

Iodine and Your Health

David T. Zava, PhD
ZRT laboratory

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Where does it come from? Why is it important for our health?

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Where does it come from? The ocean-algae-phytoplankton-fish The ocean-atmospheric-rain-plants-lactating mammals-milk

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Evolution of Dietary Antioxidants: Role of Iodine
Estuaries and the "IY" and "C" Annual Meeting of Bioger University, Feb. 8, 2007

Sudhakar Yennuri and "Marika Yennuri"
Department of Biology, 355, S. E. Rogers Road, University of Utah, Salt Lake City, Utah 84142

Corresponding Address:
Dr. Sudhakar Yennuri, c/o The Douglas S. Clark Building, 355 S. E. Rogers Road, Salt Lake City, UT 84142-2020

ABSTRACT
The authors review the role of iodine and of other antioxidants in the evolution of marine and terrestrial organisms. It is first hypothesized that iodine was a primary source of antioxidants for all life forms on earth. Iodine was then used in marine organisms and was eventually inherited from primitive green algae and transferred to plants and animals. It is then hypothesized that the iodine deficiency diseases, such as cretinism, are caused by dietary iodine deficiency and are thereby

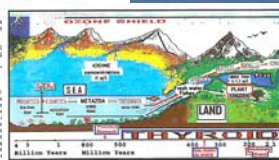
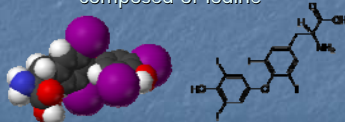


Figure 1. IODINE and EVOLUTION.
Over 3.8 billion years ago, blue-green algae were the first living producers to produce oxygen and store soluble iodine and C20I in the atmosphere. For 700 million years, iodine has been present in the atmosphere. In 450 million years ago, the iodine-rich sea and transferred to other life forms. In 450-500 Mya, some vertebrates evolved an amphibious and aquatic and transferred to land. Then, some primitive green terrestrial plants, a new "terrestrial" iodine source developed, as a reservoir for iodine. In vertebrates, thyroid hormones became active in the amniotes and mammals for a better adaptation to terrestrial environments. (From Yennuri 2004)

Why is iodine important for our health?

About 65% of thyroxine (T4) is composed of iodine



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As a component of thyroid hormones,
Iodine is essential for life

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Iodine Deficiency And Iodine Deficiency Disorders (IDD)

Consequences of Iodine Deficiency on the Fetus,
Neonate, Child, and Adult

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Iodine Deficiency The Fetus

Miscarriage
Stillbirths
Congenital anomalies-cretinism
Brain Damage-mental retardation
Increased perinatal morbidity and mortality



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Iodine Deficiency The Neonate

Endemic cretinism
Neonatal Goiter
Neonatal hypothyroidism
Endemic mental retardation



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Iodine Deficiency The Child/Adolescent



Goiter
(Subclinical) Hypothyroidism
School performance and IQs impaired
Retarded growth, hearing, speech

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Iodine Deficiency The Adult



Goiter
Apathy
Overt Hypothyroidism (low T4, high TSH)
Suboptimal brain function (sluggish thinking)
Low capacity for initiative and decision making
Spontaneous Hyperthyroidism
Iodine-induced Hyperthyroidism

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The Thyroid Gland: Thyroid Hormone Synthesis Starting with Iodine

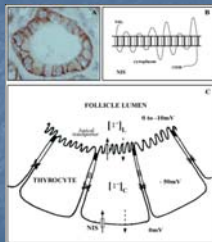


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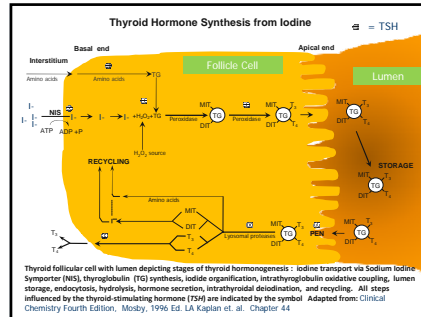
Thyroid Hormone Synthesis from Iodine

Na-Iodine
Symporter
(NIS)



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What Other Roles Does Iodine Play in the Body?

