

Hashimoto's disease

Description

Hashimoto's disease is a condition that affects the function of the thyroid, which is a butterfly-shaped gland in the lower neck. The thyroid makes hormones that help regulate a wide variety of critical body functions. For example, thyroid hormones influence growth and development, body temperature, heart rate, menstrual cycles, and weight. Hashimoto's disease is a form of chronic inflammation that can damage the thyroid, reducing its ability to produce hormones.

One of the first signs of Hashimoto's disease is an enlargement of the thyroid called a goiter. Depending on its size, the enlarged thyroid can cause the neck to look swollen and may interfere with breathing and swallowing. As damage to the thyroid continues, the gland can shrink over a period of years and the goiter may eventually disappear.

Other signs and symptoms resulting from an underactive thyroid can include excessive tiredness (fatigue), weight gain or difficulty losing weight, hair that is thin and dry, a slow heart rate, joint or muscle pain, and constipation. People with Hashimoto's disease may also have a pale, puffy face and feel cold even when others around them are warm. Affected women can have heavy or irregular menstrual periods and difficulty conceiving a child (impaired fertility). Difficulty concentrating and depression can also be signs of a shortage of thyroid hormones.

Hashimoto's disease usually appears in mid-adulthood, although it can occur earlier or later in life. Its signs and symptoms tend to develop gradually over months or years.

Frequency

Hashimoto's disease affects 1 to 2 percent of people in the United States. It occurs more often in women than in men, which may be related to hormonal factors. Hashimoto's disease is the most common cause of thyroid underactivity (hypothyroidism) in the United States.

Causes

Hashimoto's disease is thought to result from a combination of genetic and environmental factors. Some of these factors have been identified, but many remain unknown.

Hashimoto's disease is classified as an autoimmune disorder, one of a large group of conditions that occur when the immune system attacks the body's own tissues and organs. In people with Hashimoto's disease, white blood cells called lymphocytes accumulate abnormally in the thyroid, which can damage it. The lymphocytes make immune system proteins called antibodies that attack and destroy thyroid cells. When too many thyroid cells become damaged or die, the thyroid can no longer make enough hormones to regulate body functions. This shortage of thyroid hormones underlies the signs and symptoms of Hashimoto's disease. However, some people with thyroid antibodies never develop hypothyroidism or experience any related signs or symptoms.

People with Hashimoto's disease have an increased risk of developing other autoimmune disorders, including vitiligo, rheumatoid arthritis, Addison disease, type 1 diabetes, multiple sclerosis, and pernicious anemia.

Variations in several genes have been studied as possible risk factors for Hashimoto's disease. Some of these genes are part of a family called the human leukocyte antigen (HLA) complex. The HLA complex helps the immune system distinguish the body's own proteins from proteins made by foreign invaders (such as viruses and bacteria). Other genes that have been associated with Hashimoto's disease help regulate the immune system or are involved in normal thyroid function. Most of the genetic variations that have been discovered are thought to have a small impact on a person's overall risk of developing this condition.

Other, nongenetic factors also play a role in Hashimoto's disease. These factors may trigger the condition in people who are at risk, although the mechanism is unclear. Potential triggers include changes in sex hormones (particularly in women), viral infections, certain medications, exposure to ionizing radiation, eating large amounts of foods that contain animal proteins, and excess consumption of iodine (a substance involved in thyroid hormone production).

[Learn more about the genes associated with Hashimoto's disease](#)

- FOXP3
- HLA-DRB1
- PTPN22
- SLC26A4
- TG

Additional Information from NCBI Gene:

- CTLA4

Inheritance

The inheritance pattern of Hashimoto's disease is unclear because many genetic and environmental factors appear to be involved. However, the condition can cluster in

families, and having a close relative with Hashimoto's disease or another autoimmune disorder likely increases a person's risk of developing the condition.

