

IODINE DEFICIENCY

- AN IMPORTANT NUTRITIONAL PROBLEM IN SRI LANKA

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Iodine is one of the important micronutrients in the human, small amounts being required to be provided in the diet as this element cannot be manufactured in the human body. Effects of iodine deficiency and dietary treatment of iodine deficiency has been known since early times, making it the earliest nutritional disease to be recognised.

Iodine metabolism and consequences of deficiency

Iodine required by the human body has to be provided in the diet. About half the iodine in the diet absorbed through the small intestine as inorganic iodide is taken up by the thyroid gland and incorporated into protein (thyroglobulin) to form precursors of the two related types of hormone - triiodothyronine (T3) and tetraiodothyronine (T4). These hormones are released into the blood by the thyroid gland in re-

sponse to various stimuli controlled by the thyroid stimulating hormone (TSH). The syntheses and availability of the thyroid hormones are reduced in iodine deficiency, hence blood levels of T3 and T4 and of TSH are used for assessment of status of function of the thyroid gland. Effects of deficiency of thyroid hormones

have been well described even though mechanisms of action of the hormones remain to be fully worked out. One result of deficient hormone synthesis is the enlargement of the thyroid gland itself, hence the presence of an enlarged thyroid-known as goitre, is the main clinical sign of iodine deficiency.

Increased foetal deaths, stillbirths, increased deaths during infancy and childhood, developmental abnormalities have been found to be associated with iodine deficiency. Deaf mutism has been suggested as an indicator of the severity of disorders due to iodine deficiency.

Thyroid hormones have extensive effects throughout the body. They influence the metabolic rate, protein synthesis, enzyme function, cellular transport and other physiological processes. They have specific effects on growth of children - deficiency causing retarded growth. Throughout life, a normal level of thyroid hormones is needed for active intellectual function; hypothyroidism is associated with apathy. This effect is probably reversible at least in the adult in that when normal thyroid function is restored, mental functions return



to normal. Low thyroid activity at important stages of development causes irreversible brain damage, at its extreme expressed as cretinism and deaf mutism. This may result from vulnerability of the foetus to maternal hypothyroidism, soon after conception. Lesser degrees of brain damage manifest as deafness, mental retardation and lowered intelligent quotient.

In countries where the prevalence of goitre among school children range from 5 - 20%, an effective control programme could be implemented by providing iodized salt with an iodine concentration of 10-25mg/Kg. (or ppm) to the entire population.

Goitre i.e. enlargement of the thyroid gland is considered to be endemic when its prevalence rate exceeds 10% in a given region. The iodine intakes in such areas

are generally below 50 mcg/day compared to the recommended intake of 150 - 300 mcg/day. When goitre prevalences are higher than 30%, 5 -10% of the population can have severe and irreversible mental retardation associated with abnormalities of physical development, known as endemic cretinism.

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In recent times, much attention has been paid to the damage due to lesser degrees of iodine deficiency. These disorders not only affect the individual, but entire communities. All residents of a community affected by endemic goitre are exposed to sub-optimal levels of thyroid hormones during their development, hence may have serious but subtle effects on the quality of the society at large. It has been reported that motivation, spontaneity, creativity and

intelligence may be diminished. Thus, iodine deficiency may lead to a spectrum of subclinical effects which place the children born to a mother on an iodine deficient diet at a developmental disadvantage which undermines their mental and physical growth, thus slowing the progress of the individual and the community.

Causes of iodine deficiency

The commonest cause of iodine deficiency is the inadequate iodine intake due to environmental deficiency of this essential element. This occurs where iodine is leached out and washed away from the soil by heavy rains, specially in the mountainous areas. Substances referred to as goitrogens which interfere with iodine metabolism are present in some of the foods commonly consumed ex. linamarin which is present in cassava. Bacterial contaminants of water, industrial pollutants also have several substances which have goitrogenic and/or anti thyroid effects.