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Uptake of Iodine in Extra-Thyroidal Tissues

Iodine is Concentrated in Many Tissues other than the Thyroid

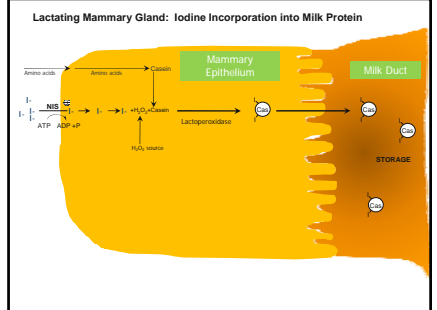
Salivary Glands	Nasopharynx	Eyes (Lacrimal Gland/Ciliary body)
Stomach	Small and Large Intestinal Mucosa	
Pancreas	Kidneys	Liver
Mammary glands	Placenta	Uterus
Ovaries	Testes	Prostate
Skin	White Blood Cells	

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Are you getting enough iodine?

How to assess iodine consumption

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Body fluids commonly used to assess iodine status

Urine

>90% of iodine consumed present in urine in 24 hr

Blood

Saliva

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Urinary Iodine-Methods for Testing

Colorimetric: Sandell-Kolthoff reaction

Chloric Acid Digestion
Ammonium Persulfate Digestion

ICP/MS

Ion Electrode

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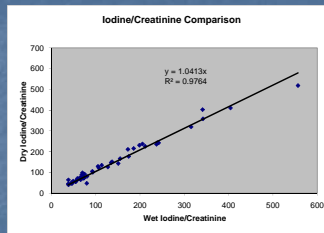
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Wet vs Dry Urine Iodine Levels (n=77)



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How much is enough (sufficient)?

At what level are you considered deficient?

At what level are you considered excessive?

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Optimal Iodine Intake for Thyroid and Extra-Thyroidal Tissues

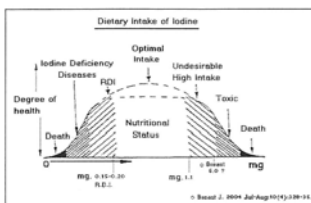
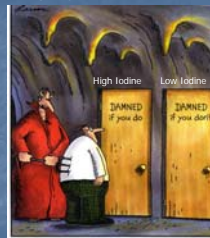


Fig. 5. Daily dietary intake of iodine, according to Food and Nutrition Board, Institute of Medicine, 2001. Note that an optimal iodine intake of 6.0 mg for breast has been reported recently by Kessler in *Breast J.* 2004, 10:328-36.

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THYROID DYSFUNCTION OCCURS WITH BOTH LOW AND HIGH IODINE



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Assessment of Iodine Sufficiency Status by Measurement of Urinary Iodine

Median urinary iodine (µg/L)	Iodine intake	Iodine nutrition
<10	Severely deficient	Severely iodine deficient
10-14	Deficient	Moderate iodine deficiency
15-24	Deficient	Mild iodine deficiency
25-49	Adequate	Optimal
50-99	More than adequate	High iodine intake
>100	Excessive	Excessive iodine intake

* The term "excessive" intake is excess of the amount required to prevent and control iodine deficiency.
 † In ascending order, the figures for median urinary iodine are lower than the iodine requirements because of the iodine excreted in breast milk.

