Immunthyreoiditis Hashimoto

„Woher kommen diese Zustände“

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HT: Was sind das für Zustände?

- Erschöpfung, Müdigkeit, Leistungsknick, Antriebslosigkeit
- Abnehmende Belastbarkeit, fehlende Streßfähigkeit
- Infektanfälligkeit, funktionelle Immundysbalance
- Nervosität, innere Unruhe, Getriebensein, „wired but tired“
- Ein- und Durchschlafstörungen
- Stimmungslabilität, Dysphorie, Depression, Ängste, Panik
- Nachlassen von Kognition und Konzentration, Fahrigkeit
- Gastointestinale Beschwerden („Gastro/Coloskopie o.B.“)
- Muskuläre und Gelenk- Beschwerden („kein Rheuma“)
- Kardiale Beschwerden („Herz kardiologisch gesund“)

HT: Was Patienten gesagt bekommen

- Bei Hashimoto ist die Schilddrüse das Hauptproblem
- Wenn der TSH-Wert normal ist, sind Sie gut eingestellt
- Bei gutem TSH-Wert sollte man sich auch gut fühlen
- Wenn es Ihnen bei guter Schilddrüseneinstellung trotzdem nicht gut geht, hat das psychische Gründe
- Wenn Ihr TSH-Wert gut ist, liegen Ihre Probleme nicht in meinem Fachgebiet („dafür gibt es Psychiater“)
- Einmal Hashimoto, immer Hashimoto
- Hashimoto ohne Antikörper gibt es nicht
- Hashimoto, das haben viele Frauen, wo ist das Problem?

HT: Was Patienten gesagt bekommen

- „Hashimoto ist eine banale Erkrankung, die sich bei normalen Menschen mit Schilddrüsenhormon problemlos behandeln läßt“
- NUK-Arzt: „Hashimoto ist so trivial, daß meine Nuklide dort nicht mal hingehen“
- „Ihr Hashimoto paßt halt nicht zu meiner Schilddrüsen-hormondosierung“
- „Bei vielen Hashimoto-Patienten sitzt das Problem im Gehirn: manche Patienten sind für ihre Erkrankung einfach zu doof“
- „Trio infernale“: Hashimoto, Fibromyalgie und Reizdarm

Autoimmunthyreopathien

Umweltfaktoren	Genetik	Andere
Rauchen	TSHR	Weibl. Geschl.
Iodid	TPO	Parität
Stress	HLA	X-chrom.
Infektionen	CTLA-4	Inaktivierung
Medikamente	PTPN22	
Selen	CD40	
Zink, Eisen	FOXP3	
Vitamin D3	MAGI3	
Coenzym Q10	KALRN	
Omega 3	ATXN2	
Sexualsteroide	BACH2	

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A polymorphism in the promoter region of the selenoprotein S gene (SEPS1) contributes to Hashimoto's thyroiditis susceptibility.

Santos LR[1], Durães C, Mendes A, Prazeres H, Alvelos MI, Moreira CS, Canedo P, Esteves C, Neves C, Carvalho D, Sobrinho-Simões M, Soares P.

Author information

Abstract

CONTEXT: The association between **selenium** and inflammation and the relevance of selenoproteins in follicular **thyroid** cell physiology have pointed to a putative role of selenoproteins in the pathogenesis of autoimmune **thyroid** diseases.

OBJECTIVE: The aim of this study was to evaluate the role of a promoter variation in SEPS1, the selenoprotein S gene, in the risk for developing Hashimoto's thyroiditis (HT).

DESIGN: A case-control study was performed to assess the association of genetic variation in the SEPS1 gene (SEPS1 -105G/A single-nucleotide polymorphism, rs28665122) and HT.

SETTING: The study was conducted in north Portugal, Porto, in the period of 2007-2013.

PATIENTS OR OTHER PARTICIPANTS: A total of 997 individuals comprising 481 HT patients and 516 unrelated controls were enrolled in the study.

MAIN OUTCOME MEASURES: Genetic variants were discriminated by real-time PCR using TaqMan single-nucleotide polymorphism genotyping assays.

