Journal of Endocrine Disorders

Austin Publishing Group

Open Access Full Text Article

Editorial

A Healthy Diet, a Healthy Thyroid

Amit Bhargava*

Division of Endocrinology, Fortis Memorial Research Institute, Haryana 122002, India

***Corresponding author:** Amit Bhargava, Division of Endocrinology, Fortis Memorial Research Institute, Sector 44 (Opposite HUDA City Centre Metro Station) Gurgaon, Haryana 122002, India, Tel: 860-272-9074; Email: abhargava99@yahoo.com

Received: September 26, 2014; **Accepted:** September 30, 2014; **Published:** October 02, 2014

Hypothyroidism is a relatively common endocrine disorder, often encountered in the outpatient setting. It may be either subclinical or overt. An elevated TSH above 10mIU/L with a below normal free thyroxine (T_4) designates overt hypothyroidism, whereas subclinical hypothyroidism is defined by a subnormal TSH in conjunction with a normal free T_4 level. According to the National Health and Nutrition Examination Survey (NHANES III), between1998-2004 the prevalence of subclinical disease in a randomly selected U.S. population above 12 years of age was 4.3%. The prevalence of overt hypothyroidism was 0.3%. The Colorado thyroid disease prevalence survey reported the prevalence of subclinical hypothyroidism to be as high as 8.5%. The prevalence of clinical hypothyroidism was found to be 0.4%. Lastly, Framingham data indicates that 5.9% of women and 2.3% of men over 60 years of age have had TSH levels above 10mIU/L [1].

Screening guidelines vary. The American Thyroid Association (ATA) recommends that all adults be screened for hypothyroidism every 5 years, starting at the age of 35. The American Association of Clinical Endocrinologists (AACE) recommends TSH measurement in the elderly, and the American Academy of Family Physicians (AAFP) advocates screening in asymptomatic patients above the age of 60. On the other hand, the U.S. Preventive Services Task Force (USPSTF) advises against adult screening [1]. Nonetheless, the number of TSH assays ordered annually is bound to increase, thus indentifying more and more individuals with TSH abnormalities. With an ever growing amount of information available at one's fingertips, patients not only look to their physicians for guidance with regards to their medical management, but also for guidance with regards to various environmental/nutritional factors that may affect their thyroid function.

Iodine, a trace element found in rocks, seawater and certain soil, is crucial for the production of thyroid hormone. Data from as far back as the early 1900s shows that thyroid goiters can be prevented by iodine supplementation. Indeed, public health programs such as universal salt iodination have been successful in doing so. The iodine content of most commonly consumed foods is quite low (estimated to be 3-80µg of iodine per serving). Marine animals and plants absorb iodine from sea water, and consequently large consumption of certain types of seaweed can provide an iodine intake of 50-80mg per day [2]. In one study, Teas et al analyzed 12 different species of sea vegetables. They found the iodine content to range from 16µg/g in nori (Porphyra tenera), to greater than 8000µg/g in processed kelp granules [3]. The amount of iodine in diary products is influenced by the use of iodine-containing compounds in fertilizers, livestock feeds and irrigation. Using iodophors to clean teats and milk cans can also increase iodine content. Boiling and baking iodated salt containing foods seems to cause only a small decrease in their iodine content (<10%). Caution must be advised with regards to over supplementation. The ingestion of many grams of iodine can lead to acute iodine poisoning, which can manifest with vomiting, diarrhea, abdominal pain, gastrointestinal irritation and even induce a coma. Excessive intake can also result in a skin disorder called iodermia, which involves the development of a puritic rash, urticaria and acneiform eruptions. In patients with an underlying thyroid disease, high supplementation doses in the microgram range may eventually lead to either hyper- or hypothyroidism [2]. The recommended daily allowance for iodine is outlined in Table 1.

Goitrogens are substances that occur naturally in food and interfere with the metabolism of the thyroid gland. Cruciferous vegetables, such as cauliflower, broccoli, kale, cabbage and turnips, contain glucosinolates. The metabolites of glucosinolates compete with iodine for uptake into the thyroid gland. In a similar fashion, lima beans, sorghum, cassava and sweet potato contain cyanogenic glucosides. These break down into thiocyanates, which also compete with iodine for uptake into the gland. For example, it is important to adequately soaking/cook cassava in order to remove linamarin. Otherwise, the hydrolysis of linamarin in the gut releases cyanide, which in turn is metabolized into thiocynate [2]. Smoking mothers have been found to have lower iodine content in breast milk. Higher serum levels of thiocyanate, caused by smoking, are thought to inhibit the sodium-iodide symporter in the lactating mammary gland [5].

