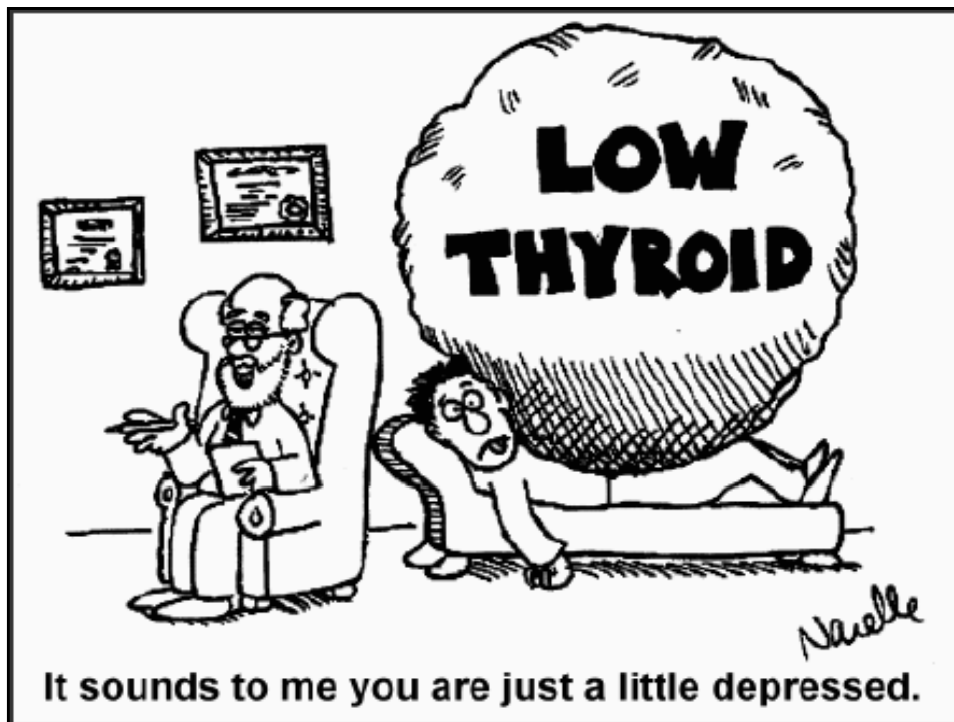


Autoimmune Thyroid Disease FMTown

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Introduction

- 27 million Americans-mostly women-half go undiagnosed
- 90% of adult hypothyroidism due to Hashimoto's
- Functional Model
- Conventional treatment is the same
- Thyroid autoimmune diseases are the most common autoimmune disease in the US affecting 8% of the population

