Hashimoto's Thyroiditis, A Common Disorder in Women: How to Treat It – Part 1

by Thierry Hertoghe, MD

Interest in Hashimoto's thyroiditis is high in the doctor's office as well as on the internet. Patients openly testify that they have Hashimoto's, and some attribute an endless list of complaints to this disorder. Most of these patients are women. What is true about this media hype? Is it merely media hype or does it reflect reality? What are the causes and what can be done about it?

Hashimoto's thyroiditis is an autoimmune disorder. In autoimmune disorders, antibodies are made against constituents of the body's tissues, and lymphocytes concentrate in the affected tissues. They are dysregulations of the immune system. The thyroid gland is the most common organ affected by autoimmune disease. Hashimoto's thyroiditis is the most frequent autoimmune disease of the thyroid gland.[1]

Two autoimmune disorders predominate in the thyroid gland: Hashimoto's thyroiditis and Graves' disease. Hashimoto's thyroiditis is also known as chronic lymphocytic thyroiditis or chronic autoimmune thyroiditis and involves the production of antibodies against thyroid peroxidase and/or thyroglobulin. Thyroid peroxidase is the enzyme that oxidizes iodide ions to form iodine atoms for incorporation into T3 and T4 hormones in the colloid. Thyroglobulin is the bigger protein that stores T3 and T4 in the colloid. [2-4]

Graves' disease, on the other hand, is also known as Basedow's disease and is characterized by the production of autoantibodies that target and stimulate the TSH (thyroid stimulating hormone) receptor on the thyroid follicular cells. It is the most common cause of hyperthyroidism.[2]

Table 1: Differences Between Hashimoto's Thyroiditis and Graves' Disease

Autoimmune disorder	Hashimoto's thyroiditis[2,4]		Graves' disease[2,5]	
Antibodies	• Antithyroid peroxidase antibodies • and/or Antithyroglobulin antibodies • (Normally no anti-TSH receptor antibodies)		• Anti-TSH receptor antibodies • (Antithyroid peroxidase antibodies (in 80% of patients)) • (Antithyroglobulin antibodies (in 50%))	
Thyroid state	Progresses into hypothyroidism		Hyperthyroidism	
Serum TSH	Variable	Progresses into hypothyroidism	Low	Hyperthyroidism
Free T3	Variable		High	
Free T4	Variable		High	
Clinical features	• Hypothyroid, • Euthyroid, • or (more rarely) Hyperthyroid		• Hyperthyroid signs and symptoms with sometimes pretibial myxedema, often thyroid eye disease	
Thyroid ophthalmopathy	• Rarely exophthalmos (<2%), • More frequently hypothyroid signs, such as suborbital edema		• Exophthalmos (in 25%), • Other signs: lid lag, eyelid edema, chemosis, extraocular muscle weakness	
Thyroid gland	• Thyroid atrophy and (micro) nodules are more common, • Occasionally goiter		• Goiter	
Histological findings	• Mononuclear cells, mostly lymphocytes (especially T cells)		• Activated follicular cells • Increased vascularization	
	• Destroyed thyroid follicles		• Hyperplastic follicles with colloidal absorption	
Thyroid ultrasound	• Reduced echogenicity (progresses to atrophic hypovascular thyroid)		• Increased echogenicity (diffusely enlarged hypervascular thyroid gland)	

Diagnosis of Hashimoto's Thyroiditis

The diagnosis of Hashimoto's thyroiditis relies on the presence of antibodies to thyroid peroxidase and/or thyroglobulin in the serum and reduced echogenicity on a thyroid ultrasound. On histological examination, the thyroid gland is infiltrated by mononuclear cells, mostly lymphocytes (especially T cells), and many thyroid follicles are destroyed. These pathological features of autoimmune thyroiditis lead to progressive atrophy and fibrosis of the thyroid gland. Clinically, the patient can present hypothyroid signs and symptoms, appear euthyroid, or (more rarely) hyperthyroid. Local symptoms such as thyroid tenderness and neck pain are only present in acute and subacute thyroiditis. As Hashimoto's disease is chronic, patients are generally devoid of local symptoms.[2,4]

Diagnosis of Graves' Disease

The diagnosis of Graves' disease is based on the presence of TSH-receptor antibodies, abnormally high T4 and T3 levels, suppressed TSH levels in the serum, and clinical features. Physical signs of Graves' disease include swelling of the anterior part of the calves (pretibial myxedema), thyroid eye disease, exophthalmos (prominence of eyes), lid lag (higher upper eyelid while the eye is directed down), eyelid edema, chemosis (conjunctival edema), extraocular muscle weakness, and possibly increased pigmentation and vitiligo. Thyroid ophthalmopathy is present in about 50% of Graves' patients, which includes exophthalmos in about 25% of cases.[2]

Table 1 reviews the differences between Hashimoto's thyroiditis and Graves' disease.