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WHO Recommendations to Achieve Iodine Sufficiency

Table 11. Tolerable upper intake level for iodine (µg/day)

Age group	EC/SCF, 2002	ICH, 2001
1-3 years	200	200
4-6 years	250	300
7-10 years	300	300
11-14 years	450	300
15-17 years	500	500
Adult	600	1100
Pregnant women >19 years	600	1100

http://www.who.int/gb/ebwha/pdf_files/WHA58/WHA58_24-en.pdf

*Iodine deficiency impairs thyroid function. Similarly, iodine excess, including overcorrection of a previous state of iodine deficiency, can also impair thyroid function. The effect of iodine on the thyroid gland is complex with a U-shaped relation between iodine intake and risk of thyroid diseases. Both a low and high iodine intake are associated with an increased risk of thyroid disorders. **Healthy adults can tolerate up to 600-1100 µg iodine/day without any side effects.**

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Thresholds for Iodine Deficiency

Classification of Iodine Deficiency	Urinary Iodine	
	µg/L	µg/g creatinine
No ID	158	150
Marginal ID	126	124
Mild ID	101	100
Intermediate ID	76	75
Moderate ID	50	51
Severe ID	25	25

© Ali et al. Eur J Clin Nutr 57: 1181-1188, 2003. Quantification of urinary iodine: a need for revised thresholds

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Iodine Toxicity Symptoms of Chronic Exposure

Coryza (head cold symptoms)
Salivary gland swelling (increased salivation)
Gastrointestinal irritation
Acneiform dermatitis (Ioderma)
Metallic taste
Gingivitis
Conjunctivitis and oedema of eye lids

Goodman and Gilman, 1970. In *Pharmacological basis of Therapeutics*

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Wolff-Chaikoff Effect (Mechanism)

Excessive iodine intake inhibits the formation of iodinated tyrosine on thyroglobulin, lowers the T4 and T3 plasma levels and raises plasma TSH. These effects are transient and most individuals escape the Wolff-Chaikoff effect within about 48 hrs. A minority of individuals do not escape with persistent exposure to high iodine and develop goiter and become hypothyroid.

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Iodine-Induced Hyperthyroidism-IIIH

In iodine-induced hyperthyroidism excess hormone is produced. This occurs especially in areas with endemic goitre or IDD, after iodine supplementation is introduced in the diet. In areas where iodine is NOT deficient, IIIH is rare with iodine intake of < 5 mg/day (e.g. Japan)

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Iodine Toxicity

Iodine-Induced Hyperthyroidism-IIIH

* IIIH is an occasional consequence of the correction of iodine deficiency, occurring most frequently in older subjects with multinodular goiter. This complication is usually mild and self-limited, but may be serious and occasionally lethal. The most important clinical manifestations are cardiovascular. Thyrotoxicosis can aggravate pre-existing cardiac disease and may also lead to atrial fibrillation, congestive heart failure, worsening of angina, thromboembolism, and rarely, death. In the absence of preexisting cardiac disease, treatment of thyrotoxicosis usually returns cardiac function to normal.Monitoring should be an important part of a successful program of iodization....IIIH, while an issue that needs serious address, is not a reason to stop iodine supplementation in deficient regions *

Dunn JT et al. The Prevention and Management of Iodine-Induced Hyperthyroidism and its Cardiac Features. *Thyroid* 8 (1): 101-106, 1998.

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Iodine and Breast Health

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Iodine Intake in Populations with Low Breast Cancer Risk

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The Average of Dietary Iodine Intake due to the Ingestion of Seaweeds is 1.2 mg/day in Japan

* Total consumption of Kombu in Japan is 20,000 tons per year by 125 million. Since the population of Japan is 130 million, the iodine intake by the ingestion of Kombu is 1.26 mg/d per person.*

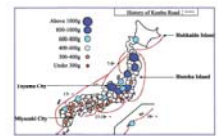


FIG. 2. Annual consumption of Kombu in urban with population concentrated obtained from the National Bureau, Ministry of Health and Consumption. The average consumption per person in Japan is 1.26 mg/d per day. The average consumption per person in the Kanto region is 1.26 mg/d per day. The average consumption per person in the Kanto region is 1.26 mg/d per day. The average consumption per person in the Kanto region is 1.26 mg/d per day.

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Are women in the US placing themselves at higher lifetime risk for breast cancer by eating a low iodine vegan diet?

