Hashimotos, Selenium and Iodine, Part Two

Posted on **March 19 2013**

Hashimotos, Selenium and Iodine, Part Two by Jeffrey Dach MD

For Part One, Click Here. For Part Three Click Here

A previous **article** (Part One) discussed Selenium supplementation is beneficial for patients with Hashimoto's Thyroiditis. Four clinical studies showed TPO and Thyroglobulin antibody levels decrease after selenium supplementation.

This article is Part Two of this series, further discussing the role of Iodine in Hashimoto's Thyroiditis, a.k.a. "The Iodine Controversy".

Left Image: Iodized salt courtesy of Morton Salt company.

Voices in Opposition to Iodine Supplementation in Hashimoto's

Vocal opponents to the use of Iodine supplementation for Hashimoto's patients include **Datis Kharrazian** ("Dr. K"), author of the 2010 book "Why Do I Still Have Thyroid Symptoms?", and Chris Kresser of The Healthy Skeptic in his **post** "Iodine for hypothyroidism: like gasoline on a fire?". (1-2). They quote the work of **Rose**, **Yoon** and and others who report that Iodine is a trigger for autoimmune thyroid disease. (4-7)

Iodine and the Thyroid Peroxidase Enzyme Activity

Followers of **Datis Kharrazian** ("Dr. K") and Chris Kresser say that Iodine is a stimulator for the thyroid peroxidase enzyme (TPO). This is contrary to published animal and human studies which examine the effect of Iodine on TPO enzyme activity, and report Iodine **inhibits** the activity of the TPO enzyme. More on this later.

Iodine Worsens Hashimoto's Thyroiditis
There is No Doubt – However Selenium Protects

The medical literature is full of studies showing Iodine supplementation worsens autoimmune thyroiditis, with measurable increases in TPO and Thyroglobulin antibody levels.(4-7) And, many practitioners, including myself, have seen this in actual clinical practice. In addition, I have seen cases of transient hyperthyroidism, called "Hashitoxicosis" after iodine ingestion. There is little doubt about this. Dr. David Brownstein, author of a book on Iodine, and strong advocate of Iodine supplementation states in an interview:

> *"I agree that iodine can aggravate autoimmune thyroid conditions. Iodine supplementation in those that have an autoimmune thyroid problem can be akin to pouring gas over a fire."(3)*

Proponents of Iodine for Hashimoto's

On the other hand, proponents of Iodine such as Drs. Guy E. Abraham, David Brownstein , Jorge D. Flechas and David Derry have successfully used Iodine treatment in many patients with Hashimoto's. Iodine message boards, such as the Yahoo Iodine Group (with 3900 members) report many Hashimoto's patients doing well and recovering on Iodine Supplementation.

Who is Right?

Who is right in this question? How can these two diametrically opposed views be reconciled?

Selenium Protects the Thyroid Gland in Hashimoto's

The answer is the role of selenium. Selenium deficiency is the underlying prerequisite for iodine induced thyroid damage in Hashimoto's Thyroiditis. Selenium supplementation is protective and prevents thyroid damage from iodine. Iodine opponents such as Dr K and Chris Kresser as well as the studies they quote tend to ignore the role of selenium.

Renato Iwakura Reviews the Literature

An excellent review of Iodine and Hashimoto's can be found in a two part article by Mario Renato Iwakura, a Brazilian engineer and Hashimoto's thyroiditis patient who is intimately familiar with the hypothyroidism literature. (8-9) (see part one, see part two)

He concludes:

> *"A survey of the literature suggests that Hashimoto's* ***is largely unaffected by iodine intake.*** *However, the literature may be distorted by three circumstances under which iodine increases may harm, and iodine restriction help Hashimoto's patients:*
>
> *1. Selenium deficiency causes an intolerance of high iodine.*

*2. Iodine intake via seaweed is accompanied by **thyrotoxic metals and halides**.*

3. Sudden increases in iodine can induce a reactive hypothyroidism.

***All three of these negatives can be avoided by supplementing selenium along with iodine, using potassium iodide** rather than seaweed as the source of iodine, and increasing iodine intake gradually." endquote Renato Iwakura.*

Iwakura quotes animal studies which support his conclusion from Drs. Xu and Yang. (10-12) In these studies, animals (mice) were given varying amounts of iodine as well as varying amounts of selenium. Dr Xu concludes in his 2011 report (10):

*"Conclusion: Excess iodine intake can cause an autoimmune thyroiditis that bears all the characteristics of Hashimoto's. However, **in animal studies this occurs only if selenium is deficient** or in excess. Similarly, in animal studies very high iodine intake can exacerbate a pre- existing autoimmune thyroiditis, **but only if selenium is deficient** or in excess. With optimal selenium status, thyroid follicles are healthy, goiter is eliminated, and autoimmune markers like Th1/Th2 ratio and CD4+/CD8+ ratio are normalized over a wide range of iodine intake. "(10)*

