



Maternal Iodine Supplementation: Clinical Trials and Assessment of Outcomes

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PRESENTATION ABSTRACT

Maternal and Child Iodine Status in Regions of Severe Deficiency

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Abstract

Iodine deficiency has multiple effects on human health due to inadequate thyroid hormone production. Some of the deleterious effects, i.e., increase of thyroid volume or thyroid nodules, are secondary to the feedback mechanisms triggered by the thyroid to prevent hormonal failure. The severity of iodine deficiency in the population is assessed by the median urinary iodine concentration (UIC) in school-aged children (SAC). When the median UIC is below 20 µg/L in SAC the population is considered severely iodine deficient. Under such conditions the population is exposed to a high risk of goiter and impaired cognitive function. In very severe deficiency endemic cretinism develops, the most dramatic consequence of iodine deficiency. The brain damage induced by severe iodine deficiency is due to maternal and fetal hypothyroidism affecting the thyroid-dependent brain development process. In addition to the overt manifestations of severe iodine deficiency, apparently healthy children and pregnant women living in severely iodine deficient regions suffer from endemic hypothyroidism.

The health consequences of severe iodine deficiency are so dramatic that the regions where the condition was present received special attention and urgent measures to control the situation in the past decades. Consequently, severely iodine deficient regions are uncommon at present and the public health burden of iodine deficiency has shifted toward mild iodine deficiency (MID) (median UIC 50–99 µg/L). Nevertheless, because iodine deficiency is a nutritional problem that can be controlled but not eradicated from a specific region it is worthwhile to remember that such severe deficiency did exist. There will always remain a risk of resurgence if prevention measures and monitoring systems are interrupted. We will discuss some of the pathophysiological and clinical features of severe iodine deficiency.

The thyroid triggers several homeostatic mechanisms to cope with low iodine intake. It is only when such mechanisms are overcome that the production of thyroid hormones and particularly thyroxine (T₄) will be impaired. The adaptive processes include stimulation of the iodide trapping mechanism and preferential production of triiodothyronine (T₃). A compensatory mechanism increasing T₄ to T₃ conversion can take place in conditions of iodine deficiency. Tissues highly dependent on T₄ for their intracellular content of T₃, such as the brain, undergo a significant increase in T₄ to T₃ conversion. This preferential protection of the brain is related to the marked increase of the activity of deiodinase D₂ in response to iodine deficiency.

Iodine deficiency may be aggravated if goitrogens are present in the staple food of the deficient regions. Goitrogens impair thyroid function by decreasing iodide thyroid uptake, as in the case of thiocyanate associated with cassava consumption. Other goitrogens inhibit the activity of thyroid peroxidase (TPO), as in the case of flavonoids present in millet. Millet and cassava are a staple food in many iodine-deficient regions of Africa. The public health impact of the interaction between iodine deficiency and goitrogens is strong because severely iodine deficient regions are usually very isolated regions with little food diversity, such as the famine and war-affected Darfur region in Sudan. In these regions the population typically consumes only locally produced food items. In addition to goitrogens, micronutrient deficiencies, selenium, iron, and



vitamin A deficiencies may interact with iodine deficiency and affect the response of iodine-deficient subjects to iodine supplements.

In conclusion, severe iodine deficiency has profound effects on human health, particularly by reducing the cognitive capacity of affected populations. Enormous progress had been made in recent years to mitigate the impact of the problem and currently severe iodine deficiency is uncommon. However, efforts to optimize iodine intake need to be maintained strictly because a re-emergence of iodine deficiency is a permanent risk.

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