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Vegans in the US are at Risk for Iodine Deficiency

Iodine Status and Thyroid Function of Boston-Area Vegetarians and Vegans

Angela M. Lacey, Andrew Latta, Suzanne Ho, David E. Braverman, and Jonathan W. Ross

TABLE 2. Concentrations (median [range]) of urinary iodine, perchlorate, and thiocyanate and serum thyroid function of entire data set

	Vegetarians (n = 78)	Vegans (n = 82)	p
Urinary iodine (μg/day)*	14.7 (0.15–1792.0)	76.1 (8.8–964.3)	<0.01
Urinary perchlorate (μg/day)**	4.6 (0.2–21.4)	3.8 (0.2–20.8)	0.70
Urinary thiocyanate (μg/day)**	341 (31–1963)	630 (108–3008)	<0.01
Urinary creatinine (mg/day)**	2.22 (0.049–9)	2.18 (0.039–9)	0.34
TSH (mIU/L)††	1.38 (0.3–5.52)	1.12 (0.14–6.81)	0.46
T4 (ng/dL)††	1.29 (0.93–1.66)	1.24 (0.92–1.67)	0.77

* n = 77 vegetarians and n = 82 vegans due to missing data.
 ** n = 76 vegetarians and n = 82 vegans due to missing data.

Analyses stratified by serum creatinine levels and those on the least concentration median serum perchlorate concentration or highest TSH values range 100 (10) μg/day and highest TSH values range 10–100 (10) μU/L, respectively. †† n = 76 vegetarians and n = 82 vegans. ‡ n = 76 vegetarians and n = 82 vegans. § n = 76 vegetarians and n = 82 vegans. ¶ n = 76 vegetarians and n = 82 vegans. ††† n = 76 vegetarians and n = 82 vegans. †††† n = 76 vegetarians and n = 82 vegans. ††††† n = 76 vegetarians and n = 82 vegans.

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Are women in the US placing their unborn and breast feeding children at higher lifetime risk for mental deficits by eating a low iodine vegan diet?

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Iodine is a trace element essential for the synthesis of triiodothyronine (T₃) and thyroxine (T₄). Inadequate intake of iodine leads to insufficient production of these hormones, which play a vital role in the process of early growth and development of most organs, especially the brain. **The neurological sequelae of iodine deficiency are mediated by thyroid hormone deficiency, varying from normal brain function to a syndrome of severe intellectual disability.** All the basic processes of neurogenesis: cellular proliferation, differentiation, migration, and selective cell death are impaired during period of brain growth spurt. Evidence suggests alterations in synaptology, neurons, myelin sheaths, glial cells, and morphology of cerebrum and cerebellum in severe iodine deficiency. Foetal thyroid ontogenesis occurs after the first trimester. Until then foetus is dependent on maternal T₄. A thyroid dependent event important for subsequent brain development occurs in the beginning of the third trimester of pregnancy.

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From: UNICEF...unicef.org and prevention of childhood diseases

When a foetus does not receive adequate iodine, the developing brain cannot establish as dense a network of interconnections among the main brain cells and intellectual capacity is compromised for life. In extreme cases children can suffer severe mental retardation, but even where iodine deficiency is less severe all children suffer a reduction of 10-15 percent in learning ability at school. **Iodine Deficiency Disorders (IDD) are rightly recognized as the world's leading preventable mental and development disabilities.**

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Studies show iodine is protective of the breast and reproductive tissues

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Animal Studies Suggest Iodine from High-Iodine Foods or Supplements May Be Breast Protective

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RAPID COMMUNICATION Jan 2, Cancer Res. 92, 463–467, May 2003

Seaweed Prevents Breast Cancer?