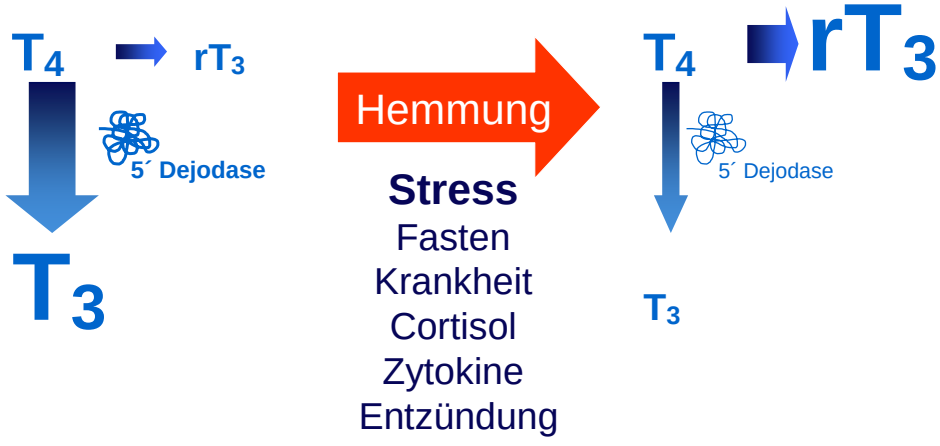
RESULTS: There is a significant association between the SEPS1 -105 GA and AA genotypes and HT [odds ratio (OR) 2.24, confidence interval (CI) 1.67-3.02, P < 5.0 × 10(-7), and OR 2.08, CI 1.09-3.97, P = .0268, respectively]. The A allele carriers are in higher proportion in the patient group than in the control population (46.2% vs 28.1%, P < 5.0 × 10(-7)) with an OR (CI) of 2.22 (1.67-2.97). The proportion of patients carrying the A allele is significantly higher in male patients with HT, representing a 3.94 times increased risk (P = 7.9 × 10(-3)).

CONCLUSION: Our findings support the existence of a link between SEPS1 promoter genetic variation and HT risk.

HT: Extrathyreoidale Assoziationen

- GI-Trakt: Autoimmungastritis, Zöliakie, Gluten-Sensitivität, Helicobact. pylori, PBC, AI-Cholangitis, AI-Hepat.
  M. Crohn, Colitis ulcerosa, kollag. Colitis
- Rheumatologie: Sjögren, RA, SA, SLE, CREST, ITTP
- Haut: Vitiligo, chron. Urticaria, Psoriasis, Pemphigus, Sweet-Syndrom, Dermatomyositis, Sklerodermie, Lichen scl.
- Neurologie: MS, Encephalopathie, Myasthenie, Polymyositis
- Endokrinologie: M. Addison, DM Typ 1, Hypoparathyreoid., vorzeitige Ovarialinsuffizienz, Hypophysitis

Stress hemmt die T_3-Bildung, fördert die rT_3-Bildung



Pathogenese der Immunthyreoiditis

- Genetische Prädispostion
- Umwelteinflüsse (Infektionen, Streß)
- Hormondysbalance ↦ Immundysbalance
- Mitochondriale Störung
- Unkontrollierter oxidativer Streß
- Versagen der antioxidativen Systeme
- Unkontrollierte Apoptose
- Zusammenbruch der Immuntoleranz
- Zytotoxizität (T-Zellen, Komplement)

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Enhanced oxidative stress in Hashimoto's thyroiditis: inter-relationships to biomarkers of thyroid function.

Rostami R[1], Aghasi MR, Mohammadi A, Nourooz-Zadeh J.

Author information

Abstract

OBJECTIVES: Oxidative stress has been implicated in the pathogenesis of several inflammatory and immune-mediated disorders including Hashimoto's thyroiditis (HT). The objectives of the present cross-sectional investigation were to estimate serum glutathione (GSH) status and the activities of its recycling enzymes in HT and to explore their interrelationships with biomarkers of autoimmunity and **thyroid** function.

DESIGN AND METHODS: Newly diagnosed females with HT (n=44) and 58 matched control subjects were recruited. **Thyroid** hormone profile, anti-thyroperoxidase anti-body (TPO-AB), anti-thyroglublin antibody (Tg-AB), **thyroid** volume (Tvol), urinary iodine excretion (UIE), GSH and the activities of glutathione peroxidase (GPx), glutathione reductase and gamma-glutamyltransferase were assessed.

RESULTS: Median UIE in HT was slightly but not significantly higher than that of controls. HT group exhibited higher levels of TSH, TPO-AB, Tg-AB and larger Tvol when compared with controls (P<0.001). The means of GSH and GPx in HT patients were significantly different from those of controls (P<0.001). In HT subjects, significant associations were seen between Tvol on TSH, GSH on TPO-AB, GSH on TSH and TPO-AB titers on TSH, respectively.