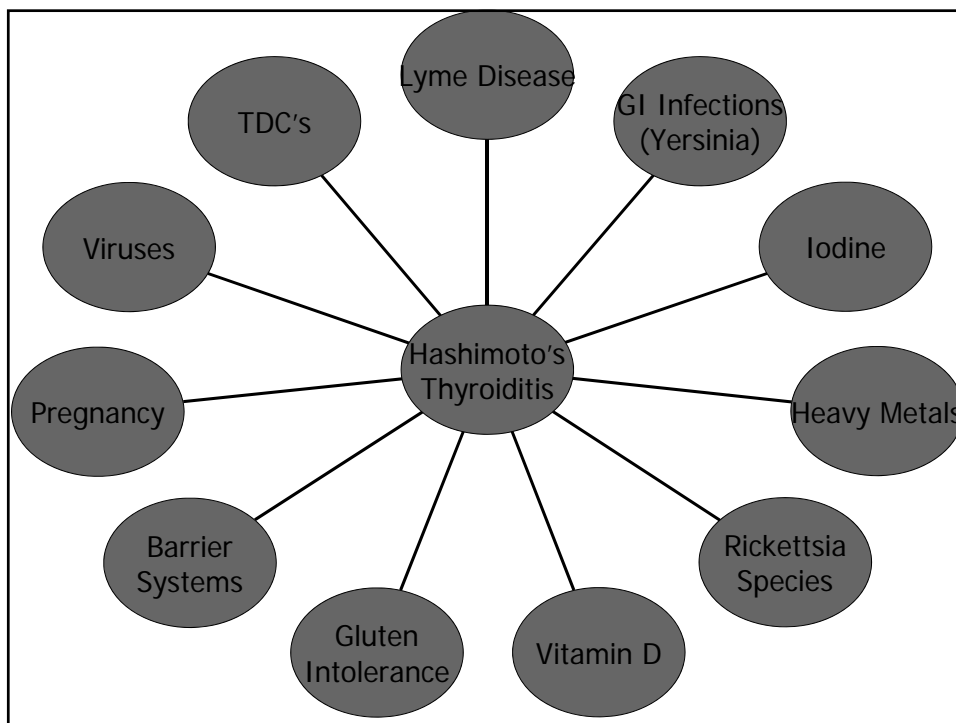


Hypothyroid Symptoms

- Fatigue
- Dry skin and hair
- Cold intolerance, hands and feet
- Constipation
- Inability to lose weight
- Excessive Menstrual flow
- Depression
- Unrefreshing sleep
- Weakness/muscle cramps
- Joint pain/arthritis
- Memory loss
- Depressed immune function
- Postnasal drip
- Blood sugar problems
- Loss of lateral third of eyebrow

Hyperthyroid Symptoms

- Anxiety/Nervousness
- Heart Palpitations
- Weight loss
- Increased appetite
- Increased sweating
- Difficulty sleeping



Autoimmune Thyroid Disease

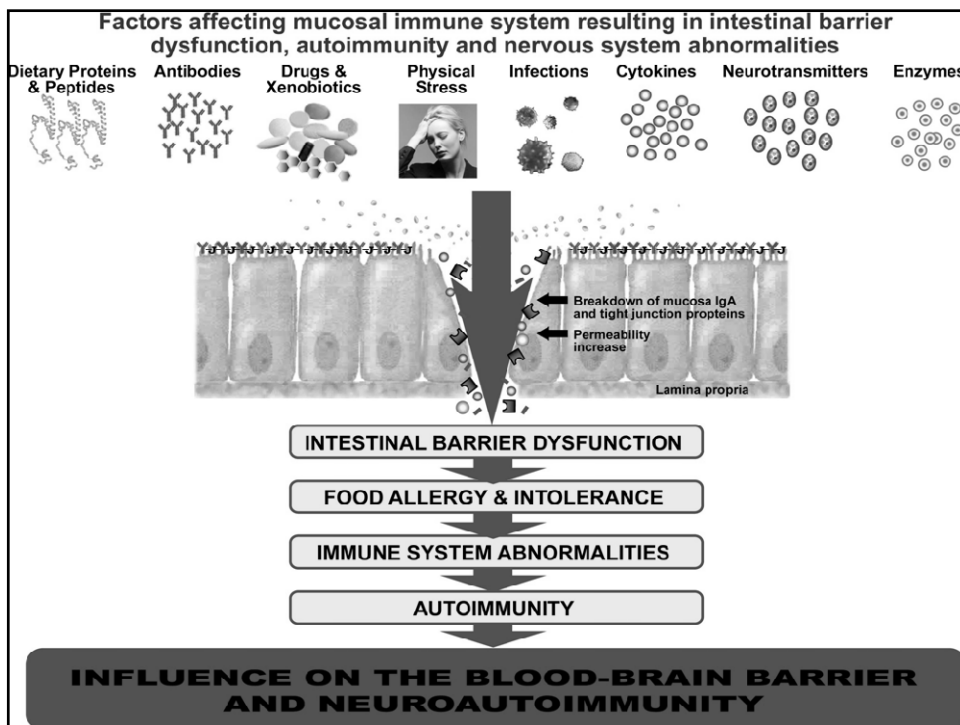
Nikolas Hedberg, D.C., D.A.B.C.I.

