CONTINUING EDUCATION

IODINE

INTRODUCTION

Iodine is an essential micronutrient that typically exists in its ionic form, iodide. The human body contains only about 15–20 mg of iodine of which about 70–80% is in the thyroid gland.1

The iodine content of foods is remarkably variable, even within the same food item, and it is difficult to obtain an accurate estimate of iodine status from dietary intake as data are limited on the iodine content in foods. This wide variability in food content can be explained by the iodine cycle.2 Iodide in soil and seawater is oxidised by sunlight to iodine, which evaporates; whereas some iodide is returned to the soil by rain, more on balance is leached out of soil by glaciation, high rainfall and flooding, increasing the content of river waters, and ultimately reaching the sea.2 Because iodide present in both animals and plants varies with the regional content of the soils and water on which they grew, the content in foods depends on the age, geography and degree of leaching of the local landscape and soil.2 One important consequence of the iodine cycle is its impact on the nutritional status of iodine, which is determined by the geographical distribution of iodine and does not necessarily respond to changes in socioeconomic status or food choices.

The following quiz is designed to enhance your understanding of iodine from a nutritional science perspective. It will test your knowledge of basic concepts including the major food sources and nutrient interactions, requirements, metabolism, deficiency and possible toxicity.

1. Which of the following food groups provide a source of iodine?
   a. Vegetables
   b. Dairy, eggs and meats
   c. Seafood
   d. All of the above

2. What is the recommended dietary intake for iodine?
   a. 150 µg
   b. 70 µg
   c. 45 µg
   d. 35 µg

3. What is the upper limit of intake for iodine?
   a. 550 µg/day
   b. 1100 µg/day
   c. 1700 µg/day
   d. There is no reported toxicity and no upper limit of intake

4. What is the main function of iodine?
   a. Cofactor for enzymes involved in the production of ATP
   b. Cofactor for enzymes involved in catabolism of purines and pyridines
   c. As a component of thyroid hormones
   d. All of the above

5. Selenium deficiency has been reported to exacerbate the effects of coexisting iodine deficiency through which of the following?
   a. Iodothyronine deiodinase activity
   b. Glutathione peroxidase activity
6. Which of the following foods have the potential to impair the absorption and/or utilisation of iodine?
   a. Cabbage, cauliflower, broccoli, Brussels sprouts
   b. Cassava, maize
   c. Lima beans and bamboo shoots
   d. All of the above

7. Which of the following are possible consequences to the infant of iodine deficiency in the mother?
   a. Cretinism
   b. Retarded physical development
   c. Impaired mental function
   d. All of the above

8. In addition to goitre, which of the following can be used to assess iodine status?
   a. Urinary iodide excretion
   b. Serum TSH
   c. Iodide in drinking water
   d. All of the above

9. What is the approximate prevalence of iodine deficiency in Australian children?
   a. 60–70%
   b. 40–50%
   c. 20–30%
   d. There is no iodine deficiency in Australian children

ANSWERS

1. d.

   Iodine is found in a wide variety of foods, including vegetables, dairy, eggs, meats and seafood. The content in foods shows a distinctive pattern, which can be explained by the iodine cycle, with highest concentrations in marine species and relatively low concentrations in land-based foods. The richest sources are seaweed (up to 500 µg/100 g), marine fish (up to 250 µg/100 g) and shellfish (up to 160 µg/100 g). In seaweeds, the concentration is higher in freshly cut blades and lower in sun-bleached blades due to ready evaporation of the iodine.

   In an analysis conducted by the United States Food and Drug Administration of the iodine content in 234 commonly consumed foods, typical values for food groups were relatively low, ranging from 2 to 50 µg/serving (Table 1). However, large coefficients of variation, up to 300%, indicated wide variation in content within individual items. In Australia and New Zealand the major sources of iodine are seafood, iodised salt, milk and eggs, with minor amounts from meat and cereals. Iodised salt and food additives, such as calcium iodate and potassium iodide, contribute to the variation in iodine content of processed foods. Cooking methods determine the final iodine content, with losses ranging from 6% for roasted food items, up to 80% when items are boiled due to leaching into cooking water.