In addition, human studies such as this 2007 report by Fan Yang in the European Journal of Endocrinology concluded:

*"Chronic iodine excess does not apparently increase the risk of autoimmune hyperthyroidism or influence the incidence and outcome of subclinical hyperthyroidism, which suggests that chronic excessive iodine intake may not be involved in the occurrence of autoimmune hyperthyroidism as an **environmental factor. " end quote Fan Yang***

Role of Selenium in Hashimoto's

Dr Elias E. Mazokopakis from Greece reports in 2007:

Selenium (Se) supplementation in patients with AITD (autoimmune thyroid disease), including HT (hyperthyroidism), seems to modify the inflammatory and immune responses, probably by enhancing plasma glutathione peroxidase (GPX) and thioredoxin reductase (TR) activity and by decreasing toxic concentrations of hydrogen peroxide (H2O2) and lipid hydroperoxides, resulting from thyroid hormone synthesis. (14)

Benefits of Iodine, Is Iodine the next Vitamin D ?

Iodine is an essential nutrient, and globally, Iodine deficiency is a massive health problem. Some researchers believe that Iodine supplementation will become the "next Vitamin D", an example of a vitamin which was initially thought to be toxic, and now is accepted in much higher doses as beneficial for health. (15-16) I tend to agree with this conclusion. Not only is iodine deficiency a leading cause of mental retardation in developing children, and miscarriage in mothers, it is also a cause of thyroid cancer, breast cancer and gastric cancer in adults.(16) (17)

Dr Zimmerman concludes in his 2011 report (16). He says:

> *"Iodine prophylaxis of deficient populations with periodic monitoring is an extremely cost effective approach to reduce the substantial adverse effects of iodine deficiency throughout the life cycle. (16)"*

World Health Organization Definition of Deficiency
Guidelines for Spot Urinary Iodine Levels (15)

50-99 mcg/L – mild iodine deficiency,
20-49 mcg/L – moderate iodine deficiency,
< 20 mcg/L – severe iodine deficiency.

Selenium supplementation is a prerequisite in all patients with elevated anti-thyroid antibody levels and Hashimoto's thyroiditis. Iodine deficiency is a health risk and Iodine supplementation is beneficial. However, Selenium supplementation is required before giving Iodine to the Hashimoto's patient. Selenium is inexpensive and readily available as a supplement in tablet or capsule form. The usual dosage is 200-400 mcg/day of seleno-methionine. Selenium can be toxic at excessive dosage, so it is best to measure selenium blood levels, and work closely with a knowledgeable physician.

For Iodine supplementation in autoimmune thyroiditis (Hashimotos) patients we follow a protocol described **here,** which starts off with selenium supplementation (200-400 mcg/d) for 2-4 weeks. After which, low dose (225mcg/d) iodine supplementation may be started.

For all other without autoimmune thyroid disease and normal antibody levels, we use Iodoral from Optimox available without a prescription on the internet. Again it is best to monitor iodine levels with spot urine iodine testing, and work with a knowledgeable physician for starting dosage.

Update: Is Iodine Safe for Hashimoto's Patients? February 26, 2013 By Marek Doyle

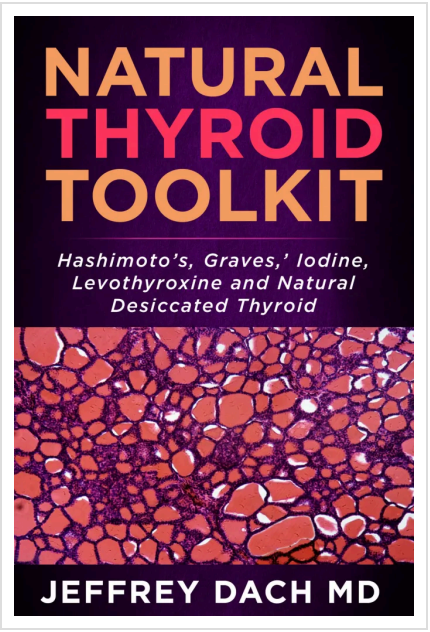
Update: Zhonghua Yi Xue Za Zhi. 2006 Dec 26;86(48):3420-4.
[Long-term effects of high iodine intake: inhibition of thyroid iodine uptake and organification in Wistar rats]. [Article in Chinese] Man N1, Guan HX, Shan ZY, Li YS, Fan CL, Guo XJ, Chen W, Tong YJ, Chong W, Mao JY, Teng WP.