Hiroaki Funahashi,^{1,2} Tetsuo Inoue,¹ Takahito Matsu,¹ Masamori Sekiya,¹ Kazuki Yokoi,¹ Hiromichi Hayashi,¹ Akihiro Shibata,¹ Takako Hayashi,¹ Mikiho Nishikawa,¹ Norihiko Saito,¹ Yumiko Hibi,¹ Yuka Mizuno,¹ Kyoko Takahashi,¹ Akemi Hayakawa¹ and Ichiro Terauchi¹
 Department of Surgery II and ¹Department Center for Research and Education, Nagoya University School of Medicine, 65 Darumai-cho, Showa-ku, Nagoya 466-8510 and ²Department of Biochemistry, Faculty of Pharmaceutical Science, Saitama University of Japan, 22 Sakigake Fumizono-machi, Utsunomiya, Tokyo 382-0828

To investigate the chemopreventive effects of seaweed on breast cancer, we have been studying the relationship between iodine and breast cancer. We found earlier that the seaweed, wakame, showed a suppressive effect on the proliferation of DMBA (dimethylbenz[a]anthracene)-induced rat mammary tumors, possibly via apoptosis induction. In the present study, powdered wakame was placed in distilled water and left to stand for 24 h at 4°C. The filtered supernatant was used as wakame solution. It showed an extremely strong suppressive effect on rat mammary carcinogenesis when used in daily drinking water without wakame in diet, wakame solution strongly induced apoptosis in 3 kinds of human breast cancer cells. These effects were stronger than those of a chemopreventive agent wakame used to treat human breast cancer. Furthermore, an apoptosis induction was observed in several human mammary cells. In Japan, wakame is widely consumed as a salt, low-iodine food. Our results suggest that wakame has potential for chemoprevention of human breast cancer.

Key words: Breast cancer — Chemoprevention — Wakame (seaweed) — Apoptosis

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RAPID COMMUNICATION Jan 2, Cancer Res. 92, 463–467, May 2003

Seaweed Prevents Breast Cancer?

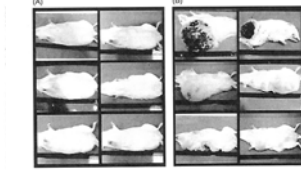


Fig. 2. Mammary tumors were not observed macroscopically in rats of the wakame group (A) despite large tumors in the control group (B).

Key words: Breast cancer — Chemoprevention — Wakame (seaweed) — Apoptosis

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Is Iodine A Gatekeeper of the Integrity of the Mammary Gland?

Carmen Aceves,^{1,2} Brenda Anguiano,¹ and Guadalupe Delgado¹

This paper reviews evidence showing iodine as an antioxidant and anti-proliferative agent contributing to the integrity of normal mammary gland. Seaweed is an important dietary component in Asian communities and a rich source of iodine in several chemical forms. The high consumption of this element (27 times more than in Occident) has been associated with the low incidence of benign and cancer breast disease in Japanese women. In animal and human studies, molecular iodine (I₂) supplementation exerts a suppressive effect on the development and size of both benign and cancer neoplasms. This effect is accompanied by a significant reduction in cellular hyperproliferation. Iodine, in addition to its incorporation into thyroid hormones, is found into anti-proliferative iodolipids in the thyroid called iodolipins, which may also play a role in the proliferative control of mammary gland. We propose that an I₂ supplement should be considered as an adjuvant in breast cancer therapy.

KEY WORDS: mammary gland; iodine; iodolipins; breast cancer; antioxidant; hyperproliferation.

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Cancer Causes and Control 18 (12):121-126, 2007
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Hypothesis: Iodine, selenium and the development of breast cancer

Stephen A. Case,¹ Johannes P. van Natten^{1,4} & Christian van Natten²
¹Special Development Laboratory, Royal Jubilee Hospital, 100 Fort Street, Victoria, BC V8R 1J6, Canada (P: 250) 378-6291; Fax: 250) 378-6222; E-mail: hvannatten@ubchulb.org and ²Department of Biology, University of Victoria, Victoria, Canada; ³Department of Health Care and Epidemiology, Faculty of Medicine, University of British Columbia, Vancouver, British Columbia, Canada (*Author for correspondence)

Received 12 October 2006; accepted in revised form 20 August 2007

Key words: breast neoplasm, iodine, selenium, thyroid disease.

