a lot better off if you took the pebble out of your shoe. Right?

THYROID DISORDERS

Let's take a closer look at why thyroid hormones often don't work, or stop working over time. The following diagram illustrates how autoimmunity affects thyroid metabolism:



Immune dysregulation is another term for autoimmune disease. We still don't know exactly what causes it, but most researchers agree it's a mixture of genetic susceptibility and environmental factors such as iodine (excess), infection, pregnancy, diet and intestinal permeability.

In autoimmune disease the body attacks itself. It does this the same way it attacks foreign invaders like bacteria and viruses: with T-cells, B-cells, natural killer cells, and cytotoxic T cells. The immune response also involves proteins called cytokines, chemical messengers that pass messages between cells.

This self-attack by the immune system increases inflammation. And inflammation has a profound effect on all aspects of thyroid metabolism and physiology.

First, inflammation suppresses the hypothalamus-pituitary-thyroid (HPT) axis. One study showed a single injection of the inflammatory cytokine TNF-alpha reduced blood levels of TSH, T3, free T4, free T3 and TRH for 5 days. This shows inflammation disrupts the production and regulatory mechanisms of thyroid hormones. Thyroid medication will increase the levels of T4 (and possibly T3), but it doesn't address the other effects of HPT axis suppression.

Second, inflammation decreases both the number and sensitivity of thyroid hormone receptors. If there aren't enough receptors, or they aren't sensitive enough, it doesn't matter how much thyroid medication we take. The cells won't be able to use it. It's like when my grandpa used to turn down his hearing aids while he was watching the football game. It didn't matter how much my grandma yelled at him - he couldn't hear a word she said.

Third, inflammation decreases the conversion of T4 to T3. T4 is the inactive form of thyroid hormone. The body has to convert it to the active T3 form before it can be used. Most synthetic hormone medications on the market are T4. If you give a T4 medication (like Synthroid, Levoxyl, Unithroid, etc.) to someone with inflammation, it's not going to work because they can't convert the T4 to T3.

Patients who don't convert T4 to T3 well do better on bio-identical hormones like Armour, because it contains both T4 and T3 (in a 4.22:1 ratio).

Inflammation disrupts thyroid metabolism in several other ways, but I think these three examples make the point.

Now let's review.

Inflammation causes HPT axis disruption, decreased receptor function, and decreased conversion of T4 to T3. Thyroid medication only increases the levels of thyroid hormone (usually T4) in the blood. No matter how much we take, it's not going to restore HPT axis coordination, improve receptor function, or increase conversion of T4 to T3.

The only way to do that is to address the problem at its root by regulating the immune system and decreasing inflammation. Unfortunately, this is rarely done in either conventional or alternative treatment of thyroid disorders.

I do want to make one thing clear. I'm not saying thyroid medication isn't necessary or useful. In fact, I think it's an important part of treating Hashimoto's - especially when TSH is consistently elevated and T4 and T3 are consistently low. My point is thyroid medication is only one piece of the puzzle, and it won't be effective on its own unless the autoimmunity and inflammation are addressed.

Basics of Immune Balancing for Hashimoto's

In order to treat all causes of Hashimoto's disease, it's important to bring the immune system back into balance using specific diet and lifestyle changes. Although I'm about to offer some general guidelines here for how to balance the immune system, if you have Hashimoto's (or any other autoimmune condition) it's in your best interest to find someone who understands immunology and is current with the latest nutritional and botanical protocols for treating autoimmune disease.

Why? Because autoimmune disease is not only extremely complex, but also highly individualized. Hashimoto's in one person is not the same as Hashimoto's in the next person. In one person, Hashimoto's could present as a Th1-dominant condition. In another, it may present as Th2 dominant. In still another, both the Th1 and Th2 systems might be overactive, or underactive. And each of these cases requires a different approach. For example, botanicals like echinacea and astragalus stimulate the Th1 system. If someone with Th1 dominant Hashimoto's takes these herbs, they'll quite possibly get worse. On the other hand, antioxidants like green tea and Gotu Kola stimulate the Th2 system, and would be inappropriate for those with Th2 dominant Hashimoto's. (For more information on the specifics of Hashimoto's autoimmune physiology, see this article on Dr. Kharrazian's blog and pick up a copy of his book.)