<table>
<thead>
<tr>
<th>Plant foods</th>
<th>µg/serving</th>
<th>Animal foods</th>
<th>µg/serving</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leafy vegetables</td>
<td>2</td>
<td>Eggs</td>
<td>25</td>
</tr>
<tr>
<td>Roots/tubers</td>
<td>16</td>
<td>Dairy products</td>
<td>41</td>
</tr>
<tr>
<td>Legumes</td>
<td>15</td>
<td>Fish</td>
<td>50</td>
</tr>
<tr>
<td>Fruit/fruit juice</td>
<td>4</td>
<td>Poultry</td>
<td>20</td>
</tr>
<tr>
<td>Bread, pasta</td>
<td>36</td>
<td>Meat</td>
<td>16</td>
</tr>
<tr>
<td>Nuts</td>
<td>2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. a.

   In the Nutrient Reference Values for Australia and New Zealand, released in 2006, the recommended dietary intake (RDI) is defined as the average daily dietary intake level that is sufficient to meet the nutrient requirements of 97–98% of the healthy individuals in a particular life stage and gender group. The RDI value of 150 µg for adults incorporates the estimated average requirement of 100 µg/day, derived from experimental iodine balance studies and studies relating urinary iodide to thyroid volume. The RDI of 150 µg/day is the same for both men and women and increases to 220 µg/day for pregnancy and 270 µg/day for lactation.

   The other values listed are adult RDIs for selenium (b) and molybdenum (c), and (d) represents the adequate intake for chromium, a figure used when an RDI cannot be determined. The adequate intake is the average daily nutrient intake level based on experimentally determined approximations, or observed estimates of intake in apparently healthy population groups.

3. b.

   The upper limit of intake for iodine is set at 1100 µg/day, which is based on a lowest observed adverse effect level of 1700 µg/day, incorporating an uncertainty factor of 1.5 for individual responsiveness. An early effect of iodine excess is an elevated concentration of serum thyroid stimulating hormone (TSH). Although exposure to high concentrations of iodine from food or supplements can increase TSH for about 24 hours, most healthy individuals compensate by reducing iodine uptake into the thyroid.
gland and excreting the excess iodine in urine. However, in some regions of Japan and China where iodine-rich seaweeds are consumed as dietary staples, the sustained high intake of iodine (50–80 times the upper limit) may produce goiter. At these high intakes, uptake of iodine into the thyroid gland appears to be inhibited, resulting in reduced synthesis of thyroxine and elevated secretion of TSH, which stimulates the thyroid follicles to enlarge and multiply, producing a goitre. Besides goiter, other reported adverse effects include thyroiditis, hyperthyroidism, sensitivity reactions and thyroid papillary cancer. Certain individuals with autoimmune thyroid disease, iodine deficiency or thyroid cancer may be particularly sensitive and respond adversely to intakes considered safe for the general population.

4. c.
The main function of iodine is to form a component of thyroid hormones. Thyroid hormones stimulate the basal rate of metabolism, oxygen consumption by cells, and heat production. They are especially important in infants, children and adolescents for normal development of the nervous system and linear growth. Unlike many other trace elements such as iron, zinc and molybdenum, iodine has not been identified as a cofactor for enzymes. Iodine is typically found in its ionic form, iodide. The main fate of absorbed iodide is uptake and concentration by the thyroid gland via an active transport mechanism known as the iodine transporter or pendrin. Iodide is then oxidised to iodine by thyroid peroxidase and successively incorporated into tyrosine residues on thyroglobulin. The tyrosines are iodinated at one (monoiodotyrosine) or two (diiodotyrosine) sites and then coupled to form the active thyroid hormones, tetraiodothyronine or triiodothyronine (T₃) and triiodothyronine (T₄). The final active thyroid hormones (T₃ and T₄) are liberated from thyroglobulin by proteases and released into the circulation. This mechanism is regulated by TSH.

