29. Iodine

Physiology [1]

Iodine is a constituent of thyroxine (T4) and tri-iodothyronine (T3). Adequate circulating levels of these thyroid hormones are necessary for optimum cellular metabolism, normal growth and development.

Both inorganic and organic iodine are absorbed efficiently by the small intestine. Extracellular fluid contains 10-15 µg iodide/L. The total size of the iodide pool is approximately 250-350 µg, but its precise mass varies with iodide intake, which, in the absence of specific dietary or exogenous supplementation, corresponds closely to the amount of element entering the local food chain from the immediate geochemical environment. Thus populations living on heavily leached soils, which are often, but not exclusively, mountainous areas are at particular risk of sub-optimal iodine intakes. Plasma inorganic iodide is loosely bound to protein. It is cleared principally by the thyroid and kidneys but other tissues such as the gastrointestinal mucosa, mammary and salivary glands, and ovaries can also concentrate the element.

Over 75 % of the 10-20 mg iodine present in the normal adult is found in the thyroid gland. Iodide is taken up into the thyroid actively by a sodium-dependent carrier-mediated pathway which is stimulated by thyroid stimulating hormone. The iodide is then rapidly oxidised by thyroperoxidase, and 'organified' by iodination of tyrosyl residues in thyroglobulin. Pairs of the resultant iodotyrosines link to form iodothyronines. The major excretory route of iodide is via the urine, and daily urinary excretion of the element is often used as a convenient index of intake. Goitrogenic cyanoglucosides in brassicas, cassava, maize, sweet potatoes, lima beans and bamboo shoots interfere with the organification of iodide by the thyroid [1,2].

Decreased circulatory levels of T3 lead to a loss of the inhibitory feed back on the release of hypothalamic thyrotrophin releasing hormone, the increased secretion of which increases the secretion of pituitary thyroid stimulating hormone, which stimulates the uptake of iodide by the gland and can lead to thyroid hyperplasia and goitre.

Deficiency and excess

Iodine Deficiency Diseases (IDD) may be widespread in Europe [3,4]. The spectrum of IDD is extensive and subtle, as are their impact on socio-economic development [5,6].

Maternal iodine deficiency causes infertility, increased incidence of abortions and stillbirths, increased perinatal and neonatal mortality, congenital abnormalities including neurological and myxoedematous cretinism, and, less obviously, degrees of psychomotor retardation evident in later childhood resulting from intrauterine iodine deficiency. In older children and adults IDD include impaired mental function, hypothyroidism with goitre and growth retardation.

High iodine intakes cause toxic nodular goitre and hyperthyroidism. Such toxicity is rare in normal populations or individuals with an intake of less than 5 mg/d but those with pre-existent iodine deficiency may be susceptible to developing toxic nodular goitre, hyperthyroidism, and thyroid cancer at intakes below this [7], and transient hyperthyroidism has been observed in previously deficient individuals on intakes of 150-200 µg/d [8]. The incidence of such complications diminishes with time as the overall iodine supply of the population is improved and the number of people previously exposed to iodine deficiency diminishes. Intakes of 1-2 mg/d appear to be safe [9] but intakes above 10 mg/d in the form of seaweed have been associated with an increased incidence of iodine goitre [10].

Requirements

Adults

Most adults can maintain iodine balance and normal thyroid function on intakes between 40 and 100 µg iodine/d. A plateau concentration of iodide in the thyroid gland is achieved at an iodine intake of 100 µg/d, and increasing intakes to 300 or 500 µg daily has no further effect on this or on reducing the incidence of goitre [11]. An Average Requirement of 100 µg is proposed and a PRI of 130 µg/d. (A daily intake of up to 200 µg is being advised by a current WHO initiative designed to eradicate iodine deficiency disorders). For the LTI 70 µg/d is proposed, as an intake below which thyroid adaptation may become inadequate with a risk of dysfunction or sub-optimal operation [6,7,12].

Children

PRIs for younger age groups have been calculated from adult values, on the basis of energy requirements.

Pregnancy and lactation

Provided the normal prepregnancy iodine intake is adequate, there is no evidence that an increased dietary intake is needed during pregnancy, so no increment is recommended.

Lactating women on a normal plane of iodine nutrition produce iodine in their milk in amounts more than sufficient for the infant. In order to replace these losses, a PRI of 160 μg/d is suggested during lactation. The increase is based on the needs of the infant rather than the amount actually produced in the milk.

Summary

Adults	µg/d
Average Requirement	100
Population Reference Intake	130
Lowest Threshold Intake	70

Population Reference Intakes of other groups

Age Group	PRI (µg/d)
6 - 11 m	50
1 - 3 y	70
4 - 6 y	90
7 -10 y	100
11 - 14 y	120
15 - 17 y	130
Pregnancy	130
Lactation	160

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