Abstract: In this paper we examine some of the evidence linking iodine and selenium to breast cancer development. Seaweed is a popular dietary component in Japan and a rich source of both of these essential elements. We hypothesize that this dietary preference may be associated with the low incidence of benign and malignant breast disease in Japanese women. In animal and human studies, iodine administration has been shown to cause regression of both iodine-deficient goiter and benign pathological breast tissue. Iodine, in addition to its incorporation into thyroid hormones, is organized into anti-proliferative iodolipids in the thyroid; such compounds may also play a role in the proliferative control of extrathyroidal tissues. Selenium acts synergistically with iodine. All three amino-selenolipids appear to be selenium-dependent and are involved in thyroid hormone regulation. In this way selenium status may affect both thyroid hormone homeostasis and iodine availability. **Conclusion:** Although there is suggestive evidence for a protective role for iodine and selenium in breast cancer, rigorous retrospective and prospective studies are needed to confirm this hypothesis.

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Role of iodine in antioxidant defence in thyroid and breast disease

Peter P.A. Smyth¹
¹Iodine Study Unit, Department of Medicine and Therapeutics, and Conway Institute of Biomolecular and Biomedical Research, University College Dublin, Ireland

Received 7 July 2005
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Accepted 30 September 2005

Abstract: The role played by thyroid homeostasis to iodine regulation is well established. Iodine deficiency may produce conditions of oxidative stress with high TSH producing a level of ROS, which because of both of their size and being small is less lipid-soluble. The oxidative action of these radicals in thyroid cells may depend on their formation in the nucleus and can be amplified to both oxidize and require mechanisms to both prevent and promote of their development and require during iodine related functions. These systems often appear to depend on the same antioxidant enzymes and only only be evident in conditions of iodine deficiency when the activity of selenium containing antioxidant enzymes is impaired. Low selenium reduces some of a role for iodine as an antioxidant in the breast. However the iodine exposure may reduce a protective effect against breast cancer for an iodine rich seaweed containing diet. Similarly thyroid neoplasms may also be associated with iodine exposure. Whether this phenomenon is breast specific and its possible relationship to iodine or selenium status awaits resolution.

Keywords: Antioxidant, thyroid, breast, iodine, thyroid antibodies

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Evolution of Dietary Antioxidants: Role of Iodine

Lecture held at the "Thyroid Club" Annual Meeting of Bologna University, Feb. 6, 2007

Sebastiano Vennart and "Marta Vennart"
Servizio di Igione, ASS. I, Regione Marche, Pesaro/RS (PU) Italy, and ²Department of Child Science, University of Bologna, Italy

Corresponding address:
Dr. Sebastiano Vennart - via Tin Galvani n. 2, 41019-Pesaro/RS (PU), Italy
Tel.: (+39) 0741 928205 - E-mail: vennart@unibo.it

KEY WORDS: Antioxidant; evolution; iodine; iodine; iodine; iodine

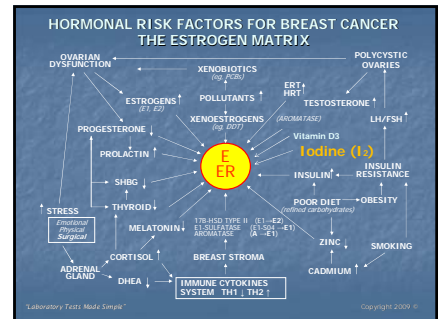
ABSTRACT: The authors review the role of inorganic and organic forms of iodine as an antioxidant in evolution of plants and animals. Iodine is one of the most abundant elements in the diet of marine and terrestrial organisms. It is transported from the diet to the cells via iodide transporters. Iodide, which acts as a peroxidase electron donor through peroxidase enzymes, seems to have an essential antioxidant function in all iodide-concentrating cells from primitive marine algae to more recent terrestrial vertebrates. Thyroxine and selenoiodine have an antioxidant activity via and, through deiodinase enzymes, are donors of iodide and iodolipids of alkenones. Thyroid cells physiologically derived from primitive gastrovascular cells, which during evolution of vertebrates migrated and specialized to uptake and storage of iodide compounds in a new follicular "thyroidal" structure, for a better adaptation to iodine-deficient terrestrial environment. Finally, some animal and human chronic diseases, such as cancer and cardiovascular diseases, fostered by dietary antioxidant deficiency, are briefly discussed.