Other forms of malnutrition, specially protein energy malnutrition and vitamin A deficiency

Figure 1: Distribution of Iodine Deficiency Disorders in Developing Countries



Source: ACC/SCN, 1987.

may have secondary effects on iodine nutritional status. Iodine availability to the thyroid gland can be altered due to dietary or other factors socially when iodine intake is marginal and dietary patterns are less varied.

Extent of the problem

Surveys of global occurrence of iodine deficiency estimate that approximately 200 million in developing countries may have this deficiency and another 800 million are "at risk". Areas of iodine deficiency disorders in developing countries are shown in Figure 1.

In community surveys, prevalence of goitre is taken to be an indicator of the problem of iodine

deficiency, even though such data may underestimate the full extent of iodine deficiency disorders. The problem of iodine deficiency was first reported in Sri

Lanka as early as 1947. The most recent prevalence study carried out in 1987 has shown that iodine deficiency, continues to be an important nutritional deficiency,

Table 1: Prevalence of Goitre among School Children Sri Lanka, 1987 (excluding the districts the districts of the north and east)

Prevalence category	Prevalence rate	Districts
Low	less than 10%	Colombo, Anuradhapura Polonnaruwa, Matale
Moderate	10% - 20%	Galle, Matara, Hambantota Puttalam, Kurunegala, Gampaha
High	more than 20%	Kandy, Kegalle, Badulla, Nuwara eliya, Ratnapura, Kalutara, Moneragala

Source: Fernando M.A: and Balasuriya S. (1989). *Asia Pacific Journal of Public Health* Vol. 4 Pg. 6.

affecting some areas of the country. The prevalence of goitre among school children was studied in all districts of Sri Lanka, excluding those of the north and east. The results are summarised in Table 1.

Prevention and Control of iodine deficiency disorders

Iodine deficiency is being considered an impediment to human development, hence the importance of prevention and control of this disorder. Though a major nutritional problem influencing almost all age groups, this deficiency can be successfully and inexpensively prevented and controlled. The most cost effective method has been to increase the intake of iodine in populations by fortification of salt. Another approach that has been used specially in countries where the problem is more severe, is the periodic administration of iodised oil.

In countries where the prevalence of goitre among school children range from 5 - 20%, an effective control programme could be implemented by providing iodized salt with an iodine concentration of 10-25 mg/Kg. (or ppm) to the entire population. The most popular and simplest technique of salt iodisation consists of applying a solution of an iodine compound on salt by a drip or a spray. If the salt is then dried well, is free of impurities and has a slightly alkaline pH, potassium iodide is well suited and will be quite stable.

For a salt fortification programme

to be effective, it is necessary that all salt available to the community is iodized. Problems may arise in the production and distribution of salt to the people who are iodine deficient, and in getting people to consume iodized salt. In Sri Lanka, a programme for iodization of salt has been in existence for some years. However, for several reasons related to production, distribution as well as due to problems in the pattern of consumption, it has not been possible to ensure that all salt that is used by the population at risk, is fortified with iodine. Experiences from countries that have successfully implemented such programmes may prove useful in making necessary improvements.

The main hazard of iodization of salt has been the toxic effects reported due to increased activity of the thyroid gland i.e. thyrotoxicosis. Such effects have been reported mainly in adults over the age of 40 years. This is caused by autonomous thyroid function resulting from long standing iodine deficiency. It can be minimized by limiting the use of iodized salt by those over 40 years of age, even though this poses problems with programme implementation.

Since the value of periodic administration of iodized oil by injection was first established in Papua New Guinea, this method has been used in several countries throughout the world. This method is appropriate for areas with high endemicity i.e. with goitre prevalence of more than 30%, specially in isolated village

communities in mountainous areas. In a suitable area, the iodized oil is administered to the vulnerable population. A repeat of the injection is required in 3-5 years. The major problems with this programme are: the cost of the injections and the need to have skilled workers to administer the injection periodically. It has been shown that administration of iodized oil by mouth may have an effect, for 1-2 years.

Other possible approaches for prevention of iodine deficiency include: modification of habitual diet consumed in endemic areas, iodine supplementation of foods and water for human consumption, iodine medications to people in endemic areas.

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