Minerva Endocrinol. 2007 Dec;32(4):239-43.Links

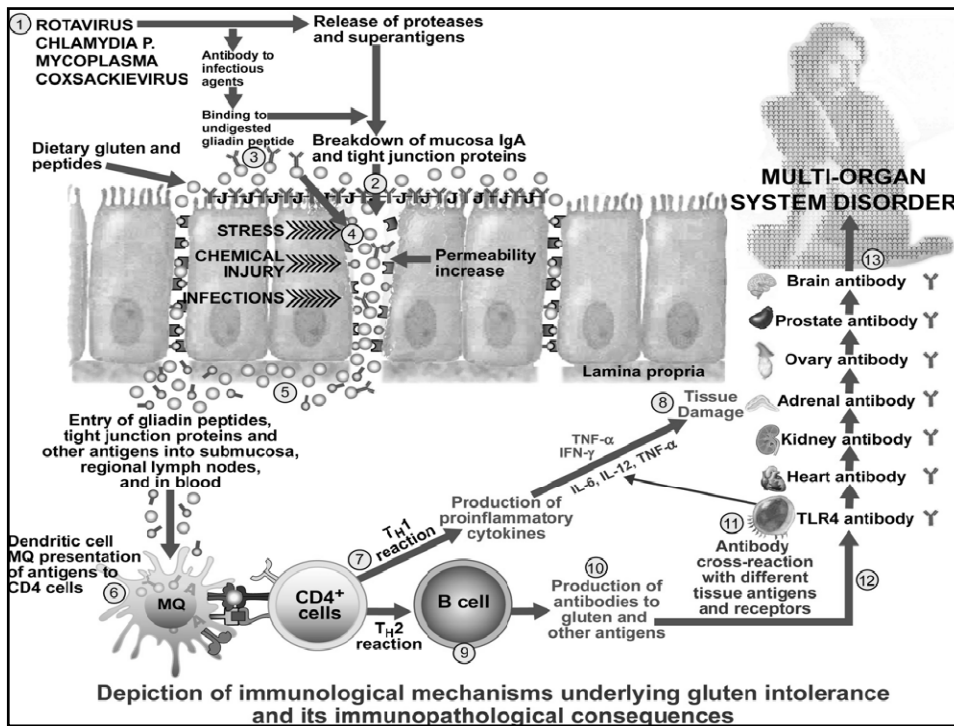
Prevalence of celiac disease in patients with autoimmune thyroiditis.
Luorio R, Mercuri V, Barbarulo F, D'Amico T, Mecca N, Bassotti G, Pietrobono D, Garqiulo P, Picarelli A.

Department of Clinical Sciences, Policlinico Umberto I, University 'La Sapienza', Rome, Italy.

AIM: Many autoimmune disorders are associated to celiac disease (CD) but the association with autoimmune thyroiditis has been more frequently documented; this is in part explained by a shared immunogenetic make-up, and in part caused to time gluten-exposition, as suggested by the significant correlation observed in celiac patients between the increase occurrence of autoimmune diseases and the length of exposure to gluten. **CONCLUSION:** On the basis of this paper, the greater frequency of CD in association to autoimmune thyroid disease suggests that all subjects should be routinely screened for CD.



Autoimmune Thyroid Disease
 Nikolas Hedberg, D.C., D.A.B.C.I.



J Clin Lab Anal. 2006;20(3):109-12.
Vitamin D receptor gene polymorphisms are associated with risk of Hashimoto's thyroiditis in Chinese patients in Taiwan.
 Lin WY, Wan L, Tsai CH, Chen RH, Lee CC, Tsai FJ.
 Department of Life Science, National Tsing Hua University, Hsinchu, Taiwan.

Abstract

The etiology of the autoimmune thyroid, Hashimoto's thyroiditis (HT) is not very clear. However, genetic susceptibility is thought to play a critical role. The vitamin D receptor (VDR)-related endocrine system has been demonstrated to be able to carry out modulation of the immune response. Here, we investigated whether single nucleotide polymorphisms (SNPs) of VDR are associated with HT patients. VDR SNP was detected by polymerase chain reaction (PCR)-based restriction analysis in 109 patients with HT and 90 normal controls. Significant differences were found in the genotype distribution of VDR SNP between Hashimoto's thyroiditis patients and controls ($P=0.0458$). Allelic frequency of the VDR gene distinguished HT patients from controls ($P=0.0089$). The results revealed a significant difference between HT patients and normal controls in VDR SNP and a statistic correlation between VDR-FokI polymorphisms and HT formation. It could be concluded that patients who carry the C/C homozygote of the VDR-FokI gene polymorphism in exon 2 may have a higher risk of developing HT in Chinese patients in Taiwan.

