An Overlooked Contributor to Obesity?

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The World Health Organization estimates that approximately 2 billion people worldwide are deficient in iodine.

Thyroid hormones are known to affect obesity and lipid disorders, and one often-overlooked component in this equation is iodine deficiency. Obesity and metabolic syndrome are increasing dramatically worldwide, contributing to cardiovascular morbidity and mortality, as well as other obesity-related endocrinopathies.

Dietary iodine is recognized as an essential nutrient, as it is necessary for the synthesis of the thyroid hormones thyroxin (T4) and triiodothyronine (T3), both of which are required for normal thyroid function. The dietary reference intake for dietary iodine, as dictated by the World Health Organization, is 90)g per day for children 5 years of age or younger, with an increase corresponding to age: 120)g/day for children 6 to 12 years, 150)g/day for adults, and 200)g/day for pregnant or lactating women.¹

Worldwide Deficiency

The World Health Organization estimates that approximately 2 billion people worldwide are deficient in iodine. Included in this figure are an estimated 285 million school-age children with iodine deficiency. In the United States, iodine status is considered marginal at best, within the range of 100 to 199 milligrams per liter. However, in some parts of the world, iodine deficiency remains a significant health problem.^{2,3} Added to insufficient intake is the recurrent exposure to the halogens chlorine, bromine, and fluoride, which also contribute to deficiency.

Of the dietary iodine intake, approximately 80% is sequestered by the thyroid gland, which uses it for the synthesis of thyroxine, via the sequential addition of four iodine molecules to the amino acid tyrosine.⁴ In addition to the thyroid, other bodily tissues also concentrate iodine, including the salivary glands, the gastri mucosa, choroids plexus, the lactating mammary gland, and the ovaries. Consequentially, as a result of decreased iodine status or deficiency, thyroxine cannot be made, which may ultimately result in thyroid dysfunction and subsequent weight gain.

Since thyroid hormones affect many bodily functions, including muscle, heart, liver, kidney, and the developing brain, optimal status is crucial for favorable health in all age groups.⁵ In one study with morbidly obese patients, a high degree of subclinical hypothyroidism (19.5%) was observed, with subjects having elevated levels of T3, T4, FT3, and TSH.⁶ There is also evidence of a significant correlation between the metabolic syndrome, of which symptoms include abdominal obesity and insulin resistance, and thyroid dysfunction, both of which contribute to cardiovascular disease.⁷

Iodine deficiency manifests as a variety of illnesses, collectively termed iodine deficiency disorders (IDD), which results when iodide intake is less than 20)g per day. With iodine deficiency, an increase in serum TSH, and a significant decrease in both serum T4 and free T4, have been noted. These observations have been correlated with marked thyroid histological alterations, including "cylindric epithelial cells, diminution or absence of colloid and dilatation of blood capillaries."⁸ In moderate iodine deficiency, the thyroid gland, under the influence of thyroid-stimulating hormone, hypertrophies to concentrate iodide in itself, resulting in a colloid goiter. Most of these cases remain euthyroid.

Iodine and the Thyroid

The pattern between thyroid diseases and iodine intake is distinctly correlated, despite the absence of marked disease patterns, including cretinism and endemic goiter.⁹ Depressed iodine status has been correlated with elevated serum TSH, and it is an indicator of an insufficient T3 receptor saturation. Additionally, iodine deficiency affects brain development, which is particularly important in young children and adolescents. Given the increased obesity level in children, iodine status is a crucial consideration. An elevated serum TSH is an indicator of a potential risk. An elevated serum TSH, along with a normal serum T4 and T3, is an indication of subclinical hypothyroidism, while overt hypothyroidism is associated with an elevated TSH and a low T4, along with variable levels of T3.¹⁰

A 64% reduction in plasma thyroxine has been confirmed with iodine deficiency.¹¹ Despite the consumption of iodinated salt, iodine deficiency disorders continue to exist, and goiters are still endemic in many populations.¹⁰ Sari et al have evidenced a "positive correlation between thyroid volume and body weight, body mass index, body fat percentage, and weight circumference," along with a positive correlation between TSH and body weight and body fat.¹²

In children and adolescents, goiter/hypothyroidism, impaired mental function, retarded physical development, and an increased susceptibility of the thyroid to nuclear radiation are correlated with a dysfunctional thyroid. Considering the marked increase in childhood obesity, a nutritional deficiency in iodine should be addressed. Implications of thyroiddysfunction in adults include goiter/hypothyroidism, impaired mental function, spontaneous hyperthyroidism in the elderly, iodineinduced hyperthyroidism, and increased susceptibility of the thyroid to nuclear radiation. Consequently, the spectrum of IDD includes serious complications. Of these implications, brain damage and irreversible mental retardation in children are considered the most significant disorders induced by iodine deficiency.¹³

Supplements Available

Although iodine deficiency has been addressed via the widespread application of public iodine supplementation programs, including the iodization of salt, in many regions of the world iodine deficiency remains a public health concern. Consequently, supplemental iodine represents an optimal approach to deficiency. Various forms of supplemental iodine are currently available including liquid and tablet forms—both of which consist of potassium iodine or iodine derived from food sources such as kelp.