Hashimoto's thyroiditis is much more frequent in women. Thyroid autoimmune diseases affect up to 5-6% of the population, but in some age groups (30-50 years) its frequency may climb up to 10-15%, and are seen mostly in women.[3,6-7] Hashimoto's thyroiditis is the most frequent autoimmune thyroid disorder, affecting more than 10% of women and only 2% of men.[8] The higher frequency in women may be due to their lower androgen levels, particularly testosterone, which is 10 to 20 times lower than in men. Androgens are known to help prevent and treat autoimmune disorders of all types (for more information, refer to the treatment section).

Graves' disease affects eight times more women than men.[9]

Scientific data suggests that most cases of Hashimoto's thyroiditis are intermediate degrees of hypothyroidism. Most cases of Hashimoto's thyroiditis ultimately progress into hypothyroidism,[10-18] although initially patients can appear euthyroid or even hyperthyroid.[2] Indeed, Hashimoto's thyroiditis may trigger flare-ups of hyperthyroidism.-

Why do most patients with antithyroid antibodies suffer from some degree of thyroid deficiency? The reason lies in the repeated damage to the thyroid tissue from the accumulation of mononuclear cells and aggression of antithyroid antibodies, resulting in atrophy of the thyroid gland. Epidemiological studies often show that autoimmune thyroiditis is significantly associated with higher serum levels of TSH[19-27] and lower levels of free T3[28-29] and T4,[30-34] signs of decreased thyroid function. Research has also shown that patients with autoimmune antithyroid antibodies more often suffer from hypothyroid signs and symptoms[31] and are significantly more prone to psychological and somatic disorders, which are typically more frequent in hypothyroidism. Furthermore, thyroid therapy for autoimmune thyroiditis usually relieves the symptoms of thyroid deficiency and the risks and severity of hypothyroid-related disorders.[36-50] Thyroid therapy also reduces the levels of antithyroid antibodies (for more information see treatment section).

This explains why Hashimoto disease is considered the leading cause of hypothyroidism in iodine-sufficient areas of the world.[2]

Hashimoto thyroiditis is associated with other important disorders, for which the scientific evidence is abundant. Hashimoto's thyroiditis appears to facilitate many disorders that are serious and stressful.[51-265] It is accompanied by a lower quality of life, fatigue,[35,54-66] reduced brain perfusion,[51-53] reduced mental health,[67] cognitive impairment,[102] depression (up to 8-9x higher risk),[67,70-75] and anxiety[67,75,79-81,87] – with up to nine times a higher risk of panic disorder.[75,79]

Specifically, in women, fertility is lower, the pregnancy rate is lower[111-112] and the risk of miscarriages is also greater,[113] as is the risk of premature ovarian failure.[124-125] Nearly half of women with polycystic ovarian syndrome (PCOS) have antithyroid antibodies, which is 5-10 times higher than the normal rate![123]

In obese individuals, males and females, antithyroid antibodies are also frequently found.[149-152] Approximately one third of patients with autoimmune type 1 diabetes also have Hashimoto's thyroiditis.[127]

Patients with hair, skin, and mucosa disorders are also more likely to experience Hashimoto's. Hence, up to 25% of patients with alopecia totalis have been reported to present Hashimoto's thyroiditis,[180] 34% of patients with vitiligo,[190] and 20-30% of those with psoriasis have antithyroid antibodies.[186] Furthermore, four times more patients with Sjögren syndrome have Hashimoto's thyroiditis than in the general population.[171]

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What about the cardiovascular risk? In patients with Hashimoto's thyroiditis, cardiac function is lower,[68-69] the risk of mitral valve prolapse has been reported to be three-fold higher,[213] that of coronary vasospasm (main cause of heart attacks in women) is five times higher when antithyroid peroxidase antibodies are elevated,[214] and the incidence of coronary heart disease is about 40% higher.[219] The likelihood of myocardial infarction has been found to be two-fold higher.[225]