Other Names for This Condition

- Autoimmune chronic lymphocytic thyroiditis
- Autoimmune thyroiditis
- Chronic lymphocytic thyroiditides
- Chronic lymphocytic thyroiditis
- Hashimoto disease
- Hashimoto struma
- Hashimoto syndrome
- Hashimoto thyroidosis
- Lymphocytic thyroiditis

Additional Information & Resources

Genetic Testing Information

- Genetic Testing Registry: Hashimoto thyroiditis (<https://www.ncbi.nlm.nih.gov/gtr/conditions/C0677607/>)

Genetic and Rare Diseases Information Center

- Hashimoto's syndrome (<https://rarediseases.info.nih.gov/diseases/6570/index>)

Patient Support and Advocacy Resources

- National Organization for Rare Disorders (NORD) (<https://rarediseases.org/>)

Clinical Trials

- ClinicalTrials.gov (<https://clinicaltrials.gov/search?cond=%22Hashimoto%27s+disease%22>)

Catalog of Genes and Diseases from OMIM

- HASHIMOTO THYROIDITIS (<https://omim.org/entry/140300>)

Scientific Articles on PubMed

- PubMed (https://pubmed.ncbi.nlm.nih.gov/?term=%28Hashimoto+Disease%5BMAJR%5D%29+AND+%28%28Hashimoto*%5BTIAB%5D%29+AND+%28thyroid*%5BTIAB%5D%29%29+AND+english%5Bla%5D+AND+human%5Bmh%5D+AND+%22last+360+days%22%5Bdp%5D)

References

- Dong YH, Fu DG. Autoimmune thyroid disease: mechanism, genetics and current knowledge. *Eur Rev Med Pharmacol Sci.* 2014;18(23):3611-8. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/25535130>)
- Eschler DC, Hasham A, Tomer Y. Cutting edge: the etiology of autoimmune thyroid diseases. *Clin Rev Allergy Immunol.* 2011 Oct;41(2):190-7. doi:10.1007/s12016-010-8245-8. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/21234711>) or Free article on PubMed Central (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3129418/>)
- Fathima N, Narne P, Ishaq M. Association and gene-gene interaction analyses for polymorphic variants in CTLA-4 and FOXP3 genes: role in susceptibility to autoimmune thyroid disease. *Endocrine.* 2019 Jun;64(3):591-604. doi:10.1007/s12020-019-01859-3. Epub 2019 Feb 15. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/30771152>)
- Hasham A, Tomer Y. Genetic and epigenetic mechanisms in thyroid autoimmunity. *Immunol Res.* 2012 Dec;54(1-3):204-13. doi: 10.1007/s12026-012-8302-x. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/22457094>) or Free article on PubMed Central (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3601048/>)
- Jabrocka-Hybel A, Skalniak A, Piatkowski J, Pach D, Hubalewska-Dydejczyk A. How far are we from understanding the genetic basis of Hashimoto's thyroiditis? *Int Rev Immunol.* 2013 Jun;32(3):337-54. doi: 10.3109/08830185.2012.755175. Epub 2013 Apr 25. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/23617710>)
- Kalantar K, Khansalar S, Eshkevar Vakili M, Ghasemi D, Dabbaghmanesh MH, Amirghofran Z. ASSOCIATION OF FOXP3 GENE VARIANTS WITH RISK OF HASHIMOTO'S THYROIDITIS AND CORRELATION WITH ANTI-TPO ANTIBODY LEVELS. *Acta Endocrinol(Buchar).* 2019 Oct-Dec;15(4):423-429. doi: 10.4183/aeb.2019.423. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/32377237>) or Free article on PubMed Central (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7200107/>)
- National Institute of Diabetes and Digestive and Kidney Diseases (<https://www.niddk.nih.gov/health-information/endocrine-diseases/hashimotos-disease>)
- Ruggeri RM, Giovinazzo S, Barbalace MC, Cristani M, Alibrandi A, Vicchio TM, Giuffrida G, Aguenouz MH, Malaguti M, Angeloni C, Trimarchi F, Hrelia S, Campenni A, Cannavo S. Influence of Dietary Habits on Oxidative Stress Markers in Hashimoto's Thyroiditis. *Thyroid.* 2021 Jan;31(1):96-105. doi:10.1089/thy.2020.0299. Epub 2020 Nov 12. Erratum In: *Thyroid.* 2021 Apr;31(4):709. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/32729374>)

- Tomer Y. Genetic susceptibility to autoimmune thyroid disease: past, present, and future. *Thyroid*. 2010 Jul;20(7):715-25. doi: 10.1089/thy.2010.1644. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/20604685>) or Free article on PubMed Central (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2949235/>)

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