Vitamin D Protocol

- Baseline test
- Optimal 50-100ng/ml, aim for 80-100ng/ml
- Supplement 10,000IU for 2 months and retest. Also include 5,000IU vitamin A, 200mcg K and at least 200IU E.
- Maintain at 4-8,000IU vitamin D
- Obesity, GI inflammation/infections, liver toxicity can impair optimal vitamin D status

Neuro Endocrinol Lett. 2006 Dec;27 Suppl 1:25-30.

Removal of dental amalgam decreases anti-TPO and anti-Tg autoantibodies in patients with autoimmune thyroiditis.

Sterzl I, Prochazkova J, Hrdá P, Matucha P, Bartova J, Stejskal V.

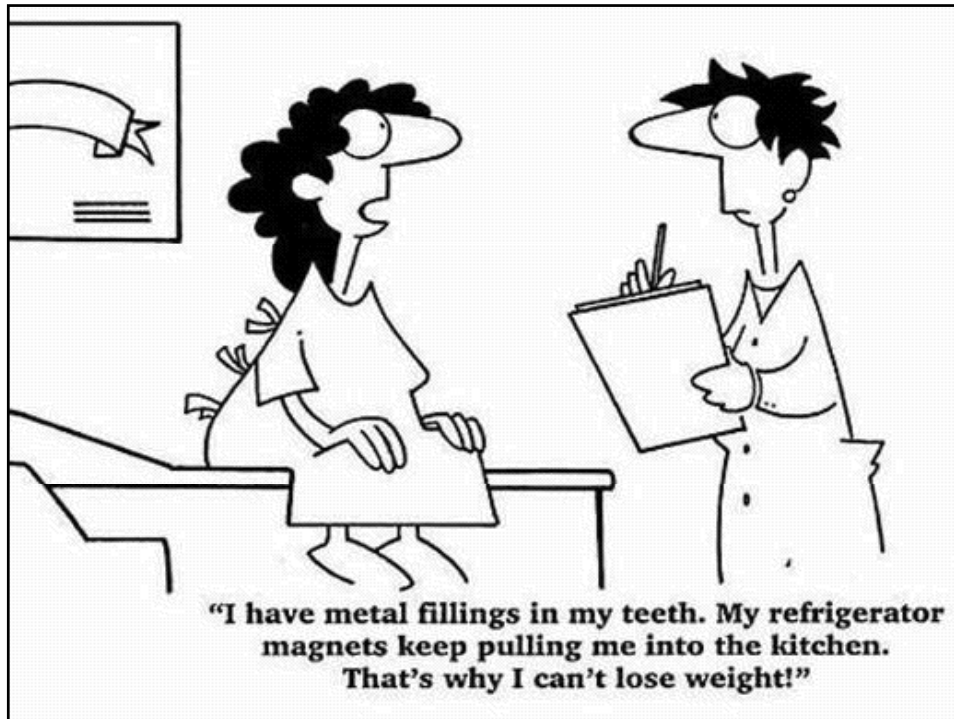
Institute of Immunology and Microbiology, 1st Medical Faculty, Charles University, General Faculty Hospital, Prague, Czech

OBJECTIVES: The impact of dental amalgam removal on the levels of anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) antibodies was studied in patients with autoimmune thyroiditis (AT) with and without mercury allergy.

METHODS: Thirty-nine patients with AT were tested by an optimized lymphocyte proliferation test MELISA for allergy (hypersensitivity) to inorganic mercury. Patients were divided into two groups: Group I (n = 12) with no hypersensitivity to mercury and Group II (n = 27) with hypersensitivity to mercury. Amalgam fillings were removed from the oral cavities of 15 patients with hypersensitivity to mercury (Group IIA) and left in place in the remaining 12 patients (Group IIB). The laboratory markers of AT, anti-TPO and anti-Tg autoantibodies, were determined in all groups at the beginning of the study and six months later.