The good news, though, is that there are general approaches to balancing the immune system that are suitable for all types of Hashimoto's regardless of the specific pattern of immune dysregulation. These approaches can be broken into three categories: removing autoimmune triggers, enhancing regulatory T cell function and reducing inflammation.

THYROID DISORDERS

REMOVING AUTOIMMUNE TRIGGERS

We've already discussed the role of gluten, iodine, stress and a leaky gut in triggering an autoimmune response. Other potential triggers include estrogens, infectious agents, and environmental toxins.

Estrogen fluctuations can trigger the gene expression of Hashimoto's in the presence of inflammation and genetic susceptibility. In addition to turning on the genes associated with Hashimoto's, estrogen surges have been shown to exacerbate the autoimmune attack on the thyroid. This may explain why the expression of Hashimoto's is so common during pregnancy and perimenopause - both times when estrogen may be fluctuating wildly.

Environmental toxins are associated with autoimmune disease, and Hashimoto's is no exception. Certain antigens like mercury that bypass our barrier system cause a potent immune response that can become chronic and overactive. If you suspect environmental toxicity may be contributing to your condition, it's probably a good idea to get a test for chemical haptens and heavy metal antibodies.

Autoimmune thyroid disease has also been associated with a variety of infectious agents, including Rubella, Rubeolla, Epstein-Barr Virus, Retrovirus, Influenza B virus, Coxsakie virus and Yersinia. The mechanism in all cases is theorized to be cross-reaction between thyroid stimulating hormone (TSH) receptors and infectious agents. Once again, if you suspect an infectious agent is involved in your condition, a screening for these pathogens is a good idea.

ENHANCING REGULATORY T CELL FUNCTION

These strategies are all designed to enhance the function of regulatory T cells (also referred to as the Th3 system). Regulatory T cells are used to balance the activity between T-helper cells (Th1 & Th2) and T-suppressor cells (which "turn off" the immune attack).

Vitamin D has been shown to influence regulatory T cells, which in turn modulate T helper cell expression and balance the Th1 and Th2 response. For more on this see The Role of Vitamin D Deficiency in Thyroid Disorders.

The gut flora play a significant role in both cell-mediated (Th1) and humoral (Th2) immunity. Studies show that this protective role can be maintained and modulated by taking probiotics. Specific probiotic strains can influence the secretion of cytokines to help direct naïve helper T cells towards either a Th1 dominant, cell-mediated immune response or towards a Th2 dominant, humoral immune response.

Acupuncture has recently been shown to regulate the Th1 and Th2 immune response. In this study of patients with depression, both Prozac and acupuncture were shown to reduce inflammation. But only acupuncture restored the balance between the Th1 and Th2 systems. In another study, acupuncture reduced inflammation and lessened the symptoms of asthma by regulating the balance between Th1 and Th2 cytokines.

REDUCING INFLAMMATION

Essential fatty acids (EFAs) play an important role in preventing and reducing inflammation. I've written an entire series of articles on this topic, which I'd recommend reading if you haven't already.

The ideal ratio between omega-6 and omega-3 fatty acids is between 1:1 and 3:1. The average American ratio is closer to 25:1, and as high as 30:1, thanks to diets high in processed and refined foods. The result of this imbalance is - among other things - inflammation.

Two steps are required to bring this ratio back into balance. First, dramatically reducing consumption of omega-6 fats, and second, moderately increasing consumption of omega-3 fats. I explain how to do this in considerable detail in this article.

Another benefit of increasing intake of omega-3 fatty acids is that they have also been shown to help balance the Th1 and Th2 systems.

Aside from ensuring a proper balance of omega-3 and omega-6 fatty acids, following an anti-inflammatory diet/lifestyle and avoiding dietary triggers like gluten and iodine is essential.

Putting these general approaches to balancing the immune system into action should give you a good start towards getting the autoimmunity under control. But if you don't see the results you'd like, I'd recommend working with someone who knows how to address your particular immune imbalance more specifically.

3 Steps to Choosing the Right Thyroid Hormone

I often get comments and emails from people asking me which thyroid hormone I think is best. My answer is always the same: "It depends." As much as some practitioners would like to make us believe, there is simply no "one size fits all" approach to thyroid hormone replacement.

Statements like "Synthroid is best" or "I prefer to use synthetic T4 with my patients" or "I only use bio-identical hormones" demonstrate a lack of understanding of thyroid pathology. Why? Because, as I've explained in this eBook, the underlying causes of thyroid dysfunction are diverse.

