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IODINE AND HEALTH

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A deficiency of iodine, which is among the body's essential trace elements, is both easy and inexpensive to prevent. Iodine deficiency nevertheless continues to be a significant public health problem in many countries. Iodine deficiency not only causes goitre; it may also result in irreversible brain damage in the fetus and infant, and retarded psychomotor development in the child. Iodine deficiency is the most common cause of *preventable* mental retardation. It also affects reproductive functions and impedes children's learning ability. The cumulative consequences in iodine-deficient populations spell diminished performance for the entire economy of affected nations.

Iodine deficiency disorders (IDD) are currently a significant public health problem in 118 countries (1). An estimated 1571 million people worldwide live in iodine-deficient environments and are thus at risk of IDD; 20 million of these are believed to be significantly mentally handicapped as a result. A large proportion of the severely deficient are women in their reproductive years whose babies are at high risk of irreversible mental retardation unless they receive adequate amounts of iodine.

In the last 50 years, many countries in the Americas, Asia, Europe and Oceania have successfully eliminated IDD, or made substantial progress in their control, largely as a result of salt iodization with potassium iodide or potassium iodate and through dietary diversification. For example, in Switzerland, where salt iodization began in 1922, cretinism has been eliminated and goitre has disappeared, while there has been negligible evidence of any adverse effects from iodine intake.

Universal salt iodization[a] has been endorsed in numerous international forums[b] by heads of state, senior government officials, and representatives of international intergovernmental and nongovernmental organizations. Nevertheless, WHO continues to receive queries from national

[a] Universal salt iodization is defined as fortification of all salt for human and animal consumption.

[b] The most important of these are the World Health Assembly, in resolutions WHA39.31 (1986) and WHA43.2 (1990), the World Summit for Children (New York, 1990), the Policy Conference on Ending Hidden Hunger (Montreal, 1991), and the International Conference on Nutrition (Rome, 1992)

health authorities and others seeking reassurance about the safety of providing iodized salt to non-deficient populations. As with all preventive public health measures, the decision to ensure universal salt iodization will be made by weighing the potential risk of excess intake for the few (see pages 3–5) against the well-documented risk of mental and physiological impairment for the many if a deficiency is uncorrected.

In response to concerns expressed, and to facilitate decision-making in countries, this statement summarizes the cumulative scientific and epidemiological evidence in this regard.

Physiological need for iodine

Based on studies of balance and excretion over a 24-hour period, a safe daily intake of iodine has been estimated to be between a minimum of 50 µg and a maximum of at least 1000 µg (2, 3). A generally accepted desirable adult intake is 100–300 µg/day. At all intake levels, a proportionate amount of iodine is excreted in the urine, which is the biochemical basis for assessing iodine status(4).

Usual food sources of iodine

Sea fish, other sea food, and seaweed are rich sources of iodine suitable for human consumption. Iodine is also found in vegetables grown in soils containing adequate amounts of this trace element, and in milk products, eggs, poultry and meat from animals whose diet contained sufficient iodine.

Usual salt intakes

Average daily salt intakes vary from country to country. Usually, consumption levels are within the 5–15 g/day range for children and adults. No increase in salt consumption is called for. Rather, the recommended level of salt iodization should be adjusted to provide approximately 150 µg of iodine/day actually consumed, taking into account usual climatic factors like heat and humidity, which can affect retention of this element. The recommended quantities of iodate to be added to salt under different conditions are provided in Table 1(5). Although potassium iodide was first used in salt iodization, the use of iodate is now recommended

since it is more stable than iodide under varying climatic conditions. Because iodate, on ingestion, is very rapidly reduced to iodide, its use in iodinated salt is equivalent to iodide.

Table 1: ***ICCIDD-UNICEF-WHO recommended levels of iodine in salt***

Examples of desirable average levels at various points in the salt distribution chain, depending on climate, salt intake, and conditions affecting packaging and distribution

Parts of iodine per million parts of salt, i.e. micrograms per gram, milligrams per kilogram or grams per tonne

Climate and daily salt consumption (g/person)	*Requirement at factory* **outside** *the country*		*Requirement at factory* **inside** *the country*		*Requirement at retail sale (shop/market)*		*Requirement at household level*
	Packaging						
	Bulk (sack)	Retail pack (< 2 kg)	Bulk (sack)	Retail pack (<2 kg)	Bulk (sack)	Retail pack (< 2 kg)	
Warm moist							
5 g	100	80	90	70	80	60	50
10 g	50	40	45	35	40	30	25
Warm dry or cool moist							
5 g	90	70	80	60	70	50	45
10 g	45	35	40	30	35	25	22.5
Cool dry							
5 g	80	60	70	50	60	45	40
10 g	40	30	35	25	30	22.5	20

Source: Adapted from *World Summit for Children – mid-decade goal: iodine deficiency disorders*. Geneva, 1994. UNICEF-WHO Joint Committee on Health Policy, document JCHPSS/94/2.7 and reference 5.

N.B. 168.6 mg of KIO_3 contains 100 mg of iodine.
N.B. These are indicative initial levels, which should be adjusted in the light of urinary iodine measurement.

Adverse effects associated with high nutritional intakes of iodine

Since iodine, when ingested in large amounts, is easily excreted through the kidneys into the urine, iodine intakes even at very high levels (milligram amounts) can be consumed safely. However, the following adverse affects, though rare, have been reported.

Allergic reactions to iodine in food. Skin rashes and acne have occasionally been attributed to iodized salt. Such reports are extremely rare, however, and thus these conditions are unlikely to occur following salt iodization. For example, among 20 000 children in the USA suffering from allergy during the period 1935–1974, not a single case was reported of allergic hypersensitivity to iodine in food. Following publication in *Annals of Allergy* of a request for notification of allergy to iodine, not a single report was recorded between 1974 and 1980 (3).