CONCLUSIONS: This is the first study to demonstrate a substantial reduction in GSH status in HT subjects. Secondly, the interrelationship between the GSH contents and TPO-AB titers in HT provides a preliminary data to support the notion that GSH diminution is a hallmark of in the events leading to **oxidative stress** activation and the development of immunological intolerance in HT. Further studies are required to elucidate the role of GSH in the etiology of down-regulation of **thyroid** function.

Horm Metab Res. 2013 Sep;45(10):754-8. doi: 10.1055/s-0033-1348262. Epub 2013 Jul 4.

Antioxidant defense in overt and subclinical hypothyroidism.

Reddy VS[1], Gouroju S, Suchitra MM, Suresh V, Sachan A, Srinivasa Rao PV, Bitla AR.

Author information

Abstract

Oxidative stress as a result of disequilibrium between free radical generation and antioxidant status has been implicated in several pathologies including **thyroid** diseases. Studies on antioxidant status in overt (OHT) and subclinical hypothyroidism (SHT) are controversial and limited. The aim of this study was to determine the effect of OHT and SHT on antioxidant status. Thirty-six patients with OHT, 36 patients with SHT, and 39 healthy euthyroid subjects as the control group were included in the study. Plasma levels of malondialdehyde (MDA), reduced glutathione (GSH) and total antioxidant capacity (TAC) as ferric reducing ability of plasma (FRAP), and erythrocyte antioxidant enzymes superoxide dismutase (SOD), glutathione peroxidase (GPx), SOD/GPx ratios, catalase (CAT), and glutathione reductase (GR) were analyzed in all groups. MDA and GPx values were elevated, while GSH, FRAP, SOD, and SOD/GPx ratio were decreased in both patient groups compared with controls. No change in activities of CAT and GR were observed in both the patient groups. Significant differences were observed between OHT and SHT groups with high MDA, GPX and low GSH, FRAP, SOD, and SOD/GPx ratio in OHT group. Thus, hypothyroid patients have a deficient antioxidant defense in the form of decreased activity of SOD, decreased levels of FRAP and GSH along with an increase in GPx activity. The severity of the disease appears to decide the degree of deficiency and our findings also point to this, in the form of decrease in SOD, FRAP, and GSH observed being more in OHT than in SHT patients. Hormonal changes and increased lipid peroxidation, which also vary with severity of disease, appear to contribute to the antioxidant deficiency.

Immunthyreoiditis: Mitochondriopathie

- Onkozyten, Hürthle-Zellen, Ashkanazy-Zellen
- TC vollgestopft mit (dysfunkt.) Mitochondrien
- Defekt in Atmungskette Komplex I
- Defekt in Atmungskette Komplex IV (Cytochrom C-Oxidase)
- Verlust des mitochondrialen Transkriptionsfaktors A
- 4977 bp-Deletion in der mitochondrialen DNA
- TPO-Fehllokalisation in Mitochondrien
- Homologie zwischen TPO und der Häm-Bindungsregion des Cytochrom C-Oxidase-Polypeptids

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Indoleamine 2,3-dioxygenase 1 (IDO1) is up-regulated in thyroid carcinoma and drives the development of an immunosuppressant tumor microenvironment.

Moretti S[1], Menicali E, Voce P, Morelli S, Cantarelli S, Sponziello M, Colella R, Fallarino F, Orabona C, Alunno A, de Biase D, Bini V, Mameli MG, Filetti S, Gerli R, Macchiarulo A, Melillo RM, Tallini G, Santoro M, Puccetti P, Avenia N, Puxeddu E.

Author information

Abstract

CONTEXT: Indoleamine 2,3-dioxygenase 1 (IDO1) is a single chain oxidoreductase that catalyzes tryptophan degradation to **kynurenine**. In cancer, it appears to exert an immunosuppressive function as part of an acquired mechanism of immune escape mediated by the inhibition of lymphocyte proliferation and survival and by the induction of FoxP3+ T regulatory cells.

OBJECTIVE: The objective of the study was to evaluate IDO1 expression in **thyroid** carcinoma and demonstrate its immunosuppressive function in the context of **thyroid** tumors.