Autoimmune thyroiditis might also affect patients with inflammatory disease of the joints and muscles. The risk of rheumatoid arthritis, for instance, is 2.5 times higher in

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Table 2: Hashimoto's Thyroiditis: Associated Pathologies

TYPE OF DISORDER	RISK, SEVERITY*
Fitness deficits	
• Reduced brain perfusion[51-53]	*2.1x ↑ perfusion defects*[53]
• Low quality of life, fatigue[35,54-66]	*+66% greater fatigue*[55]
• Reduced physical functioning[67]	*-11% reduction*
• Low cardiac function[68-69]	*-29% reduction*[69]
Psychological disorders	
• Hypothyroid symptoms[35]	*+50% increase*
• Reduced mental health[67]	*-11% reduction*
• Depression[67,70-85]	**1.5-9x higher risk**[79]
• Suicide (death by)[86]	**1.4x higher risk**
• Anxiety[67,75,79-81,87]	**4x higher risk**[80]
• Panic disorder[75,79]	**9x higher risk**[79]
• Obsessive compulsive disorder[60-75]	**1.5x higher risk**[61]
• Neuroticism[88-89]	**1.3x higher risk**[88]
• Paranoia[90]	in 40% in Hashimoto's encephalopathy
• Psychosis[91-92]	in 25%[91] in Hashimoto's encephalopathy
Mental -Neurological disorders[90]	
• Neuropathy (peripheral)[93]	in 11% of HT patients
• Orbitopathy[94-96]	in 2% of HT patients[96]
• Encephalopathy[97-99]	Rare
• Attention deficit[100-101]	**2.9x higher risk**[101]
• Cognitive impairment[102]	in 28% of HT Patients
• Alzheimer's disease[103]	Rare
• Other dementias[104-107]	Rare
• Multiple sclerosis[108]	9% of men with MS have HT
Sleep disorders	
• Sleep apnea[109-110]	47% of patients have HT[109]
Sexual /reproductive disorders	
• Sexual dysfunction[35,72]	**1.4x higher risk**[72]
• Infertility[111-112]	HT women have
	-41% lower pregnancy rate[112]
• Miscarriages[113]	**1.2-2.5x higher risk**
Endocrine disorders	
• Prolactinomas[114-116]	30% of patients have HT[114]
• Thyroid nodules, goiter[117-121]	in 36% of HT patients[120]
• Hypothyroidism[10-18]	**10x higher risk**[10]
• Polycystic ovarian syndrome[122-123]	**11x higher risk**[123]
• Premature ovarian failure[124-125]	20-30% of patients have HT[125]
• Addison's disease[126]	**Higher risk**
• Type I diabetes[127-146]	35% of patients have HT[127]
• Type II diabetes[147]	19% of patients have HT
• High estradiol-low testosterone (men)[148]	Men with HT have +10% higher E2/T ratio
Metabolic disorders	
Overweight, obesity[149-152]	*4x higher ATPO and 10x higher ATG levels*[152]
Digestive disorders	
• Celiac disease[153-160]	**4x higher risk**[160]