*"The iodine content of thyroid tissue was **negatively correlated with TPO activity**, iodine intake rate, NIS protein positive rate and expression intensity CONCLUSION: Moderate iodine excess continuously suppresses the thyroid iodine uptake and organification, which presents a mechanism for iodine-induced thyroid failure."*

Iodine deficiency, not excess, is the cause of autoimmune thyroid disease.

Update 9/19/21: BMJ Rapid Response: **Iodine deficiency, not excess, is the cause of autoimmune thyroid disease.** The link between iodine intake and thyroid autoimmunity is more complex than Neeru Gupta suggests (Response, 08 April 2016), but increasing evidence implicates iodine deficiency, not excess, as the cause of autoimmune thyroid disease…. 12 April 2016 Peter J Lewis General Practitioner with Special Interest in Integrative Medicine 15 South Steyne, Manly, NSW 2095, Australia

Natural Thyroid Toolkit



If you liked this article, you might like my new book, **Natural Thyroid Toolkit** available on Amazon. If you purchase a book, remember to leave a favorable review. That would be much appreciated. See the book cover, left image.

This article is Part Two of a Series,

For Part One, Click Here.

For Part Three Click Here

Articles with related interest:

Selenium and Thyroid , More Good News !!

Selenium

Selenium, Essential Mineral, Part One

The Case for Selenium, Part Two

Selenium, Your Vitamins Are Killing You, Part Three

Selenium for Hashimotos, Part Four

Hashimoto's Thyroiditis Articles

Hashimotos Thyroiditis and Selenium Part One

Hashimotos, Selenium and Iodine, Part Two

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TSH Wars, Part Two

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Links and References:

Opposed to Iodine use in Hashimoto's

1) http://drknews.com/iodine-and-hashimotos/
Iodine and Autoimmune Thyroid — Datis Kharrazian, DHSc, DC
References Dr K March 8th, 2010

2) http://chriskresser.com/iodine-for-hypothyroidism-like-gasoline-on-a-fire
Iodine for hypothyroidism: like gasoline on a fire? CHRIS KRESSER L.AC MEDICINE FOR THE 21ST CENTURY

3) http://www.thyroid-info.com/articles/brownstein-hormones.htm
Interview with Holistic Doctor David Brownstein, Author of the Miracle of Natural Hormones, Talking About Armour Thyroid and Natural Thyroid Treatments
"I agree that iodine can aggravate autoimmune thyroid conditions. Iodine supplementation in those that have an autoimmune thyroid problem can be akin to pouring gas over a fire."

Iodine Triggers Autoimmune Hashimoto's

4) Iodine: an environmental trigger of thyroiditis Noel R. Rose b,*, Raphael Bonitab, C. Lynne Bureka,b
Autoimmunity Reviews 1 (2002) 97–103 Iodine an environmental trigger of thyroiditis Noel R Rose Autoimmunity Reviews 2002

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.H2h4 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.

Against iodine for hashimoto's Dr Yoon

5) http://www.ncbi.nlm.nih.gov/pubmed/12728462
The effect of iodine restriction on thyroid function in patients with hypothyroidism due to Hashimoto's thyroiditis. Yoon SJ, Choi SR, Kim DM, Kim JU, Kim KW, Ahn CW, Cha BS, Lim SK, Kim KR, Lee HC, Huh KB. Yonsei Med J. 2003 Apr 30;44(2):227-35.

"Lifelong thyroid hormone replacement is indicated in patients with hypothyroidism as a result of Hashimoto's thyroiditis. However, previous reports have shown that excess iodine induces hypothyroidism in Hashimoto's thyroiditis. This study investigated the effects of iodine restriction on the thyroid function and the predictable factors for recovery in patients with hypothyroidism due to Hashimoto's thyroiditis…. In conclusion, 78.3% of patients with hypothyroidism due to Hashimoto's thyroiditis regained an euthyroid state iodine restriction alone. Both a low initial serum TSH and a high initial urinary iodine concentration can be predictable factors for a recovery from hypothyroidism due to Hashimoto's thyroiditis after restricting their iodine intake.

6) hormones.gr/preview.php?c_id=167

The role of iodine in the evolution of thyroid disease in Greece: from endemic goiter to thyroid autoimmunity. HORMONES 2007, 6(1):25-35 . Stelios Fountoulakis, George Philippou, Agathocles Tsatsoulis Department of Endocrinology, University of Ioannina, Ioannina, 45110, Greece HORMONES 2007, 6(1):25-35

The thyroid gland is dependent on dietary iodine for the production of thyroid hormones, normal iodine requirement being about 150- 200μg/day. Long-term deficiency in iodine intake is associated with the development of goiter. When the prevalence of goiter in a population rises above 5-10%, the problem is considered endemic. Greece is a country with a recent history of moderate iodine deficiency, endemic goiter being prevalent in the 1960s in inhabitants of mountainous regions. Despite recognition of the problem, an iodine prophylaxis program was never officially implemented. Instead, "silent iodine prophylaxis" took place during the 1980s and 1990s with Greece's improvement in socioeconomic conditions. This resulted in the elimination of iodine deficiency and a parallel decrease in the prevalence of goiter among schoolchildren in formerly iodine deficient areas.