SETTING: IDO1 expression was evaluated by quantitative PCR in 105 papillary **thyroid** carcinomas (PTCs), 11 medullary **thyroid** carcinomas, six anaplastic **thyroid** carcinomas, and five **thyroid** carcinoma cell lines (TCCLs), by immunohistochemistry in 55 PTCs and by Western blotting in five TCCLs. FoxP3+ Treg lymphocyte density was evaluated by immunohistochemistry in 29 PTCs. IDO1 inhibitory effect on lymphocyte proliferation was tested in coculture experiments of TCCLs and activated lymphocytes.

RESULTS: IDO1 mRNA expression resulted significantly higher in all the analyzed **thyroid** carcinoma histotypes compared with normal **thyroid**. Interestingly, an increase of IDO1 mRNA expression magnitude could be observed with gain of aggressiveness (PTCs and medullary **thyroid** carcinomas ≪ anaplastic **thyroid** carcinomas). In PTCs, IDO1 mRNA expression magnitude correlated with IDO1 immunostaining intensity in cancer cells and with FoxP3+ Treg lymphocyte density in the tumor microenvironment. IDO1 was expressed in human **thyroid** cancer cell lines in vitro, and FTC-133 cells showed high **kynurenine** concentration in the conditioned medium and a strong suppressive action on the proliferation of activated lymphocytes in coculture experiments.

CONCLUSIONS: For the first time, this study demonstrates a pivotal role of IDO1 in the suppression of lymphocyte function in **thyroid** carcinoma microenvironment.

Tryptophanabbau via Kynurenine

Tryptophanabbau

↓

Serotoninmangel

↓

Melatoninmangel

↓

Müdigkeit, Erschöpfung
Stimmungslabilität, Depression
Erhöhtes Schmerzempfinden
Schlafstörungen
Gastrointest. Störungen
Muskuläre Beschwerden
Fibromyalgie, Migräne

Immunthyreoiditis in der Praxis (n=1460)

Selenmangel (ic):	65%
Vitamin D3-Mangel:	72%
Omega-3/6-Index < 5:	78%
Zinkmangel (ic):	48%
Serotoninmangel (Urin):	71%
Coenzym Q10-Mangel (C-k):	89%
Folsäuremangel:	61%
Eisenmangel:	39%
Sexualhormon-Mangel (>45J)	68%

Low Serum Vitamin D Is Associated with Anti-Thyroid Peroxidase Antibody in Autoimmune Thyroiditis

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· The authors have no financial conflicts of interest.

Purpose: The association between autoimmune thyroid diseases (AITDs) and vitamin D deficiency is controversial. We aimed to evaluate the relationship between serum 25-hydroxy-vitamin D_3 [$25(OH)D_3$] and anti-thyroid antibody levels. **Materials and Methods:** $25(OH)D_3$, anti-thyroid antibodies, and thyroid function measured in 304 patients who visited the endocrinology clinic were analyzed. The patients were subgrouped into the AITDs or non-AITDs category according to the presence or absence of anti-thyroid antibodies. The relationship between anti-thyroid peroxidase antibody (TPOAb) and $25(OH)D_3$ was evaluated. **Results:** The patients with elevated anti-thyroid antibodies had lower levels of serum $25(OH)D_3$ than those who did not (12.6±5.5 ng/mL vs. 14.5±7.3 ng/mL, respectively, $p<0.001$). Importantly, after adjusting for age, sex, and body mass index, a negative correlation ($r=-0.252$, $p<0.001$) was recognized between $25(OH)D_3$ and TPOAb levels in the AITDs group, but this correlation did not exist in the non-AITDs group ($r=0.117$, $p=0.127$). $25(OH)D_3$ level was confirmed as an independent factor after adjusting for co-factors that may affect the presence of TPOAb in the AITDs group. **Conclusion:** $25(OH)D_3$ level is an independent factor affecting the presence of TPOAb in AITDs. The causal effect of $25(OH)D_3$ deficiency to AITDs is to be elucidated.

Thyroid. 2014 Apr;24(4):655-61. doi: 10.1089/thy.2013.0460. Epub 2014 Jan 30.

Low levels of serum vitamin D3 are associated with autoimmune thyroid disease in pre-menopausal women.

Choi YM[1], Kim WG, Kim TY, Bae SJ, Kim HK, Jang EK, Jeon MJ, Han JM, Lee SH, Baek JH, Shong YK, Kim WB.