TYPE OF DISORDER	RISK, SEVERITY*
General disorders	
• Reduced general health[67]	*-15%*
• Oxidative stress, low antioxidant capacity[161-165]	*↑ ROS, ↓ antioxidant potential, +18% ↑ AGEs*
• Other autoimmune diseases	in 20-40% of HT patients[126,166-177]
Hair, skin, mucosa disorders	
• Alopecia areata, totalis[178-180]	5-25% of patients have HT[178,180]
• Ichthyosis[43,181]	**Higher risk**
• Atopic dermatitis[182-183]	10% of patients have HT[182]
• Chronic urticaria[184-185]	18% of patients have HT[184-185]
• Psoriasis[186]	25-30% of patients have HT[184]
• Vitiligo[187-194]	34% have HT (vs 9% in the general population)[190]
• Hirsutism[43]	**Higher risk**
• Sjögren syndrome, reduced salivary output[170-174]	*4x more have HT (than the general population)*[171]
Cardiovascular disorders	
• Lipid disorders[195-200]	*High total-LDL cholesterol, triglycerides, low HDL*
• Hyperhomocysteinemia[201]	*+22% ↑ homocysteine*
• Arterial stiffness[202-204]	*+10% ↑ pulse wave velocity*[204]
• Atherosclerosis[205-208]	*Thicker intima media*
• Thrombosis[209-210]	*Fibrinolytic deficit*
• Pulmonary hypertension[211-212]	**3x higher risk**[211]
• Mitral valve prolapse[213]	**3x higher risk**
• Coronary vasospasm[214]	**5x higher risk (w/ ↑ ATPO)**
• Coronary heart disease[215-224]	**1.4x higher risk**[219]
• Myocardial infarction[225-226]	**2x higher risk**[225]
• Stroke[227]	**1.1-1.3x higher risk**
• Hepatitis C virus-related cryoglobulinemia[228-231]	**3-6x ↑ risk for high cytokines CXCL 9,10 & 11****[228]
Bone, joint, and tendon disorders	
• Temporomandibular arthritis (TMA)[232]	**100% risk** to have TMA symptoms
• Myopathy (proximal)[93,233]	in 13% of HT patients[93]
• Polymyalgia rheumatica[169,234]	**Higher risk**
• Body pains[235-236]	**Higher risk**
• Fibromyalgia[235,237-243]	in 31% of HT patients[241]
• Rheumatoid arthritis[235,244-248]	**2.5x higher risk**[247]
• Systemic lupus Erythematosus[169,175-177]	2.3x more patients have HT than controls[175]
• Systemic sclerosis[249]	20% of patients have HT
• Spinal disc degeneration[250]	**1.8x higher risk**
Cancer	
• Thyroid (papillary) cancer[117-118,251-261]	**3x higher risk**,[258] but more favorable outcome[262-264]
Premature death by	
• Suicide[86]	**1.4x higher risk**
• Unknown matters[86]	**1.4x higher risk**
• Cardiovascular causes[265]	**1.7x higher risk**

Notes: Symbols: * Approximate risks, severity found in various studies and compared to the general population or control subjects without Hashimoto's thyroiditis; **heart failure markers

Abbreviations: HT = Hashimoto's thyroiditis; ATPO = antithyroperoxidase antibodies; ATG = antithyroglobulin antibodies; x = times or -fold; ROS: reactive oxygen species = free radicals; AGE's = Advanced Glycation End Products; ↑= increased = higher

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patients with autoimmune thyroiditis.[247] Antithyroid antibodies are found in 13% of patients with proximal myopathy,[93] in 20% of patients with systemic sclerosis,[249] and in more than 30% of patients with fibromyalgia.[241] These figures are higher than the 5-6% found in the general population. Patients with systemic lupus erythematosus have 2.3-fold higher incidence of Hashimoto's thyroiditis.[175]

The gastrointestinal tract is also not spared by Hashimoto's thyroiditis with a risk of celiac disease that is approximately four times higher than in people without antithyroid antibodies.[160]

The incidence of thyroid cancer, particularly papillary cancer, has been found to be on the average three-fold as high in patients with autoimmune thyroiditis,[258] but with a more favorable outcome (less malignancy).[262-264]

Life expectancy also seems to be decreased in Hashimoto patients. Death by suicide and unknown matters, for example, has been reported to be 43% more frequent in these patients,[86] and death by cardiovascular disease 72%.[265]

Table 2 gives an overview of the most common psychological and physical disorders significantly associated with autoimmune thyroiditis. To provide an approximate idea of how much greater the risk of each disease is for patients with Hashimoto's, I have added some figures:

- **The severity of the disorder in patients with Hashimoto's thyroiditis**, which is presented in *italic characters*. For reduced brain perfusion, for example, patients with Hashimoto have *2.1x more perfusion defects* than controls without HT.[53]
- **How much higher antithyroid antibody** levels are in patients with the disorder (suggesting that higher antithyroid antibody levels promote the disease). These figures are presented in *italic characters*. In obesity, for example, *the levels of antithyroid peroxidase antibodies are 4 times higher and those of antithyroglobulin antibodies 10-fold higher.*[152]
- **The risk to have a disorder for patients with Hashimoto's thyroiditis**, which is presented in **bold characters**. A Hashimoto patient has, for example, a **2x higher risk** of suffering from an anxiety disorder.[80]
- **The percentage of patients with the disorder who have Hashimoto's thyroiditis**, a percentage that is generally higher than the 5-6% of the general population and which is presented in standard characters. For example, 34% of vitiligo patients have been reported to have HT (HT = abbreviation of Hashimoto's thyroiditis).[190]

Because Hashimoto's thyroiditis is often associated with hypothyroid symptoms and increased risks of many pathologies, even in its milder forms, it is not an innocent or safe condition. Hashimoto's thyroiditis is likely an intermediate degree of hypothyroidism. Patients suffer needlessly and need therapy. It is not to be considered as a silent condition that needs only surveillance.

