Hashimoto's Thyroiditis, A Common Disorder in Women: How to Treat It – Part 2

by Thierry Hertoghe, MD

Editor Note: In Part 1 of this article (*Townsend Letter,* April 2020), Dr. Hertoghe discussed diagnosis, associated pathologies, and underlying causes of Hashimoto's thyroiditis. He also presented details about dietary measures that can reduce antithyroid antibody levels. Part 2 continues with more treatments for underlying causes.

Causes to Treat: Nutritional Deficiencies

Nutritional therapies to treat nutritional deficiencies may decrease thyroid antibody levels by 20-50%.

Selenium and vitamin D supplementations: Patients with Hashimoto's thyroiditis have been reported to have significantly lower selenium[315-316] and vitamin D[317-339] levels than controls without the disorder. On the other hand, daily selenium supplementation can decrease the levels of thyroid antibodies by 20 to 30%,[8,340-344] particularly if the level is low-normal or below the normal limit. The same is valid for vitamin D supplements.[345-354]

What are adequate doses? In adults, the dosage of selenium supplements should be at least 200 µg a day for several months and higher in the case of a noted deficiency in this trace element. Concomitant intake of 600 mg a day of myo-inositol has shown to increase the efficacy of selenium supplements to further reduce thyroid antibody levels.[8,343-344] Efficient doses of vitamin D3 supplements are 4000 IU a day at minimum, but higher doses between 10,000 IU (lean persons) to 20,000 IU a day (obese persons and intensive sports practitioners) may be more effective.

Iodine supplementation may be crucial, as about one-quarter of studies have shown that low iodine levels, especially within the thyroid gland,[355-356] have been associated with higher levels of antithyroid autoimmune antibodies.[355-360] At least one study has shown iodine supplementation to reduce the levels of antithyroid antibodies.[361] However, the majority of epidemiological studies have reported that high iodine intake increases the risk of autoimmune thyroiditis in iodine-deficient areas.[362-370] For this reason, if iodine is given alone, it may be better to limit the dose to 200 µg a day, as a study showed that in thyroxine-treated patients the production of thyroid antibodies was not induced at that dose.[371] If higher doses of iodine are requested, it should be administered along with selenium. Selenium appears to reduce markedly the risk of inducing autoimmune thyroiditis and other toxic effects on the thyroid with excessive iodine intake.[372-375]

Omega-3 polyunsaturated fatty acid supplements: Adding 2 g per day of fish oil for patients who don't regularly eat fatty fishes might help reduce autoimmune antibodies, just as fatty food consumption does. Daily omega-3 supplementation has been shown to prevent and reduce the autoimmune antibody production in mice with autoimmune lupus[376-377] and glomerulonephritis.[380] In vitro studies of dendritic cells show that adding docosahexaenoic acid, one of the major omega-3 fatty acids of fish oil, prevents development of experimental autoimmune encephalomyelitis (multiple sclerosis), another autoimmune disorder.[379]

Thus, after confirmation that nutritional levels are low, low-normal, or even average in laboratory tests (but not if these levels are high-normal or

Table 4: Nutritional treatments that reduce antithyroid antibody levels

Treatment	Dose First 6 months	Dose Next 6-18 months
• Selenium	2×200 µg/day of selenium methionine	200 µg per day
with Myo-inositol	600 mg/day	600 mg/day
• Vitamin D3	10,000 IU/day	6000 IU
• Iodine	200 µg/day	200 µg/day of iodine
	Or with selenium: 1-3 drops of Lugol 5% solution (6.25-9.75 mg of iodine/iodide)	Or with selenium: 1-3 drops of Lugol 5%
• Omega-3 fatty acids	2 g/day of fish oil	1-2 g/day of fish oil

above the upper limit), I suggest the intake of higher amounts of the above-mentioned nutrients the first four to six months. Table 4 gives an overview of the dosages of the nutritional treatments that can efficiently calm down the autoimmune thyroiditis.