Giving all patients the same thyroid medication without understanding the mechanisms involved is analogous to not checking a patient's blood type before doing a transfusion. Granted, the consequences may not be as severe, but the underlying principle is the same.

Before we continue, let me remind you that I'm not a doctor and I'm not offering you medical advice. My intent is to educate you about the various considerations that should be made when choosing a thyroid medication, so you can discuss them with your doctor. Understood? Great. Let's move on.

THYROID DISORDERS

Choosing the right thyroid medication requires answering the following three questions:

1. What's the mechanism that led to the need for medication in the first place?
2. Are there any mechanisms that may interfere with the actions of the medication?
3. Does the patient have sensitivities to the fillers used in the medications?

Let's look at each of these in turn.

WHAT'S THE MECHANISM THAT LED TO THE NEED FOR MEDICATION IN THE FIRST PLACE?

We now know that there's no single cause for low thyroid function. Do you have an autoimmune disease (Hashimoto's) causing destruction of your thyroid gland? Do you have high levels of estrogen causing an increase in thyroid binding proteins and a decrease in free thyroid hormone? Do you have a systemic inflammatory condition affecting your ability to convert T4 to T3, or decreasing the sensitivity of the cells in your body to thyroid hormone?

In order to choose the right hormone, you have to know what the underlying mechanism causing the dysfunction is. Let's look at an example.

Say you have a problem converting T4 to T3. In this situation, your TSH may or may not be slightly elevated, but let's say it is, and your doctor prescribes Synthroid. Synthroid is a synthetic T4 hormone. Will this help you?

No. It won't help because your problem in this example isn't a lack of T4, it's an inability to convert T4 to the active T3 form. You could take T4 all day long, and it won't do a thing unless your body can convert it.

The first step in this case would be to address the causes of the conversion problem (i.e. inflammation), in the hopes that you may not need replacement hormone. If that doesn't work, though, what you'd need in this situation is either a so-called bio-identical hormone that has a combination of T4 and T3, or a synthetic T3 hormone (like Cytomel). These will deliver the T3 you need directly, bypassing the conversion problem.

ARE THERE ANY MECHANISMS THAT MAY INTERFERE WITH THE ACTIONS OF THE MEDICATION?

The vast majority of long-term hypothyroid patients that haven't been properly managed find that they constantly need to increase the dose of their medication, or switch to new medications, to get the same effect.

There are several reasons for this. First, inflammation (which is characteristic of all autoimmune diseases, and Hashimoto's is no exception) causes a decrease in thyroid receptor site sensitivity. This means that even though you may be taking a substantial dose of replacement hormone, your cells aren't able to utilize it properly.

Second, elevations in either testosterone or estrogen (extremely common in hypothyroid patients) affect the levels of circulating free thyroid hormone. For example, high levels of estrogen will increase levels of thyroid binding protein. Thyroid hormone is inactive as long as it's bound to this protein. If you take thyroid replacement, but you have too much binding protein, there won't be enough of the active form to produce the desired effect.

THYROID DISORDERS

Third, there are several medications that alter the absorption or activity of T4. These include commonly prescribed drugs like antibiotics & antifungals (i.e. sulfonamides, rifampin, keoconazole), anti-diabetics (Orinase, Diabinese), diuretics (Lasix), stimulants (amphetamines), cholesterol lowering medications (Colestid, Atromid, LoCholest, Questran, etc.), anti-arrhythmia medications (Cordarone, Inderal, Propanolol, Regitine, etc.), hormone replacement (Premarin, anabolic steroids, growth hormone, etc.), pain medication (morphine, Kadian, MS Contin, etc.), antacids (aluminum hydroxides like Mylanta, etc.) and psychoactive medications (Lithium, Thorazine, etc.).

All of these factors must be considered if a particular medication isn't having the desired effect.

DOES THE PATIENT HAVE SENSITIVITIES TO THE FILLERS USED IN THE MEDICATIONS?

Another important consideration in choosing the right hormone is the fillers contained in each medication. Many popular thyroid medications contain common allergens such as cornstarch, lactose and even gluten. As I explained earlier, most hypothyroid patients have sensitivities to gluten, and many of them also react to corn and dairy (which contains lactose).