Causes and Treatment of Hashimoto's Thyroiditis

To treat, find first the cause or causes of Hashimoto's thyroiditis. Because of all the adverse effects of Hashimoto's thyroiditis, it is important to test for antithyroid antibodies, and when antibodies are elevated to treat the autoimmune disorder. Indeed, autoimmune thyroiditis is treatable. Medical research has made substantial progress in this field, offering various solutions.

To treat it efficiently, it is essential to detect the cause or causes of abnormal productions of antithyroid antibodies. Treating the cause first is a principle in medicine for any chronic or recurrent pathology. When a patient suffers from recurrent headaches, for example, a doctor can transiently relieve the patient with pain medication, but the main focus should be put on finding the cause and eliminating it: a hormone deficiency, neck contracture due to emotional retention or poor posture, food intolerance, etc. In the case of autoimmune thyroiditis, the etiology is usually **multifactorial**. One factor is often not sufficient to trigger the production of autoimmune antibodies. For this reason, the treatment of autoimmune thyroiditis usually includes a combination of therapies, each one focused on one of the causes.

What are the causes of autoimmune thyroiditis? Basically, five categories of causes have been found:

1. Genetic predispositions,[266-269]
2. Dietary errors,
3. Nutritional deficiencies,
4. Hormone deficiencies,
5. Viral, bacterial, yeast, and parasitic infections,
6. Toxic products.

Combine various medical therapies to reduce antithyroid antibodies. Regarding causes other than genetics, physicians can act with efficacy to reduce the levels of antithyroid ➤

Table 3: Dietary Changes That Reduce Antithyroid Antibody Levels

DIETARY CHANGE	THE STUDIES; WHAT IT DOES	PRACTICAL TIPS
• Paleo diet[281-283]	This diet is easier to digest, as it consists of foods that our ancestors ate in the Paleolithic period before agriculture was discovered (the Neolithic) and humans started to consume foods less fit for their guts, such as cereal and milk products. A study showed that regular intake of fruit and berry juices increases the likelihood of autoimmune diabetes.	• Consume fresh vegetables, fruits, meat, fish, poultry, and eggs. • Consume organic foods. • Eat food raw, unprocessed[283] or steamed, boiled, or cooked at low temperature without oil. • Reducing fruit and fruit juice consumption to below-average levels might have value.[284]
• Protein-rich foods at breakfast	When protein-rich foods are eaten in the morning, there is time enough to digest them in the stomach and small intestine and absorb them as amino acids in the small intestine. At the end of the day, the gut can rest and recover better during sleep without undigested proteins remaining in the gut. A protein-rich breakfast also increases satiety.[285-286]	• Eat the main protein-rich meal (meat, poultry, fish, and eggs) in the morning. • Moderate protein intake at lunch. • Avoid consuming protein-rich foods in the evening.
• Small fatty fishes, rich in omega-3 polyunsaturated fatty acids	Regular intake of fatty (oily) fishes has been reported to reduce the incidence of postpartum thyroiditis by more than four times[287-288] and of autoimmune diabetes two-fold![289] When fish is eaten in the first part of the day, it also reduces food intake at supper, helping to not overload the gut during the night.[290]	• Increase the intake of small fatty fishes, such as sardines, mackerel, eels, and herring. • Limit the consumption of big fatty fishes, such as salmon, tuna, and trout, as they often contain too much mercury.
• Intermittent fasting • Supper: to skip or eat minimally	Intermittent fasting has been shown to reduce the production of autoimmune antibodies of a variety of diseases.[291-294] It provides a rest for the gut, allowing it to be temporarily free of new aggressors. It also permits the abdomen to get flat.	• In my experience, the best plan is to skip one meal daily, especially the evening meal, and eat proteins only in the morning. • Consuming boiled or steamed vegetables at supper is an acceptable alternative.
• Avoid soy milk	Soy milk intake is associated with a higher risk of autoimmune thyroiditis in children.[295]	
• Avoid cow-milk protein	Cow-milk protein is known to trigger autoimmune diabetes[296-299] and is suspected of triggering other autoimmune diseases.	• Avoid milk, yogurt, cheese, etc. • Clarified butter (also called ghee) is okay, as it has lost the white layer of allergenic proteins.
• Low-carbohydrate diet	• A low-carb diet alone has been reported to reduce thyroid antibody levels by 50%.[300]	Stop consumption of bread, porridge, and other cereals, particularly gluten-containing cereals, as well as high-sugar foods and drinks
	• Gluten-containing cereals can trigger autoimmune thyroiditis[301-303] and celiac disease. Celiac disease itself is often associated with autoimmune thyroiditis[153-160] and autoimmune diabetes.[304-306]	• No wheat. • Sprouted rice and other sprouted grains can be acceptable alternatives.
• Avoid artificial sweeteners	The consumption of artificial sweeteners is associated with a higher risk of autoimmune thyroiditis[307] and diabetes.[308]	Avoid aspartame, cyclamate, and other artificial sweeteners.
• Avoid sugar	The consumption of sugar and sweetened beverages is associated with a higher risk of autoimmune diabetes.[309-310]	Avoid sugar and soft drinks.
• Probiotics	Probiotic supplementation has been shown to reduce autoimmune antibody production in various autoimmune disorders, including autoimmune enteropathy, diabetes and multiple sclerosis.[311-314]	In the case of dysbiosis: • The addition of probiotics (with at least 10 billion germs of lactobacilli and bifidus bifidi strands per capsule) is recommended. • Take various types of probiotics in alternation to restore the variety in the strands better.