RESULTS: Compared to levels at the beginning of the study, only patients with mercury hypersensitivity who underwent amalgam replacement (Group IIA) showed a significant decrease in the levels of both anti-Tg (p=0.001) and anti-TPO (p=0.0007) autoantibodies. The levels of autoantibodies in patients with or without mercury hypersensitivity (Group I and Group IIB) who did not replace amalgam did not change.

CONCLUSION: Removal of mercury-containing dental amalgam in patients with mercury hypersensitivity may contribute to successful treatment of autoimmune thyroiditis.



Thyroid. 2004 Nov;14(11):964-6.

Homologies between proteins of *Borrelia burgdorferi* and thyroid autoantigens.

Benvenega S, Guarneri F, Vaccaro M, Santarpia L, Trimarchi F.

Sezione di Endocrinologia del Dipartimento Clinico Sperimentale di Medicina e Farmacologia, Università di Messina, Messina, Italy. s.benvenega@me.nettuno.it

Abstract

Subclinical exposure to microbic antigens that share amino acid sequence homology with self antigens might trigger autoimmune diseases in genetically predisposed individuals via molecular mimicry. Genetic predisposition to Graves' disease (GD) or Hashimoto's thyroiditis (HT) is conferred by HLA loci DR3 or DR5, respectively. *Yersinia enterocolitica* (YE) outer proteins (YOPs) are candidate triggers based on the high prevalence of serum antibodies (Ab) against YOPs in autoimmune thyroid diseases (AITD) and reactivity of these Ab with hTSH-R, suggesting homology between YOPs and hTSH-R. We have reported previously that the spirochete *Borrelia burgdorferi* (Bb) could be another trigger. Residues 112-205, 127-150, 141-260, 299-383 and 620-697 of hTSH-R matched outer surface protein A, flagellar motor rotation protein A, two hypothetical proteins (BBG02 and BBJ08) and DNA recombinase/ATP dependent helicase of *Borrelia* (identity 27-50%, similarity 40-75%). Our data strengthen the hypothesis of Bb and YE as environmental triggers of AITD in genetically predisposed persons through molecular mimicry mechanisms.

The Association between Viruses and Autoimmune Diseases

	EBV	CMV	Herpes-1	Herpes-2	Herpes-6	VZV	Measles
Autism	+	+	+	+	++		++
SLE	+	+			+		
RA	++	++					
Thyroiditis	++						
Sjögrens	++				++		
Myocarditis	+	+					
Multiple Sclerosis	+				++		++
Type-1 Diabetes	+	+					
Guillain-Barré Syndrome	+	+					
Uveitis		++	++	++			
Keratitis			+				
Autoimmune Hepatitis			+			++	
Reiter's Syndrome	+	+	+			+	
Polymyositis	+						
Pemphigus	+					+	
Scleroderma		+					
Psoriasis		+					
ITP	+	+					
IgA Nephritis	++	++					
Glomerulonephritis	++						

+ indicates association
 ++ indicates strong association

Published: 12 January 2009
 Virology Journal 2009, 6:5 doi:10.1186/1743-422X-6-5
 This article is available from: <http://www.virologyj.com/content/6/1/5>

Table 1: Evidence for infection in subacute thyroiditis.

in favour of infection	references	not in favour of infection	references
Levels of data: Epidemiological			
distribution of disease during outbreaks of viral infection	[4,22]	no obvious association with virus infection	[2,9,32]
seasonal distribution from June to September	[2,5,6]		
Serological and/or circulating viral genome			
mumps virus	[22-24]	mumps virus	[30]
coxsackievirus	[24,25,134]	enterovirus	[26]
adenovirus	[24]	HSV-1,	[30]
EBV	[27,28]	parvovirus B19	[30]
measles, chicken pox, CMV	[30]		
influenzae	[14,24]		
rubella	[30,31]		
CMV	[26,29]		
Direct evidence of infection			
human foamy virus	[17]	human foamy virus	[18-20]
mumps	[22]	enterovirus	[26]
		CMV and EBV	[30]