Synthroid, which is one of the most popular medications prescribed for hypothyroidism, has both cornstarch and lactose as a filler. Cytomel, which is a popular synthetic T3 hormone, has modified food starch - which contains gluten - as a filler.

Even the natural porcine products like Armour suffer from issues with fillers. In 2008, the manufacturers of Armour reformulated the product, reducing the amount of dextrose & increasing the amount of methylcellulose in the filler. This may explain the explosion of reports by patients on internet forums and in doctor's offices that the new form of Armour was either "miraculous" or "horrible". Those that had sensitivities to dextrose were reacting less to the new form, and experiencing better results, while those that had sensitivities to methylcellulose were reacting more, and experiencing worse results.

The best choice in these situations is to ask your doctor to have a compounding pharmacy fill the prescription using fillers you aren't sensitive to. Unfortunately, insurance companies sometimes refuse to cover this.

OTHER CONSIDERATIONS

Another common question that is hotly debated is whether bio-identical or synthetic hormones are best. Once again, the answer is: "It depends." In general I think bio-identical hormones are the best choice. A frequently perpetuated myth (in Synthroid marketing, for example) is that the dosages and ratio of T4:T3 in Armour aren't consistent. Studies have shown this to be false. Armour contains a consistent dose of 38mcg T4 and 9mcg T3 in a ratio of 4.22:1.

However, in some cases patients do feel better with synthetic hormones. One reason for this is that a small subset of people with Hashimoto's produce antibodies not only to their thyroid tissue (TPO and TG), but also to their own thyroid hormones (T4 and T3). These patients do worse with bio-identical sources because they increased the source of the autoimmune attack

Another issue is the use of T3 hormones. As we've discussed, T3 is the active form and has the greatest metabolic effects. The flip side of this, however, is that it's far easier to "overdose" on T3 than on T4. Patients with trouble converting T4 to T3 do well on synthetic T3 or bio-identical combination T4:T3 products. But for many patients with Hashimoto's, which is can present with alternating hypo- and hyperthyroid symptoms, T3 can push them over the edge. They are generally better off with T4 based drugs.

As you can see, the best thyroid hormone for each patient can only be determined by a full thyroid work-up and exam, followed by trial and error of different types of replacement medications. Such a work-up would include not just an isolated TSH test, but also a more complete thyroid panel (including antibodies), other important blood markers (glucose, lipids, CBC with diff, urinary DPD, etc.) and possibly a hormone panel. A history must be taken with particular attention paid to the patient's subjective response to replacement hormones they may have tried in the past.

Unfortunately, this rarely happens in the conventional model, where the standard of care is to test only for TSH. If it's elevated, the patient will get whatever hormone that particular practitioner is fond of using without any further investigation. All too often, as many of you can attest, this simplified and incomplete approach is doomed to failure.

And as we know, there are many other important components of a holistic thyroid treatment, including good nutrition and dietary changes.

Another Cause of Hypothyroidism: Low T3 Syndrome

Hypothyroidism involves high levels of thyroid stimulating hormone (TSH) and low levels of the thyroid hormones T4 and T3.

However, in my clinical practice I frequently see people with low levels of T3 with normal T4 and either low or normal TSH. This condition has been reported on in the medical literature for years but it is rarely acknowledged or discussed in conventional medical settings. Most doctors (even endocrinologists) do not seem to know what causes it, or what to do about it. (I know this because I always ask my patients with this syndrome what their doctors said about it, and my patients' response is almost always some variation of "not much").

This particular pattern goes by three different names in the medical literature: Euthyroid Sick Syndrome (ESS), Non-thyroidal Illness Syndrome (NTIS), and Low T3 Syndrome.

What's most important to understand about this condition is that, although it does involve low levels of T3 (the most active form of thyroid hormone), it is not caused by a problem with the thyroid gland. This is a crucial distinction and it's what distinguishes Low T3 Syndrome from "garden-variety" hypothyroidism.

In my series on Low T3 Syndrome, I discuss the causes of Low T3 Syndrome, its clinical significance, and if it should be treated, and if so, how. While discussing this type of hypothyroidism is outside of the scope of this eBook, I strongly recommend reading the series if you're still suffering from poor thyroid function even though your TSH and T4 values are normal.

