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*Editorial*

# Iodine Deficiency and Excess: Effect on Health

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## Importance of iodine

Iodine is an essential trace element which is required for the synthesis of thyroid hormones. Thyroid hormone is needed for normal growth and is particularly critical for neurodevelopment of fetus and infant.[1] In children the neurological and psychological defects are noticed if there is iodine deficiency (ID) during pregnancy and infancy.[2]

Iodine is distributed in the earth's environment in an uneven manner. Iodide salt is depleted in many regions due to earth erosion, flooding and other natural causes. For these reasons the iodide concentration is high in oceans. Slow and incomplete iodine cycling in many regions is leaving drinking water and soils in low iodine condition.

In healthy adults, >90% dietary iodine is absorbed.[3] From the circulation iodine is cleared mainly by the thyroid gland and kidney. Renal clearance of iodine is fairly constant but the thyroid clearance depends on iodine intake. The areas where iodine is sufficient, the adult thyroid traps ~60 mg of iodine/day to compensate the losses and to maintain thyroid hormone synthesis. Healthy adult body contains 10 to 20 mg of iodine, of which 70 to 80% is in the thyroid gland. Thyroid hormone is released into the circulation by the thyroid gland. Iodine is released after the degradation of thyroid hormone in the periphery, enters the plasma iodine pool and can be used up by the thyroid gland or excreted by the kidney. More than 90% of ingested iodine is excreted in urine.[3]

## Recommended iodine intake

UNICEF, International Council for the Control of Iodine Deficiency Disorders (ICCIDD) and WHO recommend that the daily intake of iodine should be

- 90 μg for preschool children (0 to 59 months);
- 120 μg for school children (6 to 12 years);
- 150 μg for adolescents (above 12 years) and adults;
- 250 μg for pregnant and lactating women.

Recommended single yearly dose of iodized oil supplement is 400 mg for women in any age group and 200 mg for school-going children.[4]

A number of studies found increased prevalence of thyroid disorders in both iodine deficiency and iodine excess.

## Iodine deficiency disorders

Iodine deficiency disorders (IDD) is the cardinal cause of mental retardation which is preventable.[5] In severely iodine deficient areas, the intelligence quotient (IQ) of children is 12 points lower than those of iodine sufficient area. The IQ improved after the iodine supplementation.[6] In adult, mild to moderate iodine deficiency causes increased cases of hyperthyroidism due to toxic goiter.[7] Iodine deficiency causes increased pregnancy loss, neonatal hypothyroidism, infant mortality, cretinism, endemic goiter, intellectual impairments and growth retardation.[8]

To prevent IDD the World Health Assembly in 1991 approved the goal to eliminate the iodine deficiency as a public health problem. Universal salt iodinization (USI) was recommended by WHO and UNICEF in 1993 as the main approach to achieve the goal.[2] In 2005, World Health Assembly adopted a resolution to report the global IDD situation every three years.[4]

### *Measures to compensate IDD*

The best approach to control iodine deficiency is to add iodine in salt. Depending on local salt intake WHO/UNICEF/ICCIDD recommendation is to add iodine at a level of 20–40 mg/kg salt.[9] Different forms of iodine can be added to salt like potassium iodide (KI) or potassium iodate ($KIO_3$). In the presence of salt impurities, humidity, and porous packaging $KIO_3$ has higher stability than KI.[10] In tropical countries and those with low-grade salt $KIO_3$ is the recommended form of iodine which is usually added in the dry salt. Two techniques are used to iodize the normal salt: (1) the wet method where at a

regular rate a solution of $KIO_3$ is dripped or sprayed on to salt passing by on a conveyor belt; (2) the dry method where powder of KI or $KIO_3$ is sprinkled over the dry salt. Ideally the packaging should be in low-density polyethylene bags. It was found in a multi-country study that storage of salt in high-density polyethylene bags resulted in up to 90% loss of iodine in one year because of combination of high humidity and porous packing, compared to 10–15% in low density polyethylene bags.[5]