Hashimoto's

Table 2: Evidence for infection in Hashimoto's autoimmune thyroiditis

in favour of infection	references	not in favour of infection	references
Levels of data: Epidemiological			
antithyroid antibodies following subacute thyroiditis	[32,46]	euthyroidism: nonspecific autoimmune response ?	[32,46]
unknown antithyroid antibodies following subacute thyroiditis	[45]		
seasonality of month of birth	[44]		
HTLV-I	[52,53]	SARS: central hypothyroidism	[115]
HIV	[65]	HIV	[67,68]
non-HIV retrovirus	[69]		
congenital rubella	[88,89,91]	congenital rubella	[90]
HCV, HBV	[107,108,110]	HCV	[111,112]
enterovirus infection during pregnancy	[120]	measles-mumps-rubella vaccination	[51]
Serological and/or circulating viral genome			
HTLV-I	[54-58,60,137]	HIAP-I	[78]
congenital and acquired rubella	[88-90,92-94]		
EBV	[99,100]		
Parvovirus	[104]		
Direct evidence of infection			
HTLV-I	[59]	HFV	[19]
rubella	[87]	CMV	[97]
HSV	[97]	Enterovirus: RNA detected in various thyroid disease	[119]
Parvovirus	[103]		
EBV	[132]		

Grave's

Table 3: Evidence for infection in Grave's disease

in favour of infection	references	not in favour of infection	references
Levels of data: Epidemiological			
seasonality of month of birth	[44]		
higher diagnosis and relapse rate in spring and summer	[41,42]		
geographical distribution	[43]		
antibodies or disease onset following subacute thyroiditis	[47-50]	nonspecific response to the inflammatory reaction ?	[32]
HTLV-I	[62]		
HIV	[65]	lack of anti-thyroid antibodies before the beginning of HAART	[75]
Serological and/or circulating viral genome			
HTLV-I	[57,60-62]		
HIAP-I	[78]		
HFV	[81,84]	HFV	[20,82,83]
parvovirus	[106]	Enterovirus	[121]
HHV6, HHV7	[101]		
Direct evidence of infection			
HTLV-I	[64]		
HIV-I	[70]	SV40	[72,73]
		HIAP-I	[79]
HFV	[19,80]	HFV	[83]
SV40	[85]	HSV, CMV	[97]

Anti-Virals

- Monolauric acid
- Olive Leaf Extract
- Colloidal Silver
- Larrea
- Garlic (Allicillin)
- Zinc
- Hyssop
- Shisandra berry
- Raspberry leaf

Helicobacter. 2010 Dec;15(6):558-62. **Identification of a correlation between Helicobacter pylori infection and Graves' disease.**

Bassi V, Santinelli C, Iengo A, Romano C.

BACKGROUND: Viral and bacterial antigens have been suspected to be able to mimic the antigenic profile of the thyroid cell membrane and to play an important role in the onset of the autoimmune diseases, such as Graves' disease and Hashimoto thyroiditis. The Helicobacter pylori infection is worldwide diffused and is present in the developed countries up to 50% of the population. The presence of the cytotoxin-associated gene A antigens identifies the most virulent strains of the bacterium. Previous studies have demonstrated the possible correlation between the Helicobacter pylori and Hashimoto's thyroiditis but these results are controversial.

AIMS: We studied the prevalence rate of this bacterium in the Graves' disease and two selected subgroups such as the hyperthyroid patients, at the first time of diagnosis, and the euthyroid methimazole-treated patients.

METHODS: We analyzed Helicobacter pylori in fresh stool samples with an enzyme immunoassay method and the presence of cytotoxin-associated gene A antigens with a serological test.

RESULTS: Our results show that a significative increased rate of prevalence is present in Graves' patients, when the disease is ongoing, with an overall prevalence of the strains expressing the cytotoxin-associated gene A antigens compared to the control group.

CONCLUSIONS: The association between the Helicobacter pylori and Graves' disease suggests a possible role of this bacterium in the onset and/or the maintenance of the disease.