THYROID DISORDERS

Conclusion

I hope that this eBook has been helpful in teaching you more about thyroid health and how to holistically treat a multitude of thyroid conditions. No two patients are exactly alike, and this is why I personally take a comprehensive approach to treating my patients with thyroid diseases.

Now that you've read this eBook, you'll be better equipped to discuss your options with your own doctor or practitioner, and to make the important lifestyle changes that will be crucial in optimizing your treatment success.

The Compounding Lab

We are able to compound preparations to assist in improving your Thyroid function. Classic thyroid symptoms are cold hands, feeling sluggish, thin hair, thin dry skin, constipation and a general unwell feeling. Extreme fibromyalgia and at the extreme chronic fatigue. We need to treat the symptoms as well as the disease.

Synthetic Hormones Vs. Desiccated Thyroid:

The traditional approach is to use synthetic hormones like Oroxine (levothyroxine). These products only contain T4 hormone, they have no T3. The common argument for using the synthetic T4 is it provides steady hormone levels. What is overlooked is that the vast majority of people cannot convert the T4 to the active form of thyroid that is T3. This is easy to confirm by measuring the free hormone levels. There are seven different reasons as to why the thyroid does not function and low conversion is common.

1. Pituitary (low TSH)
2. Low T4
3. Poor Conversion (T4 to T3)
4. Low TBP
5. High TBP
6. Insulin Resistance (Adrenals)
7. Autoimmune (Destruction of Thyroid)

Desiccated Thyroid - The Natural Alternative:

When one has low T3 levels, which are typical with synthetic hormone use, the brain often does not work properly. The symptom patients explain is foggy brain. It is important to use a preparation with T3 because T3 does 90% of the work of the thyroid in the body. This is when it's a place to use a combination of T4 and T3, which compensates for the inability to convert T4 to T3. Desiccated thyroid has both T3 and T4

Recently published in the New England Journal of Medicine, showed that the natural hormone product, such as amour, was far better at controlling the brain problems commonly found in hypothyroidism. Many integrative doctors tend to use desiccated thyroid which is a mixture of mono and di-iodothryonine and T3 and T4, the entire range of thyroid hormones. Desiccated thyroid dosing should be TWICE a day.

THYROID DISORDERS

Our natural thyroid capsules can also be prescribed in grains (gr) and are available in the many strengths for example:

- 16.25mg thyroid extract (¼ grain) contains 9.5mcg levothyroxine (T4) and 2.25mcg liothyronine (T3)
- 32.5mg thyroid extract (½ grain) contains 19mcg levothyroxine (T4) and 3.5mcg liothyronine (T3)
- 48.7mg thyroid extract (¾ grain) contains 28.5mcg levothyroxine (T4) and 6.75mcg liothyronine (T3)
- 65mg thyroid extract (1 grain) contains 38mcg levothyroxine (T4) and 9mcg liothyronine (T3)
- 130mg thyroid extract (2 grain) contains 76mcg levothyroxine (T4) and 18mcg liothyronine (T3)
- 195mg thyroid extract (3 grain) contains 114mcg levothyroxine (T4) and 27mcg liothyronine (T3)

The most common starting dose for patients with hypothyroidism is desiccated thyroid, 65mg a day which ideally is taken twice a day, 30 minutes after breakfast and dinner (32.5mg twice a day) or one hour before. Taking it after meals also helps to reduce volatility of the blood-level of T3.

Taking the desiccated thyroid twice a day overcomes traditional medicine's major objection and resistance to using natural thyroid preparations - its variability in its blood-levels. Most doctors using desiccated thyroid are not aware that natural thyroid should be used twice daily and NOT once a day. The major reason is that the T3 component has such a short half-life and needs to be taken twice daily to achieve consistent blood levels.

Initial dose for hypothyroid states, 60 to 300mg daily. Usual maintenance dose is 30 to 125mg daily, first thing in the morning.

Note: Desiccated thyroid 65mg is usually considered equivalent, levothyroxine sodium (T4) 100mcg or liothyronine sodium (T3) 25mcg.

Combined Synthetic T4/T3:

Only recently we have had the opportunity to compound a combined synthetic combination of T4/T3 into one capsule. This can allow for cost reductions and allows both hormones to be replaced in one capsule. Starting combination is 50mcg T4 / 12.5mcg T3.

Dose adjustments with lab monitoring:

Once on thyroid hormones the dose should be increased until the TSH falls below 0.4.

