

Thyroid microsomal autoantibodies, TPO

Thyroid gland disease	TPO Abs	TG Abs	TSHR Abs
Hashimoto-thyroiditis	+++	+++	-/+
Morbus Basedow (Graves disease)	++	+	+++
endocrine orbitopathy	++	+	++
disseminated autonomy	+	(+)	-
regional autonomy	+	(+)	-
struma	+	(+)	-

The following tests are available:

- Thyroglobulin autoantibody

General:

Thyroglobulin, a homodimer glycoprotein (MW 304.7 kDa; Chromosome 8q24.2-q24.3) is used by the thyroid gland to produce the thyroid hormones thyroxine (T4) and triiodothyronine (T3). It is synthesized within the follicle epithelial cells of the thyroid gland, absorbed by the exocytotic vesicles and secreted into the follicular lumen. It is a fast available storage form of T4 and T3.

Thyroglobulin autoantibodies belong mainly to the immunoglobulin class IgG and occur in patients with Hashimoto-thyroiditis, primary myxedema, Morbus Basedow, colloidstruma, nodular struma, adenoma und carcinoma as well as in healthy relatives of patients with autoimmune thyroiditis. Moreover they appear in pernicious anemia, Morbus Addison, type-I diabetes mellitus as well and they are detectable in healthy persons.

A predictive importance in terms of a later development of an auto-immune thyroiditis is questionable. However a negative test result almost excludes autoimmune thyroiditis.

Indication: Suspicion of chronic lymphocytic thyroiditis in thyroid peroxidase-auto-antibody-negative patients; implausible thyroglobulin results. Thyroglobulin autoantibodies are less significant in the diagnosis of autoimmune induced thyroid diseases than thyroid peroxidase-autoantibodies (anti-TPO).

Material: 1 ml serum

TAT: same day, FML

Method: ECLIA

Units: IU/ml

Ref.- range: <115

- **Thyroid peroxidase antibodies**

Synonyms: anti-TPO

General:

The thyroid peroxidase (TPO, EC 1.11.1.8; Mr 102.9 kDa; Chromosome 2p25.3) is a membrane protein with a single trans-membrane domain and a carbohydrate fraction of 10%

Due to the complex structure of TPO, the epitopes of most of the antibodies could not be defined. Antibodies against some linear epitopes were detected in patients with Hashimoto thyroiditis. A possible conformation epitope is the amino acid Lys713(K713) and/or its surroundings.

The antibodies against thyroid peroxidase belong preferentially to the immunoglobulin class IgG (mainly IgG1 and/or IgG4). The cause of the development of autoantibodies is not known. The association with HLA-DR3, -DR4 und -DR5 points to a genetic predisposition.

Occurrence: Autoantibodies against TPO are found in patients with chronic lymphatic autoimmune thyroiditis with and without struma and Morbus Basedow.

Moderately elevated autoantibody concentrations can also be found in non-immunogenic thyroid gland diseases like struma, in functional autonomy of the thyroid gland, in other immunological diseases such as Morbus Addison, pernicious anemia, type-I-diabetes mellitus, vitiligo, Hepatitis C (more frequent in women than in men) and in primary biliary cirrhosis. They can also be found in healthy individuals with normal thyroid gland function, mainly persons > 80 years. In autoimmune thyroid gland diseases the concentration of the autoantibodies correlates with the activity of the disease. Pregnant women with type-I-diabetes or thyroid gland disease family history with autoantibodies have a higher risk for gestational diabetes and for postpartum depression.

Indication: TSH increase of an unknown etiology, clarification of a poly-glandular autoimmune disease, familial examination in known autoimmune thyroid gland diseases, risk assessment of the development of a thyroid dysfunction under thyroid gland- or the immune system-influencing medication (e.g. cytokines, interferones), screening in suspicion of postpartum or pregnancy related thyroiditis, differential diagnosis of a hyperthyreosis of unknown etiology.

Material: 1 ml serum

TAT: same day , FML

Method: ECLIA

Units: IU/ml

Ref.- range: < 34

• TSI

General:

TSH receptors belong to the family of G-protein-linked receptors with 7 transmembrane domains. They are closely related to receptors of other glycoprotein hormones such as LH and FSH. The TSH receptor is synthesized as a large single precursor molecule (Mr 86.8; chromosome 14q31.1) and stored in the cell membrane.

The origination of the TSH receptor antibodies is possibly imprinted genetically as well as through environmental influences. The concordance of Morbus Basedow in monozygotic twins is 30–60 %, in dizygotic twins only 3-9%.

Caucasians have a higher rate of HLA-DR3, HLA-DQA1*0501 (relative risk 3.35). A molecular mimicry with *Yersinia enterocolitica* as an environmental influence is discussed (cross reactions with the cell membrane of thyrocytes, TSH receptor antibody induction through *Y. enterocolitica*).

Occurrence: Antibodies against the TSH-receptor are markers for Morbus Basedow, the autoimmune hyperthyroidism with a prevalence of about 1%. With sensitive methods they can be detected in almost all patients.

In a number of Morbus Basedow patients a mutation of the TSH-receptor can be found. Such receptors are more stimulated by TSH autoantibodies and these patients have an adverse etiopathology. Under adequate therapy the autoantibody concentration can decline, it also drops after a partial thyroidectomy or comes down to an adequate level.

Levels rise after ¹³¹I-therapy but will decline after a few months. If the antibodies are no longer detectable after a medicamentous therapy, it is indicative of an inactive process and the therapy can be stopped.

Persisting antibodies point to a persisting autoimmune reaction. The persistence of TSH-receptor antibodies under thyrostatic therapy is classified as a risk parameter for a relapse after

discontinuing medication.

TSH-receptor antibodies of the IgG class can be transmitted from mother to child via the placenta. High antibody titers increase the risk for a newborn hyperthyreosis.

Indication: Detection or exclusion of Morbus Basedow, differential diagnosis of an autonomic hyperthyreosis, prognosis of the etiopathology of Morbus Basedow, therapy monitoring, risk estimation for the development of a hyper-thyreosis in fetuses of mothers with Morbus Basedow.

Material: 1 ml serum

TAT: 5-7 days*

Method: LIA

Ref.- range: < 0.55

For complete list of laboratory test offered at Freiburg Medical Laboratory, please visit <http://www.fml-dubai.com/parameter-listings/>