However, the transition from iodine deficiency to iodine sufficiency or excess was followed by the emergence of autoimmune thyroiditis, especially among young girls, indicating that exposure to excess iodine may trigger thyroid autoimmunity. Thus, the modification of an environmental factor, ie dietary iodine, over the last 40 years in Greece has been associated with changes in the phenotypic expression of thyroid disease from endemic goiter to goiter associated with autoimmune thyroiditis.

Supplementing with Iodine without adding Selenium

7) Reinhardt, W., et al. "Effect of small doses of iodine on thyroid function in patients with Hashimoto's thyroiditis residing in an area of mild iodine deficiency." European journal of endocrinology 139.1 (1998): 23-28. Effect small doses iodine on thyroid function Hashimoto's thyroiditis mild iodine deficiency Reinhard W European journal of endocrinology 1998

Results: Seven patients in the iodine-treated group developed subclinical hypothyroidism and one patient became hypothyroid. Three of the seven who were subclinically hypothyroid became euthyroid again when iodine treatment was stopped. One patient developed hyperthyroidism with a concomitant increase in TBII titre to 17 U/l, but after iodine withdrawal this patient became euthyroid again. Only
one patient in the control group developed subclinical hypothyroidism during the same time period. All nine

patients who developed thyroid dysfunction had reduced echogenicity on ultrasound. Four of the eight patients who developed subclinical hypothyroidism had TSH concentrations greater than 3 mU/l. In 32 patients in the iodine-treated group and 42 in the control group, no significant changes in thyroid function, antibody titres or thyroid volume were observed. Conclusions: Small amounts of supplementary iodine (250 mg) cause slight but significant changes in thyroid hormone function in predisposed individuals.

Mario Renato Iwakura

8) perfecthealthdiet.com/?p=3621
Iodine and Hashimoto's Thyroiditis, Part I by Mario Renato Iwakura is a Brazilian engineer and Hashimoto's thyroiditis patient who is intimately familiar with the hypothyroidism literature.

9) perfecthealthdiet.com/?p=3650
Iodine and Hashimoto's Thyroiditis, Part II by Mario Renato Iwakura

10) www.ncbi.nlm.nih.gov/pubmed/20517655
Biol Trace Elem Res. 2011 Jun;141(1-3):110-8. Epub 2010 Jun 2.

Supplemental selenium alleviates the toxic effects of excessive iodine on thyroid. Xu J, Liu XL, Yang XF, Guo HL, Zhao LN, Sun XF. Source Shenzhen Center for Chronic Disease Control, Shenzhen, 518020, People's Republic of China.

As excessive iodine intake is associated with a decrease of the activities of selenocysteine-containing enzymes, supplemental selenium was hypothesized to alleviate the toxic effects of excessive iodine. In order to verify this hypothesis, Balb/C mice were tested by giving tap water with or without potassium iodate and/or sodium selenite for 16 weeks, and the levels of iodine in urine and thyroid, the hepatic selenium level, the activities of glutathione peroxidase (GSHPx), type 1 deiodinase (D1), and thyroid peroxidase (TPO) were assayed. It had been observed in excessive iodine group that hepatic selenium, the activities of GSHPx, D1, and TPO decreased, while in the groups of 0.2 mg/L, 0.3 mg/L and 0.4 mg/L supplemental selenium, the urinary iodine increased significantly.

Compared with the group of excessive iodine intake alone, supplemental selenium groups had higher activities of GSHPx, D1, and TPO.

We could draw the conclusion that supplemental selenium could alleviate toxic effect of excessive iodine on thyroid. The optimal dosage of selenium ranges from 0.2 to 0.3 mg/L which can protect against thyroid hormone dysfunction induced by excessive iodine intake.

Conclusion Excess iodine intake can cause an autoimmune thyroiditis that bears all the characteristics of Hashimoto's. However, in animal studies this occurs only if selenium is deficient or in excess. Similarly, in animal studies very high iodine intake can exacerbate a pre- existing autoimmune thyroiditis, but only if selenium is deficient or in excess. With optimal selenium status, thyroid follicles are healthy, goiter is eliminated, and autoimmune markers like Th1/Th2 ratio and CD4+/CD8+ ratio are normalized over a wide range of iodine intake.

It seems that optimizing selenium intake provides powerful protection against autoimmune thyroid disease, and provides tolerance of a wide range of iodine intakes.