In the iodized salt the actual amount of iodine at the consumer level can vary. It is because of the difference in the amount of iodine added within batches and individual bags and/or due to inadequate mixing of salt after the salt iodization. There is also loss of iodine due to salt impurities, packaging (for instance, 1 kg versus 20 or 50 kg), and environmental conditions during storage and distribution, in food processing, and washing and cooking processes in the household. Almost 30–80% is lost because of high humidity and porous packaging. WHO recommend that the iodine concentration should be 20–40 mg/kg salt to compensate 20% loss in packaging and handling and 20% from cooking.[4]

## Excess iodine intake

For thyroid hormone synthesis iodine is a major component but it is also a microenvironment for thyroid cells to grow. USI has been introduced in many countries over the last 25 years. It is a safe, cost-effective and sustainable method to eliminate iodine deficiency disorders (IDDs). For more than 10 years an increased iodine intake has brought new challenges in long-term in iodine sufficient regions covered by USI.[11] In a study in 2013 iodine excess was found in 10 countries; it was thought that the incidence of thyroid disorders might transiently increase after introduction of iodized salt in regions of chronic IDD, and programs should include monitoring for both iodine deficiency and excess.[8]

After exposure to high iodine levels due to the acute Wolff–Chaikoff effect, the synthesis of thyroid hormone is normally inhibited.[12] If it persists, within a few days the thyroid is able to ''escape'' from this effect.[8] The Jod–Basedow phenomenon, or iodine-induced hyperthyroidism (IIH) occurs in individuals with nontoxic diffuse or nodular goiter. The prevalence of IIH is due to autoimmunity. There is also incidence of iodine induced hypothyroidism because of autoimmune thyroiditis. The incidence of autoimmune thyroiditis increased in Poland, Greece and Sri Lanka after USI. In 1966 it was found in Japan to have iodine induced goiter. Zimmerman et al[5] also found that chronic excess iodine also increased the volume of thyroid gland in children. In China, the area with high iodine level in water, also has prevalence of goiter. In Sweden and Australia the incidence of papillary carcinoma was observed after the USI started.[11]

In Somalia iodine excess during pregnancy existed. Until approximately 36 weeks of gestation, the fetal thyroid does not acquire the capacity to suppress the acute Wolff–Chaikoff effect. Therefore, a maternal iodine excess could cause fetal congenital hypothyroidism.[13] Nohr found that iodine supplementation during pregnancy could cause 27.3% higher TSH of cord blood than that of those with no artificial iodine supplementation. The fetal thyroid is more sensitive to the inhibitory effect of iodine than it was thought.[14]

## Monitoring of UIS program

UIS program needs careful monitoring because both iodine deficiency and iodine excess have adverse effects on health. The two commonly used indicators are the household coverage of iodized salt and the median urinary iodine concentration (UIC) value for the sampled population. UIC can assess the iodine intake but cannot indicate the thyroid function. In a recent WHO report it was found that the thyroglobulin is a sensitive measure for both iodine excess and deficiency.[3]

## Recommended urine iodine content to assess iodine status

Less than 20 μg/L is severe iodine deficiency, 20–49 μg/L is moderate iodine deficiency, 50–99 μg/L is mild deficiency, 100–199 μg/L is adequate, 200–299 μg/L is more than adequate, >300 μg/L is excessive which has risk of iodine induced health problem. On the other hand, for pregnant women <150 μg/L is insufficient, 150–249 μg/L is adequate, 250–499 μg/L is more than adequate and ⩾500 μg/L is excessive.[4] In children and non-pregnant women,

median urinary iodine concentrations of between 100 µg/L and 299 µg/L define a population which has no iodine deficiency. In addition, not more than 20% of samples should be below 50 µg/L. In non-pregnant, non-lactating women, a urinary iodine concentration of 100 µg/L corresponds roughly to a daily iodine intake of about 150 µg under steady-state conditions.

## Conclusion

Even though iodine supplementation is needed to prevent and treat IDDs, a safe level of iodine intake must be maintained. The excessive iodine exposure cases are not clinically fatal but could be harmful. Hypothyroidism, hyperthyroidism and autoimmune thyroiditis occur in cases with more than adequate or excessive iodine levels, which are unsafe especially for susceptible populations with recurring thyroid disease, the elderly, fetuses, and neonates.

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