Then one needs to optimize the 2 thyroid hormones by using the Free T4 and Free T3 levels. It takes 4-6 weeks to see the maximal benefit once started.

The Free T3 and Free T4 are used to monitor the treatment. They should be above the median (middle) but below the upper end of the laboratory normal reference range. The goal for healthy young adults would be to have numbers close to the upper part of the range, and for cardiac and/or elderly patients, the numbers should be in the middle of its range.

The Free T3 and Free T4 levels should be checked every 2-3 months and the hormone therapy readjusted until the FT3 and FT4 levels are in the therapeutic range described. Once a therapeutic range is achieved the levels should be checked at least once a year. A small number of large, overweight, thyroid-resistant women may need larger dose of Natural Thyroid to see results.

THYROID DISORDERS

Autoimmune Thyroid (Hashimoto's):

Treating this is difficult as the T3 & T4 are irregular and the patient experiences from Hypo to Hyper Thyroid Symptoms. The patient needs Hydrocortisone to control the Inflammation and destruction of the Thyroid gland. The addition of Hydrocortisone to T4/T3 capsules of 1 - 5mg each morning allows a great improvement to their overall improvement.

Symptoms of Excessive Thyroid Hormone Patients need to know:

These are frequently but only temporary during the adaptation stage. The symptoms may include: palpitations, nervousness, feeling hot and sweaty, rapid weight-loss, fine tremor and clammy skin.

If patients can't tolerate desiccated hormones:

Over 90% of people do much better on desiccated thyroid. However, there are a small number of people who do not tolerate it. However, tertroxin, which is synthetic T3 only, can be used in combination with one of the T4 only synthetic preparations. It is important to recognize that T3 should always be prescribed twice daily due to its shorter half-life. This is typically after breakfast AND supper for compliance reasons. If patients are currently taking Oroxine (thyroxin), the Free T4 level is usually at or above the high end of its normal range and your Free T3 level is usually below. In this situation, one may then add 5 - 12.5mcg compounded T3 (pure-T3) after breakfast and supper daily, rather than desiccated thyroid. Once or twice daily dosing one can then optimize both the T4 and T3 levels, with whatever thyroid preparation is required. This is not possible in most hypothyroid patients with T4 only preparations.

People Who Should Not Take Compounded T3:

The only exception to using compounded thyroid hormones is in severe acute cardio-pulmonary conditions, such as congestive heart failure, when the metabolic slowing effect of a low FT3 level can actually be life-saving. However, the vast majority of hypothyroid patients do not have this problem.

All our Thyroid compounds are lactose and gluten free and can have a filler of choice such as tyrosine, magnesium, theanine or cellulose.

1. L-Thyroxine may be prescribed for hypothyroidism
   The dosage form available for L-Thyroxine is Capsule. We can compound more than 2,000 formulations in capsule form. The potency of each compounded capsule is verified through weight and yield checks before it is dispensed and mixed with precision in our RAM mixer.
2. Levothyroxine T4 may be prescribed for hypothyroidism
   The dosage form available for levothyroxine is capsule. We can compound more than 500 formulations in capsule form. The potency of each compounded capsule is verified through weight and yield checks before it is dispensed and mixed in our RAM mixer. Levothyroxine is also available in this dosage form. Different strengths may be available for different dosage forms such as Oral suspension.

THYROID DISORDERS

3. Levothyroxine Sodium/Liothyronine T4/T3 may be prescribed for Hypothyroidism
   The dosage form available for Levothyroxine Sodium/Liothyronine is a capsule. The potency of each compounded capsule is verified through weight and yield checks before it is dispensed. An example of strength combination of Levothyroxine Sodium/Liothyronine Capsule is available. Levothyroxine Sodium 16.2mcg/cap + Liothyronine Sodium 3.8mcg/cap.
4. Liothyronine (T3) may be prescribed for Hypothyroidism
   The dosage form available for Liothyronine is Capsule. The potency of each compounded capsule is verified through weight and yield checks before it is dispensed. Many strength combinations of Liothyronine Capsule, slow release are available.
   - Liothyronine (T3) 5mcg/cap
   - Liothyronine Sodium from 1.5mcg/cap to 30mcg/cap
5. Dessicated / Natural Thyroid NDT may be prescribed for Hypothyroidism. *My personal favourite.*
   The dosage form available for our Natural Dessicated Thyroid in compounds has more than 500 formulations in capsule form when dispensed. Starting Natural Thyroid Capsule is 15mg/cap increasing up to 190mg.