11) Selenium protects from excess iodine

Selenium supplement alleviated the toxic effects of excessive iodine in mice.

http://www.ncbi.nlm.nih.gov/pubmed/16943608
Xu J, Yang XF, Guo HL, Hou XH, Liu LG, Sun XF.
Biol Trace Elem Res. 2006 Summer;111(1-3):229-38. [abstract only]

“The relationship between the iodine intake level of a population and the occurrence of thyroid diseases is U-shaped. When excessive iodine is ingested, hypothyroidism or hyperthyroidism associated with goiter might develop. The aim of the study was to evaluate the effect of Se supplementation on the depression of type 1 deiodinase (D1) and glutathione peroxidase (GSHPx) activities caused by excessive iodine. D1 activity was assayed by the method with 125I-rT3 as a substrate. Compared to the effect of iodine alone, iodine in combination with selenium increased the activities of D1 and GSHPx. The addition of selenium alleviated the toxic effects of iodine excess on the activities of D1 and GSHPx.”

12) http://www.ncbi.nlm.nih.gov/pubmed/17044649
Effect of selenium supplementation on activity and mRNA expression of type 1 deiodinase in mice with excessive iodine intake.

Yang XF, Hou XH, Xu J, Guo HL, Yinq CJ, Chen XY, Sun XF. Biomed Environ Sci. 2006 Aug;19(4):302-8. [abstract only]

“OBJECTIVE: To investigate the effect of selenium supplementation on the selenium status and selenoenzyme, especially the activity and mRNA expression of type 1 deiodinase (D1) in mice with excessive iodine (EI) intake and to explore the mechanism of selenium intervention on iodine-induced abnormities. METHODS: Weanling female BALB/c mice were given tap water or 3 mg/L of iodine or supplemented with 0.5 mg/L or 1.0 mg/L of selenium in the presence of excessive iodine for 5 months. Selenium status, thyroid hormone level, hepatic and renal D1 activity and mRNA expression were examined.

RESULTS: Excessive iodine intake significantly decreased the selenium concentration in urine and liver, and the activity of glutathione peroxidase (GSH-Px) in liver. Meanwhile, serum total T4 (TT4) increased while serum total T3 (TT3) decreased. Hepatic D1 enzyme activity and mRNA expression were reduced by 33% and 86%, respectively. Renal D1 enzyme activity and mRNA were reduced by 30% and 55%, respectively. Selenium supplementation obviously increased selenium concentration, activity of GSH-Px and DI as well as mRNA expression of D1. However, increasing the supplementation of Se from 0.5 to 1.0 mg/L did not further increase selenoenzyme activity and expression.

CONCLUSION: Relative selenium deficiency caused by excessive iodine plays an essential role in the mechanism of iodine-induced abnormalities. An appropriate dose of selenium supplementation exercises a beneficial intervention.” [Effect of selenium supplement on the disordered lipid metabolism induced by the overdose of iodine in mice]

Iodine NOT A Factor in AutoImmune Thyroiditis in this China Study

13) Yang, Fan, et al. “Chronic iodine excess does not increase the incidence of hyperthyroidism: a prospective community-based epidemiological survey in China.” European Journal of Endocrinology 156 (2007): 403-408.Chronic iodine excess does not increase hyperthyroidism in China Yang Fan European J Endo 2007

In conclusion, iodine supplementation may not lead to an increase in hyperthyroidism in previously mildly iodine-deficient populations. Chronic iodine excess does not apparently increase the risk of autoimmune hyperthyroidism or influence the incidence and outcome of subclinical hyperthyroidism, which suggests that chronic excessive iodine intake may not be involved in the occurrence of autoimmune hyperthyroidism as an environmental factor.

Role of Selenium in Hashimoto’s

14) nuclmed.web.auth.gr/magazine/eng/jan07/8.pdf
Hashimoto’s thyroiditis and the role of selenium. Current concepts Hell J Nucl Med 2007; 10(1): 6-8 by Elias E. Mazokopakis1, Vassiliki Chatzipavlidou

Selenium (Se) supplementation in patients with AITD, including HT, seems to modify the inflammatory and immune responses, probably by enhancing plasma glutathione peroxidase (GPX) and thioredoxin reductase (TR) activity and by decreasing toxic concentrations of hydrogen peroxide (H2O2) and lipid hydroperoxides, resulting from thyroid hormone synthesis. [11,12].

15) www.lmreview.com/articles/view/iodine-the-next-vitamin-d-part-I/

biolargo.com/industry-news/iodine-the-next-vitamin-d-part-ii/

Iodine: The Next Vitamin D? Part I and II

According to the International Council for the Control of Iodine Disorders, WHO and UNICEF, borderline iodine deficiency is indicated by average daily excretion rates of 100 mcg/L per day.

