Iodine, A Need for Optimization

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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 discovery of Iodine by Bernard Courtois occurred early in the nineteenth century,[1] but it was not until the early 1850's that Chatin made the correlation between iodine and goiter,[2] although, a prior correlation by the Swiss physician J.F. Coindent had been documented. He associated the successful use of burnt sponge and seaweed for the treatment of goiter due to iodine deficiency.[3] Between 1820 and 1840 a tincture of iodine, iodoform, was routinely used and was considered a "new and potent remedy." [4] The Dietary Reference Intake (DRI) for iodine, as dictated by the World Health Organization (WHO), is 90 mcg/day for children 5 years of age or younger, with an increase corresponding to age; 120 mcg/day for children 6-12 years, 150 mcg/day for adults, and 200 mcg/day for pregnant or lactating women.[5] Currently, WHO estimates that approximately 2 billion people worldwide are deficient in iodine. Included in this figure are school-age children with iodine deficiency, estimated at 285 million children. In the United States iodine status is considered marginal at best, within the range of 100-199 ug/L. However in some parts of the world iodine deficiency remains a significant health problem. [6, 7]

Once consumed, dietary iodine is easily absorbed and is subsequently converted to iodide ion (I-). Of the dietary intake approximately 80% is sequestered by the thyroid gland, which utilizes it for the synthesis of thyroxine. Thyroxine is synthesized *in vivo* by the sequential addition of four iodine molecules to the amino acid tyrosine.[8] The sequential addition of iodine molecules to tyrosine produces monoiodothyronine, diiodothyronine, triiodothyronine, and thyroxine, respectively. In addition to the thyroid, other bodily tissues also concentrate iodine. These tissues include the salivary glands, the gastric mucosa, the choroids plexus, the mammary glands and the ovaries. Consequentially, as a result of iodine deficiency, or with decreased status, thyroxine cannot be made, which may ultimately result in thyroid dysfunction. 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.[9]

Iodine deficiency manifests as a variety of illnesses, which have collectively been termed Iodine Deficiency Disorders (IDD). Iodine deficiency results when iodide intake is < 20 µg/day. With iodine deficiency, an increase in serum TSH, and a significant decrease in both serum T4 and free T4 has 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".[10] 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. The pattern between thyroid diseases and iodine intake are distinctly correlated, despite the absence of marked disease patterns, including cretinism and endemic goiter.[11] Depressed iodine status has been correlated with elevated serum TSH, and it is an indicator of an insufficient T3 receptor saturation. In children an elevated serum TSH is an indicator of a potential risk of iodine deficiency, which consequently effects brain development. 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.[12] A 64% reduction in plasma thyroxine has been confirmed with iodine deficiency.[13] Despite the consumption of iodinated salt, iodine deficiency disorders continue to exist, and goiters are still endemic in many populations.[12]

T4, along with other thyroid hormones, is in part responsible for the regulation of growth and metabolism. Accordingly, sufficient iodine status is extremely important in the developing fetus, young children and adolescents. Iodine deficiency is said to have the greatest impact on cognitive and neurological function, and this effect is most significant during gestation and early infancy.[14] Subsequently, sufficient iodine status is particularly vital in pregnant women, since iodine is critical to early brain development, as well as in young children and adolescents, due to the consequences of mental retardation and irreversible damage to the brain and central nervous system.[15] Correction of any deficiency is specifically pertinent early in pregnancy in order to avoid the neurological manifestations of deficiency. In the developing fetus, as a result of a suboptimal level of iodine in the mother, there is a risk of impaired thyroid hormone synthesis, inevitably ensuing in mental retardation due to insufficiency. Even mild or moderate degrees of iodine deficiency have the potential to affect neurodevelopment, resulting in "neurological cretinism and/or decreased mental capacity,"[16] thus necessitating the need for intervention. The consequences of iodine deficiency in the developing brain and central nervous system are irreversible, the most serious form resulting in cretinism.[17] In a randomized study of pregnant women at early gestation (nine weeks) thyroid testing was performed to detect the presence of thyroid dysfunction. Of the women with elevated TSH levels (40), twelve were considered low-risk, thus concluding that testing of only those in a high-risk category would neglect approximately one-third of pregnant women with overt or subclinical hypothyroidism.[18] Observed implications of deficiency in pregnancy include abortions or stillbirths, congenital anomalies, increased perinatal mortality and endemic cretinism in the fetus, thus implicating the significance of intervention. In neonates the ramifications include neonatal goiter/hypothyroidism, endemic mental retardation, and increased susceptibility of the thyroid to nuclear radiation, while in both children and adolescents goiter/hypothyroidism, impaired mental function, retarded physical development and an increased susceptibility of the thyroid to nuclear radiation are observed. Implications in adults include goiter/hypothyroidism, impaired mental function, spontaneous hyperthyroidism

in the elderly, iodine-induced hyperthyroidism and increased susceptibility of the thyroid to nuclear radiation. In addition to adults, the spectrum of IDD includes serious complications in the fetus, and neonate. Of these implications brain damage and irreversible mental retardation in children are considered the most significant disorders induced by iodine deficiency.[19]

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. Supplemental iodine, however, represents an optimal approach to deficiency. There are currently various forms of supplemental iodine available, including liquid and tablet forms, both of which consist of either 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 J.G.A. Lugol. Lugol's solution is composed of 5% iodine along with 10% potassium iodide (KI), with a final concentration of 130mg/mL iodine.[20] Iodine derived from kelp 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."[21] The FDA limit on arsenic is two parts per million (2ppm) in kelp containing products. In one assessment, eight of nine samples of kelp evaluated showed levels of arsenic above the FDA limit, with reports as high as 8.5mg/kg (ppm) of arsenic.[22] A correlation between herbal kelp supplementation and arsenic toxicity has been reported, which was alleviated once supplementation was ceased. A preferred form of supplementation is the use of a liquid form of potassium iodine. This form of supplementation has shown much promise, as it is both 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, it can readily be administered to all population ranges, including infants, children and adults. More importantly, there is no risk of heavy metal contamination, as it is not derivated from souces with known contaminants, such as sea vegetation.

Iodine supplementation offers a valid and theraputically beneficial advantage towards deficiency. It has been suggesed that the benefits of iodine therapy far outway the risks induced by excess iodine.[24, 25, 26] Continued monitoring and the use of an easily regulated source of iodine is a definitive confirmation of adequate iodine status.

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