The earliest form of potassium supplementation was that of Lugol's solution, first made in 1829 by the French physician JGA Lugol. Lugol's solution is composed of 5% iodine along with 10% potassium iodide, with a final concentration of 130 mg/mL iodine.¹⁴ Iodine derived fromkelp offers a natural alternative to supplementation. However, concerns with kelp-derived iodine should be considered, specifically with the knowledge that they are typically heavily contaminated with heavy metals, including arsenic, cadmium, and mercury.

It has been noted that "more attention should be devoted to heavy metal levels in kelp, particularly where they may play a role in a subsistence diet."¹⁵ In some species of kelp, as much as 65.5 parts per million arsenic has been extracted.¹⁶ The FDA tolerance level on arsenic is 2 ppm in kelpcontaining products.¹⁷ Additionally, as a result of herbal kelp supplementation, arsenic toxicity has been reported, which was alleviated once supplementation was ceased.¹⁸ A preferred form of supplementation is the use of liquid potassium iodine. This form of supplementation has shown much promise, as it is well absorbed and offers a broader dosing range, as dosage adjustments are easily implemented to corresponding iodine status. Furthermore, considering the fact it has little taste and odor, liquid potassium iodine can readily be administered to all population ranges, including infants, children, and adults. More importantly, no risk of heavy metal contamination exists, as it is not derivated from souces with known contaminants, such as sea vegetation.

Determining iodine status in overweight patients offers a valid and theraputically beneficial approach toward deficiency. It has been noted that the benefits of iodine therapy far outweigh the risks induced by excess iodine.^{19,20,21} Continual monitoring along with the use of an easily regulated source of iodine offers a definitive approach toward the ultimate goal of adequate iodine status. Additionally, correcting the iodine deficiency plays an important role in optimizing thyroid function, and in turn may be an important factor in weight reduction.

References

^{1.} WHO global database on iodine deficiency. Geneva: World Health Organization. Available at: www.who.int/gb/ebwha/pdf_files/WHA58-REC1/english/WHA58_24en.pdf. Accessed June 27, 2007.

^{2.} Utiger RD. Iodine Nutrition – More is Better. N Eng J Med. 2006;354(26):2819-2821.

3. Boyages SC. Clinical Review 49: Iodine Deficiency Disorders. J Clin Endocrinol Metab. 1993;77(3):587-591.

 Berdanier CD. Advance Nutrition Micronutrients. Boca Raton, Fla; CRC Press; 1998.
Venkatesh Mannar MG, Dunn JT. Salt iodization for the elimination of Iodine Deficiency. The Netherlands,

International Council for Control of Iodine Deficiency Disorders, 1995. Available at:

www.who.int/nutrition/publications/. Accessed June 27, 2007.6. Michalaki MA, Vagenakis AG, Leonardou AS, et al. Thyroid function in humans with morbid obesity. Thyroid.

2006;16(1):73-78.

7. Roos A, Bakker SJ, Links TP, Gans RO, Wolfffenbuttel BH. Thyroid function is associated with components of the

metabolic syndrome in euthyroid subjects. J Clin Endocrinol Metab. 2007;92(2):491-496. 8. Ruz M, Codoceo J, Galgani J, et al. Single and multiple selenium-zinc-iodine deficiencies affect rat thyroid

metabolism at ultrastructure. J Nutr. 1999;129(1):174-180.

9. Laurberg P. Iodine intake – What are we aiming at? J Clin Endocrinol Metab. 1994;79(1):17-19.

10. Verma M, Raghuvanshi RS. Dietary iodine intake and prevalence of iodine deficiency disorders in adults. J Nutr

Environ Med. 2001;11(3):175-180.

11. Moreno-Reyes R, Egrise D, Boelaert M, Goldman S, Meuris S. Iodine deficiency mitigates growth retardation and

osteopenia in selenium-deficient rats. J Nutr. 2006;136(3):595-600.

12. Sari R, Balci MK, Altunbas H, Karayalcin U. The effect of body weight and weight loss on thyroid volume and

function in obese women. Clin Endocrinol. 2003;59(2):258-262.

13. Mastorakos G, Nezi M, Papadopoulos C. Chapter 20. The Iodine Deficiency Disorders. Available at:

www.thyroidmanager.org/Chapter20/20-frame.htm. Accessed June 26, 2007.

14. Lugol's Iodine. Available at: en.wikipedia.org/wiki/Lugol's_iodine. Accessed June 27, 2007.

15. Burger J, Gochfeld M, Jeitner C, et al. Kelp as a bioindicator: Does it matter which part of 5 M long plant is used for

metal analysis? Environ Monit Assess. 2007;128(1-3):311-321.

16. Van Hulle M, Zhang C, Zhang X, Cornelis R. Arsenic speciation in Chinese

seaweeds using HPLC-ICP-MS and HPLCES-

MS. Analyst. 2002;127(5):634-640.

17. Agency for Toxic Substances and Disease Registry 2006. Available at: www.atsdr.cdc.gov. Accessed June 26, 2007.

18. Amster E, Tiwary A, Schenker MB. Case report: Potential arsenic toxicosis

secondary to herbal kelp supplement.

Environ Health Perspect. 2007;115(4):606-608.

19. Delange F, Lecomte P. Iodine supplementation: benefits outweigh risks. Drug Saf. 2000;22(2):89-95.

20. Delange F. Risks and benefits of iodine supplementation. Lancet.

1998;351(9107):923-924.

21. Braverman LE. Adequate iodine intake-the good far outweighs the bad. Eur. J. Endocrinol. 1998;139(1):14-15.

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