Cause to Treat: Hormone Deficiencies

Hormone treatments can decrease thyroid antibody levels by 20-70%.

Thyroid treatment: Thyroid therapy is necessary not only to reduce thyroid antibodies[380-394] but to relieve the patient's hypothyroid symptoms and the risks and severity of psychological and somatic disorders that often accompany autoimmune thyroiditis. Many studies have shown the efficacy of thyroxine treatment. The best efficacy is reached when the dose is high enough to suppress the TSH level in the serum.

What is the best thyroid therapy? In most cases, I suggest desiccated thyroid extracts such as Armour, Erfa, or Nature Thyroid (doses between 30-180 mg/day) because they work better, in my experience, due to their high content in T3, T2, T1, and T0, which are not found in treatments with thyroxine alone. Be careful with patients who are allergic to pork. Most desiccated thyroid are of porcine origin and should be avoided by patients with pork meat allergy. In this case, synthetic T3-T4 combinations might be an acceptable alternative, but are not as efficient as desiccated thyroid. Triiodothyronine alone is not indicated because of the lack of persistence of beneficial effects over a 24-hour period (except if administered in 5 divided doses per day). Thyroxine alone may help on the condition that it clearly reduces the TSH level, even suppresses it. Titrate the dose up to just below the level that causes signs and symptoms of thyroid excess.

Glucocorticoid and DHEA treatments: One of the roles of cortisol is to prevent autoantibody production. In cases of cortisol deficiencies, such as Addison's disease, the risk of autoimmune thyroiditis considerably increases.[126] This explains why glucocorticoid treatments may significantly decrease the production of antithyroid antibodies but, in my experience, rarely eradicates it totally at physiological doses. Hydrocortisone (bioidentical cortisol) at doses of 15-35 mg/day in at least two divided doses (more in the morning, less at lunch) is recommended for patients with adrenal deficiency and autoimmune thyroiditis. When levels of antithyroid antibodies are high, treatment with a long-acting synthetic derivative of cortisol during the first six months may be more efficient to reduce the antithyroid antibody titers.[97,393-395] Prednisolone, which has more persistent effects over the next 24 hours than bioidentical hydrocortisone whose biological action fades after 6-9 hours, is then a good choice.

Add anabolic DHEA (dehydroepiandrosterone) to any glucocorticoid treatment in doses that are at least equivalent in milligrams to the dose of hydrocortisone that is given (15-35 mg/day). The anabolic actions of DHEA neutralize any excessive catabolism from the glucocorticoid treatment and have the additional benefit of further reducing antithyroid antibodies, as shown in women with Hashimoto's thyroiditis and premature ovarian failure. In these women, DHEA sulfate levels were found to be significantly lower than in women without Hashimoto's and normalized with a substantial reduction of anti-thyroperoxidase antibodies at 30 mg/day of DHEA. DHEA treatment also normalizes the natural killer cell toxicity, which is deficient, in patients with Hashimoto's thyroiditis.[397]

Testosterone therapy, along with female hormone treatments in women, has been reported to oppose the development of various types of autoimmune disease. In autoimmune thyroiditis, a significantly lower testosterone to estradiol ratio is found in men.[148] Testosterone therapy, on the other hand, has been shown to reduce antithyroid peroxidase and antithyroglobulin antibodies in men and animals with the disorder.[398-402] In women with autoimmune lupus, the testosterone is also significantly lower in the serum.[403]

Table 5 gives an overview of various autoimmune pathologies in which testosterone therapy was shown to reduce the autoimmune antibody levels.

As discussed above, the reason autoimmune thyroiditis is 5 to 10 times more common in women than men may be due to their much lower testosterone levels, which leave them less protected against autoimmune dysregulation. Regarding lupus, another autoimmune disorder, testosterone levels are lower in women who have lupus than in women without it, and testosterone therapy has been reported to reduce the production of autoimmune antibodies in women with lupus. I have not (yet) found studies on testosterone therapy for autoimmune thyroiditis in women.