As noted in Part I of this review, the World Health Organization has determined 50-99 mcg/L indicates mild deficiency,
20-49 mcg/L indicates moderate deficiency,
and less than 20 mcg/L indicates severe deficiency.14

For comparison, median urinary iodine excretion in the U.S. population was 145 µg/L during the years 1988 through 1994, which was a significant decrease from the 321 µg/L found in a similar survey two decades prior.10 Among the Japanese, urinary iodine excretion in euthyroid Japanese subjects has been reported to be as high as 9.3 mg per day, and mean urinary iodine levels are approximately twice those reported in the U.S, NHANES 2001-2002 data.11,15

Importance of iodine to developing fetus, children

16) www.ncbi.nlm.nih.gov/pubmed/21802524
Semin Cell Dev Biol. 2011 Jul 23. [Epub ahead of print]
The role of iodine in human growth and development.
Zimmermann MB. Source Laboratory for Human Nutrition, Swiss Federal Institute of Technology Zürich,

Switzerland; The International Council for the Control of Iodine Deficiency Disorders (ICCIDD), Zürich, Switzerland.
Abstract

Iodine is an essential component of the hormones produced by the thyroid gland. Thyroid hormones, and therefore iodine, are essential for mammalian life. Iodine deficiency is a major public health problem; globally, it is estimated that two billion individuals have an insufficient iodine intake. Although goiter is the most visible sequelae of iodine deficiency, the major impact of hypothyroidism due to iodine deficiency is impaired neurodevelopment, particularly early in life. In the fetal brain, inadequate thyroid hormone impairs myelination, cell migration, differentiation and maturation. Moderate-to-severe iodine deficiency during pregnancy increases rates of spontaneous abortion, reduces birth weight, and increases infant mortality. Offspring of deficient mothers are at high risk for cognitive disability, with cretinism being the most severe manifestation. It remains unclear if development of the offspring is affected by mild maternal iodine deficiency. Moderate-to-severe iodine deficiency during childhood reduces somatic growth. Correction of mild-to-moderate iodine deficiency in primary school aged children improves cognitive and motor function. Iodine prophylaxis of deficient populations with periodic monitoring is an extremely cost effective approach to reduce the substantial adverse effects of iodine deficiency throughout the life cycle.

17) eje-online.org/content/140/4/371.long

European Journal of Endocrinology (1999) 140 371–372 Iodide, thyroid and stomach carcinogenesis: evolutionary story of a primitive antioxidant?

Use of Iodine to Treat Hyperthyroidism

18) jcem.endojournals.org/content/90/12/6536.full December 2005 Bal et al. 90 (12): 6536 Endocrine Care Effect of Iopanoic Acid on Radioiodine Therapy of Hyperthyroidism: Long-Term Outcome of a Randomized Controlled Trial C. S. Bal, Ajay Kumar and Prem Chandra

19) www.uptodate.com/contents/iodinated-radiocontrast-agents-in-the-treatment-of-hyperthyroidism
Iodinated radiocontrast agents in the treatment of hyperthyroidism As of this writing, neither iopanoic acid nor ipodate are available in the United States. It is unclear when, or even whether, they will ever again be marketed in the United States.

20) Is Iodine Safe for Hashimotos Patients by Mark Doyle

21) www.ncbi.nlm.nih.gov/pubmed/12487769

iron and selenium – Thyroid. 2002 Oct;12(10):867-78.
The impact of iron and selenium deficiencies on iodine and thyroid metabolism: biochemistry and relevance to public health. Zimmermann MB, Köhrle J. Source Laboratory for Human Nutrition, Swiss Federal Institute of Technology, Zürich, Switzerland.

Several minerals and trace elements are essential for normal thyroid hormone metabolism, e.g., iodine, iron, selenium, and zinc. Coexisting deficiencies of these elements can impair thyroid function. Iron deficiency impairs thyroid hormone synthesis by reducing activity of heme-dependent thyroid peroxidase. Iron-deficiency anemia blunts and iron supplementation improves the efficacy of iodine supplementation. Combined selenium and iodine

deficiency leads to myxedematous cretinism. The normal thyroid gland retains high selenium concentrations even under conditions of inadequate selenium supply and expresses many of the known selenocysteine-containing proteins. Among these selenoproteins are the glutathione peroxidase, deiodinase, and thioredoxine reductase families of enzymes. Adequate selenium nutrition supports efficient thyroid hormone synthesis and metabolism and protects the thyroid gland from damage by excessive iodide exposure. In regions of combined severe iodine and selenium deficiency, normalization of iodine supply is mandatory before initiation of selenium supplementation in order to prevent hypothyroidism. Selenium deficiency and disturbed thyroid hormone economy may develop under conditions of special dietary regimens such as long-term total parenteral nutrition, phenylketonuria diet, cystic fibrosis, or may be the result of imbalanced nutrition in children, elderly people, or sick patients.