Author information

Abstract

BACKGROUND: Low serum **vitamin** D levels have been associated with several autoimmune diseases, but their association with **thyroid** autoimmunity is unclear. We evaluated the association of serum **vitamin** D levels with the prevalence of autoimmune **thyroid** disease (AITD).

METHODS: Our cross-sectional study included subjects who underwent routine health checkups, which included assays of serum 25-hydroxy **vitamin D3** [25(OH)**D3**] and anti-**thyroid** peroxidase antibody (TPO-Ab), as well as **thyroid** ultrasonography (US) between 2008 and 2012 at the Asan Medical Center. We defined AITD according to the levels of TPO-Ab and US findings.

RESULTS: A total of 6685 subjects (58% male; 42% female) were enrolled for this study. Overall prevalence of TPO-Ab positivity and both TPO-Ab/US positivity were 10.1% (6.3% male; 15.3% female) and 5.4% (2.3% male; 9.7% female) respectively. In female subjects, mean serum 25(OH)**D3** levels were significantly lower in the TPO-Ab(+) (22.0 vs. 23.5 ng/mL, p=0.030) and TPO-Ab(+)/US(+) groups (21.6 vs. 23.4 ng/mL, p=0.027) compared with the control group, respectively. According to the levels of serum 25(OH)**D3**, the prevalence of TPO-Ab positivity (21.2%, 15.5%, and 12.6% in deficient, insufficient, and sufficient group, respectively; p=0.001) and both TPO-Ab and US positivity (14.7%, 9.9%, and 7.1% in deficient, insufficient, and sufficient group, respectively; p<0.001) decreased in female subjects. Interestingly, this pattern was significant only in pre-menopausal women (p=0.003 and p<0.001; respectively), but not in postmenopausal women. Multivariate analysis indicated that the adjusted odds ratios (OR) for AITD among those in the 25(OH)**D3**-deficient [TPO-Ab(+): OR 1.95, p=0.001; TPO-Ab(+)/US(+): OR 2.36, p<0.001] and -insufficient groups [TPO-Ab(+): OR 1.31, p=0.043; TPO-Ab(+)/US(+): OR 1.50, p=0.017] were significantly increased when compared with the sufficient group.

CONCLUSIONS: The levels of serum **vitamin** D were significantly lower in pre-menopausal women with AITD. **Vitamin** D deficiency and insufficiency were significantly associated with AITD in pre-menopausal women.

Effects of 1,25-dihydroxyvitamin D3 in rats with experimental autoimmune thyroidits

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Abstract: **Objective** To observe the effect of 1,25 $(OH)_2D_3$ on thyroid inflammation and Th1/Th2 cells in rats with experimental autoimmune thyroidits (EAT). **Methods** Forty-eight female Wistar rats were randomly divided into 4 groups, namely the prevention group treated with 1, 25-$(OH)_2D_3$ from 0 to the 6th week (n=10), treatment group with 1,25$(OH)_2D_3$ treatment from the 2nd to the 8th week (n=10) after immune sensitization, positive control group (n=12) and the negative control group (n=16). All the rats were challenged with porcine thyroglobulin for immune sensitization until the 6th or 8th week except for those in the negative control group. In the prevention group and treatment group, the rats received 1,25$(OH)_2D_3$ at 5 μg/kg by intraperitoneal injection every other day, while those in the positive and negative control groups were given peanut oil instead. The thyroid pathologies, serum autoantibody level and cytokine levels were examined after the treatments. **Results** The thyroid gland remained structurally intact in the negative control group. In the positive control group, the thyroid showed obvious inflammatory change with structural disruption and even disappearance of the thyroid follicle. The structure of the thyroid gland follices was intact in the prevention group and treatment group. No significant differences were found in the autoantibody and cytokine levels between the prevention group and negative control group (P>0.05). Compared with the positive control groups, the autoantibody and IFN-γ and IL-12 levels decreased significantly in the treatment group, but the levels of IL-4 and IL-10 were markedly increased (P<0.05). **Conclusion** 1,25 $(OH)_2D_3$ given before the establishment of the EAT model helps maintain structural integrity of the thyroid gland and normal levels of the antibodies and cytokines in rats. 1,25$(OH)_2D_3$ can ameliorate the pathological changes of the thyroid gland and correct the cytokine disequilibrium in rats with EAT.

Key words: 1,25$(OH)_2D_3$; experimental autoimmune thyroidits; Th1/Th2 cells

Endocrinology. 2008 Jul;149(7):3626-34. doi: 10.1210/en.2008-0078. Epub 2008 Mar 27.