However, if testosterone therapy is administered to a female patient, it should always be done in combination with sufficient female hormone therapies to avoid masculinization. Estrogen and progesterone therapy protect women against hair loss, body hair overgrowth, acne, and other undesirable effects of testosterone ➤

Because of its frequency and adverse impact, Hashimoto's thyroiditis should be systematically screened and treated in women.

Table 5: Autoimmune diseases that may improve (decrease) with testosterone therapy

- Autoimmune thyroiditis (men,[398] rats,[399-400] chickens) [401-402]
- Autoimmune encephalomyelitis (rats)[404-405]
- Autoimmune demyelinating disease (mice)[406]
- Autoimmune epilepsy (men)[407-410]
- Autoimmune disease in general (mice)[423]
- Sjögren syndrome (men,[411] mice)[412]
- Systemic lupus erythematosus (men, women,[413-418] mice)[419-420]
- Autoimmune orchitis (rats)[421]
- Autoimmune prostatitis (mice)[422]

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therapy administered alone. In case high amounts of intramuscular testosterone injections are administered, finasteride, a progesterone derivative, which reduces the conversion of testosterone to the masculinizing dihydrotestosterone, may have to be added and is safe only in presence of testosterone supplementation.

Table 6 overviews the most important hormone treatments for Hashimoto's thyroiditis.

Causes to Treat: Viral, Bacterial, and Yeast Infections

Infections by microorganisms have been reported to cause or contribute to the development of autoimmune thyroiditis. Both a leaky gut, which permits these microorganisms to penetrate into the body, and an inefficient immune system contribute to make a patient's thyroid gland prone to infection by viruses, bacteria, and yeast, which trigger autoimmune reactions. In autoimmune thyroiditis, two immune failures have been discovered: a defect in the number of CD8(+) suppressor lymphocytes[424] and a decrease in efficacy (toxicity) of natural killer cells.[425-426] CD8(+) suppressor lymphocytes are those that oppose the development of autoimmune thyroiditis. Natural killer cells serve to contain viral infections to provide time for the immune system to produce antigen-specific cytotoxic T cells that respond to the invaders and clear the infection. Natural killer cells control viral infections by secreting interferon γ and tumor necrosis factor α.

Viruses seem to trigger or amplify autoimmune thyroiditis more than other infectious agents.[427-429] Especially herpes viruses, particularly the Epstein-Barr virus[430-435] (also called herpes virus 4) that causes mononucleosis seems to be worst, but the herpes virus[436-437] that causes herpes labialis has also been incriminated as facilitating Hashimoto's thyroiditis. The hepatitis C virus,[438] parvovirus,[439] human T-cell lymphotropic virus type 1,[440-442] and HIV virus[443-444] have also been associated with autoimmune thyroiditis.

Yeast is another trigger of autoimmune diseases, and, therefore, potentially also of autoimmune thyroiditis. Patients with autoimmune diabetes, for example, are four times more likely to have anti-