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antibodies. Therefore, let's focus on dietary adjustments and nutritional and hormone treatments complemented by avoiding toxic products as much as possible. The best results are obtained by combining these therapies, which should result in a 50 to 100% reduction of thyroid antibody levels within the next six to 12 months.

Changing the diet and improving the digestive system may decrease the levels of antithyroid antibodies by more than 50%.

What are the mechanisms? *Dietary maladjustments* cause *dysbiosis*[270-274] and a *leaky gut,*[275-279] which bring into the body foreign compounds and microorganisms that accumulate in the thyroid and trigger autoimmune thyroiditis.

Inappropriate foods may aggress the intestinal wall and break open the tight junctions that hold the intestinal wall cells together, causing leaks in the gut wall (leaky gut). Such foods may also cause some strands of gut flora to proliferate excessively while other strands are lacking. This *dysbiosis* can cause further harm and leaks in the intestinal wall. Through breaches of the gut wall, unusually large molecules (that form antigens), undesirable microorganisms, and toxic products may leak into the tissues surrounding the gut. From there, they penetrate the bloodstream and nest themselves in tissues and organs with high blood flow. The thyroid has some of the highest blood flow of any tissue in the human body. Brought by the bloodstream, these irritating compounds and microorganisms accumulate in the thyroid gland, where they trigger the production of antibodies against them and components of the thyroid gland that surround them.

The same mechanisms intervene when we consume good food at the wrong time of the day; protein-rich foods at supper, for example. Protein-rich foods, such as meat, poultry, fish, and eggs, provide essential amino acids to build the body but take much more time than carbohydrates and fats to be digested in the gut.[280] The stomach provides an initial (lengthy) digestion of these proteins, which takes about three to nine hours.

If this type of food is ingested in the morning, there is usually no problem. Proteins are fragmented by the stomach acid into smaller peptides and amino acids and leave the stomach in the afternoon, pushed by gravity and movements of daily living into the small intestine, where they are further digested and absorbed as amino acids. At the end of the day, the proteins have then been completely digested and absorbed. The abdomen appears flat and the gut empty of disturbing substances. If no food or light foods such as boiled vegetables are eaten in the evening, the stomach is empty during sleep and the gut can rest.

In most families, however, supper is the main meal and takes place in the evening. It contains plenty of proteins, making it impossible for the gut to digest everything before bedtime. This leaves the stomach overloaded and the abdomen bloated the whole night and next morning. The food overload overwhelms the gut with undigested foods that ferment and putrefy – "rotten" would be a better word – causing dysbiosis and damage to the intestinal wall and thus, a leaky gut during the nighttime.

How can the human immune system be stopped from producing autoimmune antibodies against its own thyroid gland because of a leaky gut and dysbiosis? Make the gut stop leaking by intermittent fasting and dietary improvements and recover good gut flora via probiotic supplementation.

So, what is the appropriate diet? Table 3 (page 41) gives an overview of the main dietary recommendations for patients.

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http://www.hertoghe.eu

References available online only
www.townsendletter.com