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www.ncbi.nlm.nih.gov/pubmed/19594417
Endocr Metab Immune Disord Drug Targets. 2009 Sep;9(3):277-94. Epub 2009 Sep 1.

Role of iodine, selenium and other micronutrients in thyroid function and disorders. Triggiani V, Tafaro E, Giagulli VA, Sabbà C, Resta F, Licchelli B, Guastamacchia E. Source Endocrinology and Metabolic Diseases. University of Bari. Bari, Italy. v.*********@en**.it Abstract

Micronutrients, mostly iodine and selenium, are required for thyroid hormone synthesis and function. Iodine is an essential component of thyroid hormones and its deficiency is considered as the most common cause of preventable brain damage in the world. Nowadays about 800 million people are affected by iodine deficiency disorders that include goiter, hypothyroidism, mental retardation, and a wide spectrum of other growth and developmental abnormalities. Iodine supplementation, under form of iodized salt and iodized vegetable oil, produced dramatic improvements in many areas, even though iodine deficiency is still a problem not only for developing countries. In fact, certain subpopulations like vegetarians may not reach an adequate iodine intake even in countries considered iodine- sufficient. A reduction in dietary iodine content could also be related to increased adherence to dietary recommendations to reduce salt intake for preventing hypertension. Furthermore, iodine intakes are declining in many countries where, after endemic goiter eradication, the lack of monitoring of iodine nutrition can lead to a reappearance of goiter and other iodine deficiency disorders.

Three different selenium-dependent iodothyronine deiodinases (types I, II, and III) can both activate and inactivate thyroid hormones, making selenium an essential micronutrient for normal development, growth, and metabolism. Furthermore, selenium is found as selenocysteine in the catalytic center of enzymes protecting the thyroid from free radicals damage. In this way, selenium deficiency can exacerbate the effects of iodine deficiency and the same is true for vitamin A or iron deficiency. Substances introduced with food, such as thiocyanate and isoflavones or certain herbal preparations, can interfere with micronutrients and influence thyroid function. Aim of this paper is to review the role of micronutrients in thyroid function and diseases.

Control of Iodine Uptake- thyroglobulin down regulates Iodine uptake

www.ncbi.nlm.nih.gov/pubmed/10537174
Endocrinology. 1999 Nov;140(11):5422-30.
Follicular thyroglobulin suppresses iodide uptake by suppressing expression of the sodium/iodide symporter gene.

Suzuki K, Mori A, Saito J, Moriyama E, Ullianich L, Kohn LD. Source Cell Regulation Section, Metabolic Diseases Branch, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, Maryland 20892-1800, USA. Abstract

A major function of the thyrocyte is to take up and concentrate iodide. This is needed for thyroid hormone synthesis and is accomplished by the sodium iodide symporter (NIS), whose expression and activity are up-regulated by TSH. Recently, we reported that follicular thyroglobulin (TG) is a potent suppressor of thyroid-specific gene expression and can overcome TSH-increased gene expression.

We suggested this might be a negative feedback, autoregulatory mechanism that counterbalanced TSH stimulation of follicular function. In this report, we support this hypothesis by coordinately evaluating TG regulation of NIS gene expression and iodide transport.

We show that physiological concentrations of TG similarly and significantly suppress TSH-increased NIS promoter activity, NIS protein, and NIS-dependent iodide uptake as well as RNA levels. We show, in vivo, that TG accumulation at the apical membrane of a thyrocyte facing the follicular lumen is associated with decreased uptake of radioiodide.

It is likely, therefore, that TG suppresses NIS-dependent iodide uptake and NIS gene expression in vivo, as is the case in vitro. RNA levels of NIS and vascular endothelial growth factor/vascular permeability factor, which has been reported to be TSH regulated and possibly associated with TSH- increased iodide uptake, are coordinately decreased by follicular TG as a function of concentration and time. Also, removal of follicular TG from the medium, but not TSH, coordinately returns NIS and vascular endothelial growth factor/vascular permeability factor RNA levels to their TSH-stimulated state. TG accumulated in the follicular lumen appears, therefore, to be a negative feedback regulator of critical TSH-increased follicular functions, iodide uptake, and vascular permeability.

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Jod-Basedow Phenomenon.