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Breast Cancer and the Hormonal Matrix

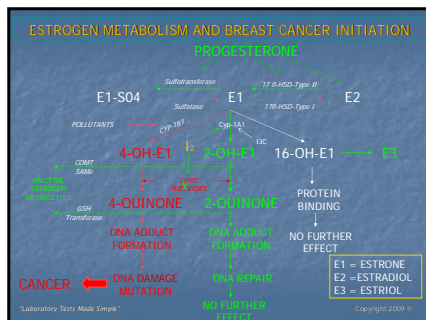
Iodine's Role in Maintaining Homeostasis

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Estrogen Metabolism and Breast Cancer

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Molecular Iodine (I₂) Enhances Safe Estrogen Metabolism

- Induces Cyp -1A1
- Inactivates oxidized peroxilipids

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Progesterone/Iodine Synergy

Estrogen Metabolism and Breast Cancer

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Progesterone increases iodine uptake in the lumen of the uterus

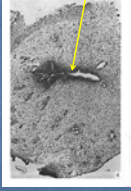
J. Biol. Chem. 1975; 250: 300-303
With Iodine
Printed in Great Britain

THE SITES OF IODIDE CONCENTRATION IN THE OVIDUCT AND THE UTERUS OF THE RAT

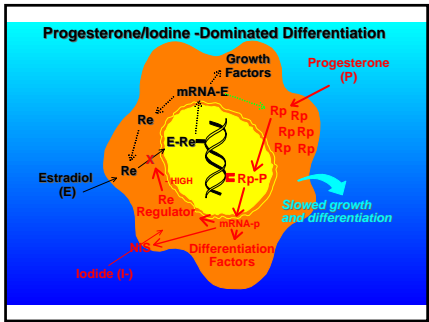
K. BROWN GRANT AND A. W. ROGERS
Medical Research Council Neuroendocrinology Unit, Department of Human Anatomy, South Parks Road, Oxford OX1 3PS

"After treatment with progesterone, the iodine concentrating mechanism appears to be restricted to the luminal epithelium....if one assumes that all the iodide in excess of the general tissue level in the luminal epithelium, which forms about 33% of the total uterine tissue, in the ovariectomized rats, the concentration of iodide in these epithelial cells is hundred times the plasma level."

"Amount of Radioiodine" 1 uCi, 30, 30, 3



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Iodine Deficiency

Breast Cancer Case History

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Case History 1-Breast Cancer

49 y/o premenopausal 195#, 5'5"

Hormone Therapy: Intermittent use of topical Pg (6 days since last used)

Hot Flashes	Mood Swings
Night Sweats	Nervous-Irritable
Memory Lapses	Low Stamina
Fatigue	Swelling/Puffy Eyes
Stress	Nails Breaking/Brittle
Cold Intolerance	
Low Libido	

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Case History 1-Breast Cancer

49 y/o premenopausal 195#, 5'5"

Hormone Therapy: None

Urinary Iodine Status:

Test	Test Results	Normal Range
Iodine (urine)	16 ug/L	100-1100 ug/L
Creatinine (urine)	0.45 mg/ml	0.3-3.5 m/ml
Iodine/Creatinine	36 ug/g Cr.	100-1000 ug/g

Iodine Status: Moderate-Severe Deficiency!

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Case History 1-Breast Cancer

49 y/o premenopausal 195#, 5'5"

Summary

Pre/peri-menopausal patient suffering from a severe iodine deficiency, progesterone deficiency and estrogen dominance. Very recently diagnosed with infiltrating ductal carcinoma-grade III.

Low iodine consistent with symptoms of estrogen dominance/progesterone deficiency and breast cancer.

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