High intakes of dietary iodine and thyroid diseases. Through adaptive mechanisms, normal people exposed to excess iodine remain euthyroid and free of goitre. In certain susceptible individuals, iodide goitre and Hashimoto thyroiditis with hypothyroidism have been observed after iodine intakes of 500–3000 µg/day. The prevalence of susceptible individuals in different countries is not fully known. It has been suggested that high nutritional intake of iodine substantiated by urinary iodine of 1000–10 000 µg/litre—as observed in one country in up to 2% of the population—could have an adverse effect in susceptible individuals and in patients with pre-existing abnormalities of the thyroid gland (3). In this small proportion of the population, chronic excess intake might contribute to the development of Hashimoto thyroiditis, iodide and colloid goitre, and thyroid carcinoma. However, the incidence of follicular thyroid cancer, a more severe form of cancer, is lower in iodine-sufficient than in iodine-deficient areas. There is little indication that iodine in the amounts noted influences the development of any of these thyroid diseases.

In Japan, where dietary iodine intakes are high, it has been shown that:

- normal people who are not iodine-deficient can maintain normal thyroid function states even at intakes of several milligrams of dietary iodine/day;
- the incidence of non-toxic diffuse goitre and toxic nodular goitre is markedly decreased by high dietary iodine intake;
- the incidence of Graves disease and Hashimoto disease does not appear to be affected by high intakes of dietary iodine.

However, high intakes of dietary iodine may induce hypothyroidism in auto-immune thyroid diseases and may inhibit the effects of thionamide drugs (6).

There are well-documented reports of iodine-induced hyperthyroidism (Jod–Basedow phenomenon) where iodine, sometimes in normal quantities, was introduced among iodine-deficient populations. Administration of ordinary amounts of iodine has also been reported to induce hyperthyroidism in people with nodular thyroids, and in other individuals who have no apparent underlying thyroid disease. However, these are transient phenomena, which cease after correction of iodine deficiency; they do not occur in populations with sufficient (i.e. normal) iodine intake.

Current estimates of daily iodine intakes in Canada and the USA are substantially above physiological need—in the range of 460 µg/day among 9–16-year-old children, to greater than 1 mg among as many as 10–20% of adults (7). With a level of iodization that provides these populations approximately 260 µg/day of iodine from salt, it is thus apparent that much of the intake comes from non-salt sources (see below). A survey conducted in 1968–1970 in ten states (USA) showed that where total goitre prevalence was greater than 3.5%, the percentage of individuals with high iodine-excretion values, i.e. more than 800 µg/litre, was 16% compared with 6% in states with lower total goitre prevalence (8).

Other sources of iodine

In industrialized countries there are many adventitious sources of iodine which increase daily intake levels far above the physiological amount provided through iodized salt, for example (3):

- poultry and eggs from animals that consume fish flour as part of their feed and iodoform in water that is used as a disinfectant;
- cow's milk and dairy products from animals fed seaweed, producing an iodine content of milk as high as 694 µg/litre, or that come into contact with iodophors used to clean milking apparatus or as teat dips and udder washes;
- bread and baked goods through the iodates used as oxidants in dough conditioners and cleaning agents for bakery equipment (reports of the iodine content of bread in the USA range from 0 to 268 µg/slice);

- the iodine-containing colouring agents added to some drugs (including many multivitamins, minerals, and antacids as a coating or colouring agent), beverages, foods (including some brands of dry cereal that contain as much as 850 µg of iodine per 20g of product) and cosmetics.

Iodine availability

The iodine content of food actually consumed is not necessarily equivalent to that of raw food since some iodine is lost during cooking. For example, losses of about 20% occur in the iodine content of fish by frying or grilling and as much as 58% by boiling. Iodine consumed in food is generally well absorbed, with the possible exception of people suffering from protein–energy malnutrition, which is of particular concern in high-prevalence, endemic-goitre areas of developing countries.

The uptake of radioactive iodine by an individual thyroid is dependent on the amount of stable, i.e. non-radioactive, iodine in the diet. This is the basis for using radioactive iodine to evaluate thyroid function. Studies from Chernobyl following the nuclear reactor accident in 1986 indicate high thyroid cancer rates, especially among young children. It is postulated that the thyroids of children in this iodine-deficient area experienced an unusual uptake of radioactive iodine released into the atmosphere following the accident. It has been estimated that, in general, iodine prophylaxis, e.g. use of iodized salt, should reduce by twofold to threefold the risk of thyroid irradiation resulting from a nuclear accident (9).

Conclusion

Issues relating to the safety of universal salt iodization have been carefully examined by WHO and by joint FAO/WHO, ICCIDD/UNICEF/WHO and WHO/FAO/IAEA expert groups in the process of preparing recommendations (1, 2, 5, 6). All concerned agree that universal salt iodization is the principal public health measure for eliminating IDD.

Daily iodine intakes of up to 1 mg, i.e. 1000 µg, appear to be entirely safe. Iodization of salt at a level that assures an intake of 150–300 µg/day thus keeps intakes well within a safe daily range for all populations,

irrespective of their iodine status. Daily consumption of 10 g of salt containing 50 parts per million of iodine would add a maximum of only 500 µg of iodine. Thus the likelihood of exceeding an iodine intake of 1 mg/day from iodized salt is quite small.

In susceptible individuals—a minority of adults, usually over 45 years of age, who may or may not have nodular goitres—transient side-effects have been reported at usual intakes exceeding 500–3000 µg/day. The benefits to be derived from universal salt iodization by the more than 1500 million people estimated to be at risk or deficient, and the absence of significant adverse effects among others in the same areas who are not iodine-deficient, far outweigh any risk of excess intake for a small minority.

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