El-Shirbiny, Ayda M., et al. “Jod-Basedow syndrome following oral iodine and radioiodinated-antibody administration.” *The Journal of Nuclear Medicine* 38.11 (1997): 1816.Jod Basedow syndrome following oral iodine and radioiodinated-antibody El-Shirbiny J Nuc Med 1997
This is a case of thyrotoxicosis, presumably due to Jod-Basedow syndrome, after stable iodine ingestion for thyroid blockade in a patient with ovarian carcinoma having 131I-labeled monoclonal antibody imaging. With the increased use of radioiodinated antibodies, for therapy and imaging, this possible side effect of excess stable iodine administration should be noted, especially in patients with pre-existing goiter.

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Iodine-Induced Hyperthyroidism (Jod-Basedow Phenomenon) in the Elderly
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Iodine-induced hyperthyroidism, or Jod-Basedow phenomenon, a thyrotoxic condition caused by exposure to increased amounts of iodine, has historically been reported in regions deficient in iodine.1 However, with advances in contrast imaging, this hyperthyroidism has more recently been reported in patients following studies that require

administration of iodine-containing contrast media,2-5 but has received little attention in the elderly,6,7 who frequently undergo such studies. The increasing application of these imaging techniques to evaluate and prognosticate diseases of advanced age, in combination with our growing life expectancy, make the geriatric population especially susceptible to the development of Jod-Basedow hyperthyroidism under this clinical setting. We report a case of Jod-Basedow hyperthyroidism in an elderly patient with no known prior thyroid disease who was exposed to iodinated contrast media for cardiac computed tomography (CT) imaging. Given that weight loss was the only clinical manifestation of hyperthyroidism in our patient, we contend that this phenomenon may be an increasing but underrecognized consequence of modern imaging procedures, which entail larger iodine loads, in the geriatric population.

CASE PRESENTATION An 83-year-old Caucasian man was referred to the Endocrinology Clinic of the University of California, Los Angeles, Healthcare System, complaining of a 7-pound weight loss over one week. On initial presentation to his primary care physician, the patient reported losing approximately one pound per day with only a modest reduction in his appetite. He denied insomnia, heat intolerance, or diaphoresis, nor did he complain of tremor, palpitations, or change in bowel habit. Approximately 10 days prior to the visit, the patient underwent elective coronary imaging, which involved a thin-section CT scan and administration of an iodinated contrast media. Two doses (20 mL, then 140 mL) of a contrast agent, iohexol, were given intravenously. The patient's medications included dipyridamole/aspirin 200/25 mg 1 tablet daily, atorvastatin 10 mg nightly, tamsulosin 0.4 mg daily, and aspirin 81 mg daily. His past medical history was significant for hypertension, hyperlipidemia, and a right-sided cerebrovascular accident 3 years previously, from which he had recovered full motor function. He denied any family history of thyroid or other endocrine disease. He had grown up in Hungary and immigrated to the United States as a young man. On physical exam, the patient was a healthy-appearing gentleman who was alert and in no apparent distress. His weight was 160 pounds, and height was 5 feet 4 inches. He was afebrile, with a temperature of 96.7 degrees Fahrenheit. Pulse rate was 80 beats per minute, and blood pressure was 140/60 mm Hg. There was no eyelid lag, stare, exophthalmos, or scleral icterus. Oropharynx was clear. There was no palpable thyroid goiter, and the gland was nontender without bruits. Cardiovascular exam was significant for a grade II over VI systolic murmur radiating to the axilla and a soft mid-systolic murmur at the upper-left and -right sternal borders. Respiratory and abdominal examinations were unremarkable. Assessment of extremities showed a trace of pitting edema at both ankles, with no cyanosis or clubbing. His hands were warm, without tremor or diaphoresis. Neuromuscular exam revealed symmetrical and normal power and tone, with grossly normal coordination and reflexes. Laboratory data obtained at an outside laboratory five days after the cardiac imaging study showed an elevated total thyroxine (T4) level of 12.8 µg/dL (reference range, 4.5-12.5), elevated free T4 index of 4.9 ng/dL (reference range, 1.0-4.4), and suppressed thyrotropin, or thyroid-stimulating hormone (TSH), of 0.02 µIU/mL (reference range, 0.34-5.6).

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Andreas
on **February 11, 2017 at 2:15 PM** said:

Dear Dr. It is impossible to take 225mcg of iodine and jodide alone eating that from seaweed (i.e Kelp) because it has Halidesn as you has quoted. Secondly, there is not such a tiny drop or pill in Lugol's or Iodoral! One drop has milligrams! Third, if I start with 225mcg after how long should I increase and which is the limite. Dr. Brownstein said, that elevated iodine intake as 50mg is better for Hashimotos because it shut down the antibodies. Could you please elucidate me something of that? Thank you..

Bob Thrasher
on **October 16, 2017 at 11:28 AM** said:

Andreas,you can achieve whatever iodine dose you want with Lugol's. For example, if a drop has 5 mg you can add one drop to a cup of water and then only drink 1/5th if you want a 1 mg dose.