Anti-Thyroid Herbals

- Thyrocalm:
- Bugleweed dry extract 8:1
(*Lycopus virginicus*)
- Lemon Balm fresh extract 8:1
(*Melissa officinallis*)
- Work by regulating Thyroid Stimulating Immunoglobulin (TSI)

L-carnitine

- Taken from *The Thyroid Alternative*:
- L-carnitine is a peripheral antagonist of thyroid hormone action. In particular, L-carnitine inhibits both T3 and T4 entry into cell nuclei. This is relevant because thyroid hormone action is mainly mediated by specific nuclear receptors. In the randomized trial, we showed that 2 and 4 grams per day of oral L-carnitine are capable of reversing hyperthyroid symptoms. L-carnitine was acting in the periphery, namely as an inhibitor of thyroid hormone action in thyroid hormone target tissues, and not at the level of the thyroid gland as an inhibitor of thyroid hormone synthesis. Overall the two doses of carnitine (2 and 4 grams) were equally effective in reversing hyperthyroid symptomatology; asthenia, nervousness, and palpitations were the symptoms that benefited the most. Amelioration occurred 1 or 2 weeks after commencement of carnitine.
- One teaspoon with breakfast and dinner (1,500mg/teaspoon)

Goitrogens

- Soybeans (and soybean products such as tofu)
- Pine nuts
- Peanuts
- Millet
- Strawberries
- Pears
- Peaches
- Spinach
- Bamboo shoots
- Radishes
- Horseradish
- Sweet Potatoes
- Vegetables in the genus Brassica
 - Bok choy
 - Broccoli
 - Broccolini (Asparagions)
 - Brussels sprouts
 - Cabbage
 - Canola
 - Cauliflower
 - Chinese cabbage
 - Choy sum
 - Collard greens
 - Kai-lan (Chinese broccoli)
 - Kale
 - Kohlrabi
 - Mizuna
 - Mustard greens
 - Rapeseed (*yu choy*)
 - Rapini
 - Rutabagas
 - Tatsoi
 - Turnips

J Autoimmun. 2009 Nov-Dec;33(3-4):183-9. Epub 2009 Oct 9.

Environmental triggers of autoimmune thyroiditis.

Burek CL, Talor MV.

Johns Hopkins Medical Institutions, Baltimore, MD 21205, USA. lburek@jhmi.edu

Autoimmune thyroiditis is among the most prevalent of all the autoimmunities.

Autoimmune thyroiditis is multifactorial with contributions from genetic and environmental factors. **The best-established environmental factor is excess dietary iodine.** Increased iodine consumption is strongly implicated as a trigger for thyroiditis, but only in genetically susceptible individuals. However, excess iodine is not the only environmental agent implicated as a trigger leading to autoimmune thyroiditis. There are a wide variety of other synthetic chemicals that affect the thyroid gland or have the ability to promote immune dysfunction in the host. These chemicals are released into the environment by design, such as in pesticides, or as a by-product of industry. Candidate pollutants include polyaromatic hydrocarbons, polybrominated biphenols, and polychlorinated biphenols, among others. Infections are also reputed to trigger autoimmunity and may act alone or in concert with environmental chemicals.

Autoimmun Rev. 2002 Feb;1(1-2):97-103.

Iodine: an environmental trigger of thyroiditis.

Rose NR, Bonita R, Burek CL.