Table 6: Hormone treatments that reduce antithyroid antibody levels

TREATMENT	INDICATION	DOSE	DOSE APPLICATION	EFFICACY**
Thyroid				
• Oral desiccated thyroid	Mild to severe HT*	30-150 mg/day	• Start at a low dose and gradually (every 10-14 days) increase the dose to the optimal level	+±
• T4/T3 combinations		10 µg		+
• Thyroxine		75-200 µg/day		+
Cortisol				
• Hydrocortisone (bioidentical)	Mild to severe HT	15-35 mg/day (women) 20-60 mg/day (men)	• Start directly at the useful dose (in rare cases of acute inflammation with very high antithyroid antibody levels, start at 50%-100% higher than the ideal dose [for 2-3 months], then decrease to the physiological dose) • Always with a sufficient amount of DHEA	+
• Or Prednisolone	Mild HT	2.5-5 mg/day		+
	Severe HT	5-10 mg/day (max. 2-3 months)		++
• Or Methyl-prednisolone (in overweight or hyper-tensive patients)	Mild HT	2.5-5 mg/day		+
	Severe HT	5-10 mg/day (max. 2-3 month		++
DHEA				
• DHEA	Mild to severe HT	Women 10-30 mg /day	DHEA should always be added to a glucocorticoid treatment	± to +
		Men 20-60 mg/day		± to +
Testosterone				
• Transdermal testosterone gel or liposomal cream	Mild HT	Women: 0.5%: 0.5 -1 g/day (2.5-5 mg/day) Men: 10%: 0.5 -3 g/day (50-300 mg/day)	• Always in combination with transdermal estradiol 1-3 mg/day and 100 mg/day of progesterone	+
• IM testosterone enanthate injections	Severe	50-100 mg/month	• Almost always with finasteride (2.5-7.5 mg/day) to block masculinization	+±

Note: *HT = Hashimoto's thyroiditis: **mild:** antithyroid antibody levels reach a maximum of 10 times the upper limit; **severe:** antithyroid antibody levels are far higher than 10 times the upper limit; ** efficacy to reduce autoimmune antibodies.

Candida (enolase) IgG antibodies,[445] and twice more likely to have Candida albicans overgrowth in the stools.[446] Furthermore, experimental autoimmune encephalomyelitis is considerably aggravated if mice are beforehand infected by Candida. Bacteria that have been associated with thyroiditis include streptococci, staphylococci,[437] Yersinia, Borrelia (Lyme disease),[448-449] and Helicobacter pylori.[435] Among the parasites that have been associated with autoimmune thyroiditis is Treponema gondii.[450]

demonstrated that thymosin-alpha-1 strongly opposes the development of autoimmune thyroiditis in mice prone to produce antithyroid antibodies. However, in mice, relatively resistant to autoimmunity, thymosin-alpha-1 may trigger a mild form of autoimmune thyroiditis.[456-457] For this reason, treatment with thymosin-alpha-1 is probably and mainly indicated in patients with high titers of thyroid antibodies.

Thymosin-alpha-1 activates the natural killer cell activity.[458-460] In vitro, it has been shown to inhibit the proliferation of the HIV virus in infection of macrophages and polymorphonuclear cells by activating CD8(+) cells so powerfully that some researchers think it makes a re-evaluation of the approach to antiretroviral therapy necessary.[461] In Lyme disease, this treatment is, in my experience, efficient in reducing the aggressiveness of the disease, much more than prolonged use of antibiotics that has toxic effects that thymosin-alpha-1 does not have. I cannot stress enough that the best therapies are those that treat the cause (the immune deficiency due to a lack of thymus hormones, for example) than the consequences (providing long-term antibiotics to kill intracellular microorganisms which install themselves when there is an immune deficiency). Furthermore, thymosin-alpha-1 is anti-inflammatory, a beneficial effect in autoimmune diseases.

Table 7 shows the most important immune-enhancing treatments for patients with Hashimoto's thyroiditis.

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Table 7: Treatments that reduce antithyroid antibody levels by improving the immune system

TREATMENT	INDICATION	DOSE	DOSE APPLICATION	EFFICACY**
Dietary adjustments See above table 3				
Vitamin D See above table 4				
Thyroid See above table 6				
Thymus hormone				
• Thymosin-alpha-1	Moderate to severe HT	0.05 mg/day (0.03-0.15 mg/day)	Start directly at the useful dose. The higher the dose the greater the efficacy.	+ to +±