Elocalcitol inhibits inflammatory responses in human thyroid cells and T cells.

Borgogni E[1], Sarchielli E, Sottili M, Santarlasci V, Cosmi L, Gelmini S, Lombardi A, Cantini G, Perigli G, Luconi M, Vannelli GB, Annunziato F, Adorini L, Serio M, Crescioli C.

Author information

Abstract

T-helper 1 (Th1) cell-mediated inflammatory responses predominate in the early pathogenesis of Graves' disease (GD), whereas Th2 cell-mediated immunity may play a role in later stages. The chemokine CXCL10 and its receptor CXCR3 are expressed in most **thyroid** glands of early GD patients. Circulating CXCL10 levels inversely correlate with disease duration; CXCL10 maximal expression also correlates with interferon (IFN)gamma levels in recent GD onset. Methimazole (MMI) reduces CXCL10 secretion by isolated thyrocytes, decreases serum CXCL10 levels, and promotes a transition from Th1 to Th2 dominance in patients in GD active phase. **Vitamin** D receptor agonists exhibit antiinflammatory properties and promote tolerance induction. We investigated the effects and the mechanism of action of a nonhypercalcemic **vitamin** D receptor agonist, elocalcitol (BXL-628), compared with MMI on CXCL10 secretion induced by proinflammatory cytokines. Furthermore, we studied the effects of both drugs on Th1, Th17, and Th2 cytokine secretion in CD4+ T cells. ELISA, cytometry, immunocytochemistry, Western blot, and quantitative real-time PCR were used for protein and gene analysis. In human thyrocytes, elocalcitol inhibited IFNgamma and TNFalpha-induced CXCL10 protein secretion more potently than MMI. Elocalcitol impaired both cytokine intracellular pathways, whereas MMI was effective only on the IFNgamma pathway. In CD4+ T cells, elocalcitol decreased Th1- and Th17-type cytokines, and promoted Th2-type cytokine secretion. Elocalcitol and MMI inhibited Th1 cytokine-mediated responses in thyrocytes and CD4+ T cells. In addition, elocalcitol promoted a shift toward a Th2 response. In conclusion, elocalcitol could represent a novel pharmacological tool in the treatment of autoimmune **thyroid** diseases.

Clin Endocrinol (Oxf). 2013 Feb;78(2):155-64. doi: 10.1111/cen.12066.

Selenium and the thyroid gland: more good news for clinicians.

Drutel A[1], Archambeaud F, Caron P.

Author information

Abstract

The **thyroid** is the organ with the highest **selenium** content per gram of tissue because it expresses specific selenoproteins. Since the discovery of myxoedematous cretinism and **thyroid** destruction following **selenium** repletion in iodine- and **selenium**-deficient children, data on links between **thyroid** metabolism and **selenium** have multiplied. Although very minor amounts of **selenium** appear sufficient for adequate activity of deiodinases, thus limiting the impact of its potential deficiency on synthesis of **thyroid** hormones, **selenium** status appears to have an impact on the development of **thyroid** pathologies. The value of **selenium** supplementation in autoimmune **thyroid** disorders has been emphasized. Most authors attribute the effect of supplementation on the immune system to the regulation of the production of reactive oxygen species and their metabolites. In patients with Hashimoto's disease and in pregnant women with anti-TPO antibodies, **selenium** supplementation decreases anti-**thyroid** antibody levels and improves the ultrasound structure of the **thyroid gland**. Although clinical applications still need to be defined for Hashimoto's disease, they are very interesting for pregnant women given that supplementation significantly decreases the percentage of postpartum thyroiditis and definitive hypothyroidism. In Graves' disease, **selenium** supplementation results in euthyroidism being achieved more rapidly and appears to have a beneficial effect on mild inflammatory orbitopathy. A risk of diabetes has been reported following long-term **selenium** supplementation, but few data are available on the side effects associated with such supplementation and further studies are required.

J Formos Med Assoc. 2014 Mar;113(3):155-60. doi: 10.1016/j.jfma.2012.04.003. Epub 2012 Jun 29.

Hemoglobin, iron, and vitamin B12 deficiencies and high blood homocysteine levels in patients with anti-thyroid autoantibodies.