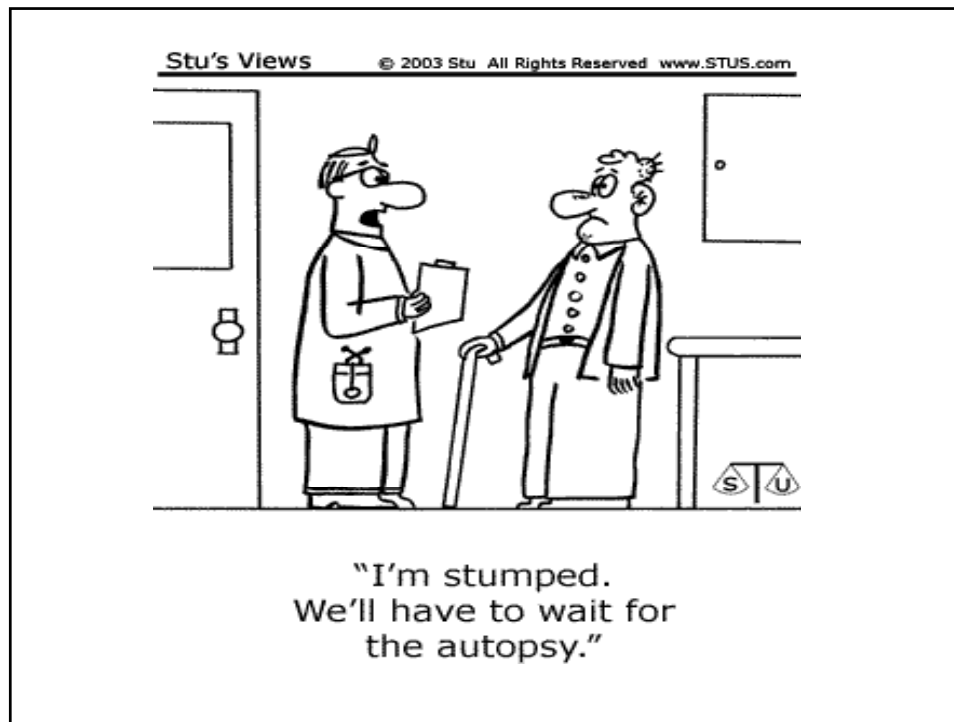
MCP Hahnemann University School of Medicine, 2900 Queen Lane, Philadelphia, PA
19129, USA. nrrose@jhsph.edu

Abstract

Like most autoimmune diseases of humans, chronic lymphocytic (Hashimoto's) thyroiditis results from the combination of a genetic predisposition and an environmental trigger. **A body of clinical and epidemiologic evidence points to excessive ingestion of iodine as an environmental agent.** In genetically determined thyroiditis in animals, iodine enrichment has been shown to increase the incidence and severity of disease. Its mechanism of action is still uncertain. Using a new animal model of autoimmune thyroiditis, the NOD.H2(h4) mouse, we have been able to show that iodine enhances disease in a dose-dependent manner. Immunochemical studies suggest that iodine incorporation in the thyroglobulin may augment the antigenicity of this molecule by increasing the affinity of its determinants for the T-cell receptor or the MHC-presenting molecule either altering antigen processing or by affecting antigen presentation.

Iodine

- Thyroid. 2008 Jun;18(6):667-8.
- **The average of dietary iodine intake due to the ingestion of seaweeds is 1.2 mg/day in Japan.**
- Nagataki S.
- Also read Dr. Jeff Moss's iodine series from www.mossnutrition.com



■ Int J Androl. 2008 Apr;31(2):209-23. Epub 2008 Jan 22.

- **Thyroid disrupting chemicals: mechanisms and mixtures.**
- **Crofton KM.**
- Neurotoxicology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development, US Environmental Protection Agency, Research Triangle Park, NC 27711, USA. crofton.kevin@epa.gov
- Environmental contaminants are known to act as thyroid disrupting chemicals (TDCs). Broadly defined, TDCs are xenobiotics that alter the structure or function of the thyroid gland, alter regulatory enzymes associated with thyroid hormone (TH) homeostasis or change circulating or tissue concentrations of THs. TDCs include a wide range of chemical structures that act through a variety of mechanisms. Concern about TDCs has increased because of the critical role that thyroid hormones play in brain development.

TABLE 1. EXAMPLES OF CHEMICALS THAT CAN INTERFERE WITH THE THYROID SYSTEM

Inhibit iodide uptake	ClO ₄ , ClO ₃ , NO ₃ , thiocyanate
Inhibit TPO activity	PTU, methimazole, isoflavones
Inhibit deiodinase	PCBs, iapanoic acid, thiouracils
Displace T4 from TTR	Hydroxylated PCBs, PBDEs
Activate liver UDPGTs	AhR agonists, agonists of PXR/CAR
Direct action on TR	PCBs, PBDEs(?), BPA, triclosan

Conclusion

- Autoimmune thyroid disease requires a thorough functional medicine analysis
- Test all patients with thyroid imbalances for autoimmunity: thyroid peroxidase and anti-thyroglobulin antibodies
- Identification of underlying cause is vital
- The Thyroid Alternative:
www.drhedberg.com