Flavonoids, substances found in foods like soy and millet, impair thyroperoxidase activity. For example, using soy-based formula without iodine supplementation can result in hypothyroidism in infants [2]. However, a review by Messina showed negligible changes in the thyroid function of healthy individuals, after the consumption of soy foods/is flavones [6]. A double-blind randomized crossover study conducted by Teas et al further demonstrated that soy protein isolate supplementation was not associated with a biochemical change

Table 1: Recommended Dietary Allowances (RDAs) for Iodine [4].

Age	Male	Female	Pregnancy	Lactation
Birth to 6 months	110 mcg*	110 mcg*		
7–12 months	130 mcg*	130 mcg*		
1–3 years	90 mcg	90 mcg		
4–8 years	90 mcg	90 mcg		
9–13 years	120 mcg	120 mcg		
14–18 years	150 mcg	150 mcg	220 mcg	290 mcg
19+ years	150 mcg	150 mcg	220 mcg	290 mcg
*Formula and food should be the only sources of iodine for infants				

Citation: Bhargava A. A Healthy Diet, a Healthy Thyroid. J Endocr Disord. 2014;1(3): 1011.

in thyroid function [7]. Furthermore, there is no strong evidence that patients on levothyroxine need to avoid soy products. According to some experts, it is best to wait 4 hours after taking levothyroxine to consume soy [8].

Lastly, deficiencies of certain trace elements and vitamins minerals can worsen the effects of iodine deficiency. Deiodinases and enzymes like glutathione peroxidase are dependent on selenium for their function. Consequently, selenium deficiency can impair thyroid hormone metabolism. Thyroperoxidase activity is heme dependent. Therefore, iron deficiency can disrupt the production of thyroid hormone. Pregnant women with iron deficiency anemia have been shown to have a higher TSH and lower T4 level. Vitamin A mediates suppression of the pituitary TSHβ gene. Hence, vitamin A deficiency can result in increased TSH stimulation and possibly potentiate goiter formation [2]. Deficiencies of zinc and copper have also been associated with hypothyroidism [9]. Zinc is thought to be required by the T3 receptor to undergo a biologically active conformational change. Thus, Zinc deficiency may impair the action of T3 [10].

In conclusion, given the current prevalence of hypothyroidism in the population and a likely increase in screening in the medical community, more and more patients with subclinical or overt hypothyroidism are likely to present to their respective physicians. Clinicians must remain abreast of various foods that can affect thyroid function, as well as the nutritional supplements that a patient might consider taking. In a diet replete with iodine or in an individual on levothyroxine supplementation, the intake of potential goiterogens should not cause a problem. Care must be taken to ensure that patients take levothyroxine in an appropriate fashion, and do not over supplement on elements such as iodine. This approach will help tailor therapy and provide safe, holistic and patient-centric care.

References

1. Garber JR, Cobin RH, Gharib H, Hennessey JV, Klein I, Mechanick JI, et al. Clinical practice guidelines for hypothyroidism in adults: cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. Endocr Pract. 2012; 18: 988-1028.
2. Zimmermann MB. Iodine deficiency. Endocr Rev. 2009; 30: 376-408.
3. Teas J, Pino S, Critchley A, Braverman LE. Variability of iodine content in common commercially available edible seaweeds. Thyroid. 2004; 14: 836-41.
4. Institute of Medicine (US) Panel on Micronutrients. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Washington (DC): National Academies Press (US). 2001.
5. Laurberg P, Nøhr SB, Pedersen KM, Fuglsang E. Iodine nutrition in breast-fed infants is impaired by maternal smoking. J Clin Endocrinol Metab. 2004; 89:181-187.
6. Messina M, Redmond G. Effects of soy protein and soybean isoflavones on thyroid function in healthy adults and hypothyroid patients: a review of the relevant literature. Thyroid. 2006;16: 249-258.
7. Teas J, Braverman LE, Kurzer MS, Pino S, Hurley TG, Hebert JR. Seaweed and soy: companion foods in Asian cuisine and their effects on thyroid function in American women. J Med Food. 2007;10: 90-100.
8. Nippoldt TB. Is it true that people who have hypothyroidism should avoid soy? *Mayo Foundation for Medical Education and Research*. 2014.
9. Betsy A, Binitha M, Sarita S. Zinc deficiency associated with hypothyroidism: an overlooked cause of severe alopecia. Int J Trichology. 2013; 5: 40-42.
10. Freake HC, Govoni KE, Guda K, Huang C, Zinn SA. Actions and interactions of thyroid hormone and zinc status in growing rats. J Nutr. 2001; 131: 1135-1141.

J Endocr Disord - Volume 1 Issue 3 - 2014
ISSN : 2376-0133 | www.austinpublishinggroup.com

Citation: Bhargava A. A Healthy Diet, a Healthy Thyroid. J Endocr Disord. 2014;1(3): 1011.