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Opinion Article

Effect of Iodine Deficiency in Human Body

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DESCRIPTION

Iodine insufficiency is a major public health issue around the world, especially among young children and pregnant women. Mental retardation is the most devastating symptom of iodine deficiency. It is one of the leading causes of preventable cognitive impairment in the globe, posing a threat to countries social and economic progress. This is the driving force behind the present global campaign to eradicate iodine deficiency.

Iodine insufficiency is caused by a shortage of iodine in the diet, which is common in areas where the soil lacks iodine due to prior glaciation and is sometimes exacerbated by precipitation or flooding. These deplete iodine in the soil, causing iodine deficiency in plants and livestock.

Natural goitrogens in staple foods like cassava, which contain thiocyanate, which slows thyroid iodide transport, might increase iodine deficit. Iodine insufficiency is exacerbated by deficiencies in selenium, iron and vitamin A. The deoidinases and glutathione peroxidase are selenium dependent enzymes. Accumulated peroxides in selenium shortage can harm the thyroid and deiodinase deficiency inhibits thyroid hormone synthesis. The aetiology of myxedematous cretinism has been linked to these outcomes.

Iron deficiency inhibits thyroid hormone synthesis by reducing heme dependent thyroperoxidase activity. Iron deficiency anaemia reduces the efficacy of iodine prophylaxis in goitrous children, whereas iron supplementation improves the efficacy of iodized oil and iodized salt. Vitamin A deficiency in iodine-deficient children raises TSH levels and increases the incidence of goitre, most likely due to reduced vitamin A mediated regulation of the pituitary TSH gene.

Iodine insufficiency causes a range of abnormalities known as Iodine Deficiency Disorders (IDD), which are caused by thyroid dysfunction. The most evident signs are goitre and cretinism, others include hypothyroidism, decreased fertility, increased perinatal death and newborn mortality. Iodine deficiency has the most significant effect on neurocognitive development.

Hypothyroidism caused by a lack of iodine during the most susceptible phase of brain development, prenatal life and the first year, can result in irreversible damage to brain structure and function. Cretinism affects 5% to 15% of the population in places with significant iodine deficiency. Individuals who live in places where iodine shortage is minor or moderate have milder neurological and intellectual abnormalities. According to a meta-analysis, severe iodine shortage causes an average IQ loss of 13.5 points in affected groups. Iodine deficiency is corrected in a population, which minimizes or eliminates all of its repercussions.

When an effective IDD control programme in a previously iodine deficient population expires, IDD recurrence is predictable. The median Urine Iodine (UI) concentration and total goitre prevalence are recommended markers for determining the severity and extent of iodine deficiency in a community. Because it responds faster to iodine deficiency correction and is sensitive to current iodine intake, the median UI is recommended for monitoring measures for IDD control rather than goitre prevalence. IDD is a public health issue, according to WHO, in populations with a median UI of 100 g/L or in places where more than 5% of children aged 6 years to 12 years have goitre.

Pregnancy, nursing and infancy should be the emphasis of IDD prevention strategies. However, determining iodine status during these phases of development is difficult and monitoring these groups has traditionally been neglected. For pregnancy, nursing or infancy, there are no recognized reference criteria for median UI. Although a median UI of 100 g/L to 199 g/L in School Age Children (SAC) and adults indicates adequate iodine intake, this criterion has not been validated in pregnant and lactating women or infants and may underestimate the true burden of IDD if applied to these target groups. RNI for iodine during pregnancy and lactation from 200 g/day to 250 g/day, a daily intake of more than this is not necessary and should ideally not exceed 500 g per day. The RNI for infancy has remained same at 90 grams per day.

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The International Council for Control of Iodine Deficiency Disorders (ICCIDD), the United Nations Children's Fund (UNICEF), the World Health Organization (WHO) and the World Food Program (WFP) have all contributed to the majority of the effort in producing and marketing salt iodization, implementing policies, educating people, leaders and monitoring.