How to avoid getting the thyroid invaded by microorganisms that trigger autoimmune thyroiditis? First of all, by blocking the passage of these microorganisms through a leaky gut by a healthy diet, as explained earlier in this article. Second, by restoring an optimal immune system by hormone and nutritional supplementations (of any deficiency) so that even if microorganisms pass through the gut wall, they are destroyed by the body's natural defenses. *Thyroid therapy* is a strong immune booster that has been shown to increase natural killer cell toxicity[451-453] and increase both the number of CD4 helper and CD8 suppressor cells.[454-455]

Moreover, daily subcutaneous injections of *thymosin-alpha-1,* probably the body's most potent immune-enhancing hormone may be an additional powerful tool. Research has

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Causes to Treat: Pollutants

Breathe pure air. Pollutants trigger the development of autoimmune thyroiditis by attacking the immune system and tissues. There are so many toxins that we cannot list them all, but let's cover some of them.

Smoking increases the risk of autoimmune thyroiditis.[462-463] High cadmium levels have been associated with autoimmune thyroiditis,[464] as has radioactive fallout from Chernobyl, for example.[465-466]

Endocrine disruptors such as polychlorinated biphenyls (PCBs), used as coolants and insulating fluids (transformer oil) for transformers and capacitors or plasticizers in cement, are so persistent that even if a baby receives them through breast-feeding from his mother, they remain in the body as a young adult. Young adults who were breastfed as babies have been reported to have higher levels of the various types of persistent PCBs and antithyroid peroxidase antibodies than individuals who were not breastfed.[467]

Table 8 provides an overview of the main recommendations to give to patients to lessen the toxic load.

Table 8: Pollution-free living to reduce antithyroid antibody levels

Globally, the recommendations include the following:

- **Drink lots of purified water** to evacuate pollutants in the urine.
- **Consume organic foods** and avoid toxic foods, including phthalates that leak into food from packaging.
- **Breathe pure air** by living and working in safe neighborhoods.
- If indoor air is not clean, use **indoor air filters**. Any odor of synthetic projects or exhaust gases should prompt an alarm to evacuate the area.
- **Stop smoking.**

Conclusion

Because of its frequency and adverse impact, Hashimoto's thyroiditis should be systematically screened and treated in women. The treatment of autoimmune thyroiditis (particularly Hashimoto's thyroiditis) is generally a combination of therapies consisting of, at least, dietary adjustments, nutritional therapies (selenium and vitamin D3 being the most important ones), and reduction of any toxic overload. However, in more severe or treatment-resistant forms with highly elevated antithyroid antibody levels, hormone therapies, such as thyroid, glucocorticoid, DHEA, and/or testosterone (associated with female hormone therapy in women), are generally inevitable, and optimization of the immune system might be necessary. In case the patient is hypothyroid, thyroid therapy becomes essential to treat the hypothyroidism and can considerably reduce antithyroid antibody levels too. ◆

Born in Antwerp, Belgium, Dr. Hertoghe practices his medicine in his clinic in Brussels. With his sister, Dr. Thérèse Hertoghe, they proudly represent the fourth successive generation of physicians working with hormonal treatments – and this since 1892 (after Eugène Hertoghe, former vice president of the Royal Academy of Medicine in Belgium, and Luc and Jacques Hertoghe, endocrinologists). Dr. Thierry Hertoghe devotes his life to the promotion of a better, patient-oriented, and evidence-based medicine.

Author of numerous books, Dr. Thierry Hertoghe also travels a lot to take part in numerous conferences and congresses throughout the world. He co-organizes many of these specialized gatherings and holds important positions in several international and national medical organizations (which usually tend to fight against aging). He is the president of the International Hormone Society (over 2500 physicians), and of the World Society of Anti-Aging Medicine (over 7000 physicians), as well as the supervisor of two important postacademic trainings for doctors.

http://www.hertoghe.eu

Full references for

"Hashimoto's Thyroiditis, A Common Disorder in Women: How to Treat It" Parts 1 and 2 by Thierry Hertoghe, MD

and

"An Integrative Medical Approach to Macular Degeneration" Parts 1 and 2 by Marc Grossman, OD, LAc

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