Click here for all our thyroid products.

Action Plan

1. CLEAN Fatigue & Thyroid Support (with activated BI = Benfotiamine) Order here
2. Book Oligoscan 07 3862 6000 or email enquiries@compoundinglab.com.au
3. Know your levels and download our blood tracker sheet on our website Click here
4. Clean Food Fast Inflammation Reduction Click here

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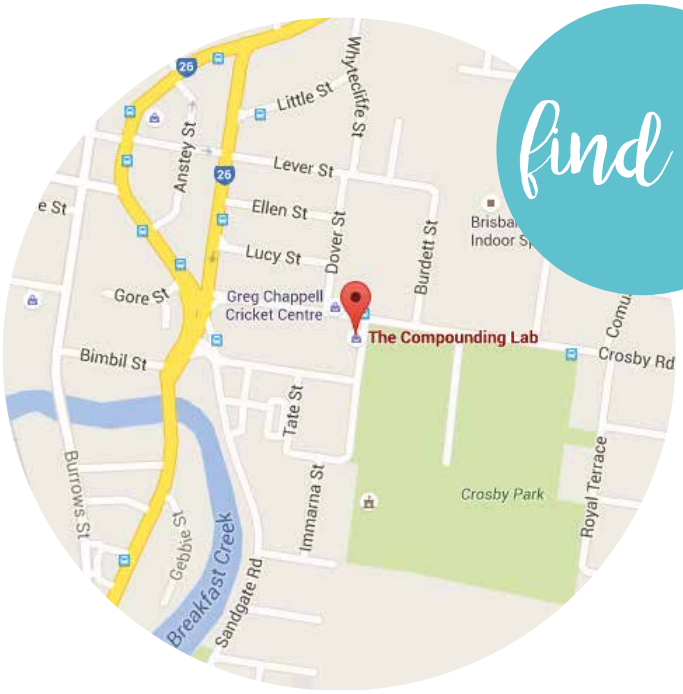
THYROID CONVERSION CHART

OUR Compounded Thyroid		Compounded NDT (Natural Dessicated Thyroid)	Oroxine/ Eutroxsig/ Eltroxin (Lactose Free)	Nature-Thyroid/ Westhroid	Tertroxin or Compounded
(Immediate Release Capsules, Sustained Release Capsules +/- Conversion Co-Factors)*		(Thyroid Capsules)	(Levothyroxine Tablets)	(Thyroid Tablets)	(Liothyronine Capsules)
Compounded		Desiccated, Porcine, Compounded	Synthetic	Desiccated, Porcine	Synthetic
T4 (4.2)	T3 (1)	T4/T3	T4	T4/T3	T3
15 mcg	2.25 mcg	16.25 mg	25 mcg (0.025mg)	¼ grain (16.25 mg)	5 mcg (only compounded capsules)
30 mcg	4.5 mcg	32.5 mg	50 mcg (0.05mg)	½ grain (32.5 mg)	
40 mcg	6.75 mcg	48.75 mg	75 mcg (0.075mg)	¾ grain (48.75 mg)	10 mcg (only compounded capsules)
50 mcg	7.92 mcg		88 mcg (0.088mg)		
60 mcg	9 mcg	65 mg	100 mcg (0.1mg)	1 grain (65 mg)	25 mcg
42.56 mcg	10.08 mcg		112 mcg (0.112mg)		
75 mcg	11.25 mcg	181.25 mg	125 mcg (0.125mg)	1 ¼ grain (81.25 mg)	
52.06 mcg	12.33 mcg		137 mcg (0.137mg)		
90 mcg	13.5 mcg	97.5 mg	150 mcg (0.15mg)	1 ½ grain (97.5 mg)	
66.5 mcg	15.75 mcg	113.75 mg	175 mcg (0.175mg)	1 ¾ grain (113.75 mg)	
120 mcg	18 mcg	130 mg	200 mcg (0.2mg)	2 grain (130 mg)	50 mcg
85.5 mcg	20.25 mcg	146.25 mg		2¼ grain (146.25 mg)	
150 mcg	22.5 mcg	162.5 mg		2 ½ grain (162.5 mg)	



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