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Author information

Abstract

BACKGROUND/PURPOSE: Autoimmune thyroiditis can be diagnosed by measuring patients' serum levels of **thyroid** stimulating hormone (TSH), anti-thyroglobulin antibody (TGA), and anti-**thyroid** microsomal antibody (TMA). This study evaluated whether there were hematinic deficiencies, high blood homocysteine levels, and serum gastric parietal cell antibody (GPCA) positivity in patients with TGA or TMA.

METHODS: The blood hemoglobin (Hb), iron, vitamin B12, **folic acid**, homocysteine and TSH concentrations and the serum GPCA level in 190 TGA- or TMA-positive patients were measured and compared with the corresponding levels in 190 age- and sex-matched healthy control subjects.

RESULTS: We found that 31 (16.3%), 27 (14.2%), 12 (6.3%), and 2 (1.1%) TGA- or TMA-positive patients had deficiencies of Hb (Men<13g/dL, Women<12g/dL), iron (< 60μg/dL), vitamin B12 (< 200pg/mL), and **folic acid** (< 4ng/mL), respectively. Moreover, 25 (13.2%) and 48 (25.3%) TGA- or TMA-positive patients had abnormally high blood homocysteine level and serum GPCA positivity, respectively. TGA- or TMA-positive patients had a significantly higher frequency of Hb ($p<0.001$), iron ($p<0.001$), or vitamin B12 deficiency ($p=0.001$), of abnormally elevated blood homocysteine level ($p=0.001$), or of serum GPCA positivity ($p<0.001$) than healthy control subjects. Of 190 TGA- or TMA-positive patients, 8 (4.2%) had lower serum TSH level (< 0.1μlU/mL, suggestive of hyperthyroidism), 163 (85.8%) had serum TSH level within normal range (0.1-4.5μlU/mL), and 19 (10%) had higher serum TSH level (>4.5μlU/mL, suggestive of hypothyroidis).

CONCLUSION: There are significant deficiencies of hemoglobin, iron, and vitamin B12, abnormally high blood homocysteine levels, and serum GPCA positivity in TGA- or TMA-positive patients. In addition, the majority (85.8%) of TGA- or TMA-positive patients had euthyroid and only a small portion (14.2%) of TGA- or TMA-positive patients had either hypothyroidism or hyperthyroidism.

Mikronährstoffe und Schilddrüse

ZINK

- **Zinkmangel beeinflußt den peripheren SDH-Metabolismus**
- **Zinkmangel hemmt Aktivität der Typ I-5´-Deiodinase um 67%, reduziert die T4-T3-Konversion und senkt die T3- und fT4-Spiegel um 30%**
- **Zink-Supplementierung normalisiert das erniedrigte fT3 und senkt das erhöhte rT3**

Int J Trichology. 2013 Jan-Mar; 5(1): 40–42.
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PMCID: PMC3746228

Zinc Deficiency Associated with Hypothyroidism: An Overlooked Cause of Severe Alopecia

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HT: Woher kommen solche Zustände?

- Inadäquate Schilddrüsenhormondosis (meist nicht)
- Alles nur psychisch (meist nicht)
- Tatsächliche Ursachenzusammenhänge nicht erkannt (meist)
- Immunsystem ist krank, weniger die Schilddrüse
- Assoziierte Autoimmunerkr. (AIG, Gluten, Sjögren, diverse)
- Entzündungsstreß hemmt T4-T3-Konversion, rT3 steigt
- Erschöpfte zelluläre Antioxidantien-Systeme (Selen, Q10)
- Vitamin D3-Mangel und Mikronährstoff-Defizite
- Dysbalance der gonadalen Steroidhormone (Prog./Androg.)
- Gestörter Fettsäure-Stoffwechsel (Omega3/6)
- Gestörter Tryptophan- und Neurotransmitter-Stoffwechsel

HT: Wie verschwinden diese Zustände ?

- Optimale Schilddrüsenhormon-Einstellung
- Ausgleich eines Selen- und Zinkmangels
- Ausgleich eines Vitamin D3-Mangels
- Ausgleich des Coenzym Q10-Mangels
- Maximierung der Omega3-Fettsäureversorgung
- Ausgleich von Mikronährstoffdefiziten (B12, FS, Eisen etc.)
- Intensive Zufuhr serotoninerger Aminosäuren (T, 5-HTP)
- Intrazelluläre Glutathion-Optimierung (ALA, NAC)
- Ausgleich des gonadalen Steroidhormonmangels (E2,P,A)
- Ernährung: Gluten-arm, Kuhmilch reduzieren