5. a.
The selenium-dependent iodothyronine deiodinases have a specific role in the assimilation of iodine, which explains why selenium deficiency can exacerbate the effects of coexisting iodine deficiency. Deiodinases catalyse the deiodination of thyroid hormones and are found widely in body tissues including the liver, kidney and muscle, skin, pituitary, adipose tissue and brain. The selenium-dependent 5′-deiodinase, which converts T₄ to T₃, is especially important because T₃ is the most biologically active form. Although the concentrations of circulating T₃ are nearly 50 times those of T₄, T₃ has relatively low biological activity and serves as a reservoir for the more active form following removal of iodine. Selenium deficiency decreases 5′-deiodinase activity resulting in reduced conversion of T₄ to T₃ and hence reduced activity of the thyroid hormone. An inverse association has been observed between selenium status and thyroid volume in French adults (cited in the study by Gibson). The enzymes glutathione peroxidase (b) and thioredoxin reductase (d) are selenium-dependent enzymes and selenoprotein P (c) is the transport protein for selenium.

6. d.
Substances in some foods that inhibit the utilisation of absorbed iodide are called goitregens. These may interfere with iodide metabolism by affecting iodide uptake into the thyroid gland, oxidation of iodide to iodine by thyroid peroxidase, or release of thyroid hormone from the thyroid gland. The sulphur-containing thionamides found in brassica vegetables such as cabbage, cauliflower, broccoli and Brussels sprouts can prevent oxidation of iodide to iodine, and interfere with synthesis of thyroid hormones by impairing the binding of iodine to thyroglobulin. The cyanogenic glucosides found in some staple foods, such as cassava, maize, bamboo shoots, sweet potatoes, lima beans and millet, release the goitregen thiocyanate, which competes with iodide, blocking its uptake by the thyroid gland. With the exception of cassava, these glucosides are located in the inedible parts of the food, or occur in such small amounts that they do not cause a major problem. However, consumption of cassava as a staple food, especially in iodine-deficient areas, has been associated with aetiology of goitre.

7. d.
The term iodine deficiency disorders refers to a spectrum of effects of iodine deficiency on growth and development at all stages of the life cycle. In moderate deficiency, the thyroid gland enlarges to increase its capacity to absorb iodine, hence forming a goiter. The goiter may be reversible by treatment with iodine administration. Severe iodine deficiency in the mother has serious consequences for the foetus, resulting in cretinism that may occur in one of two forms, depending on the nature and severity of the deficiency and genetic factors. Neurological cretinism is characterised by
mental deficiency, deaf mutism and disorders of gait (spastic diplegia). In contrast, myxoedematous cretinism is less common and characterised by dwarfism and mental deficits. Myxoedematous cretinism is partially reversible with treatment but neurological cretinism is irreversible. Endemic cretinism affects up to 10% of the populations living in iodine-deficient areas in India, Indonesia and China. The most cost-effective and widely used method to prevent goiter and cretinism has been the use of iodised salt; alternative methods include iodised oil injections, iodised bread and iodised water. Goitre is usually seen with iodine intakes of less than 50 µg/day and cretinism can occur with intakes of less than 30 µg/day in the mother, both may be exacerbated by coexisting selenium deficiency and/or consumption of goitrogens.

8. d.
Iodine status is determined by both physical examination of the thyroid gland and biochemical assessment, mainly the measurement of iodide in urine. To determine the prevalence of goiters, the size of the thyroid is measured by clinical inspection and palpation or ultrasound. Urinary iodide excretion (UIE) is reduced during deficiency and is often used to measure iodine status in children. The kidneys provide a major route for iodide excretion (80–90%) and urinary iodide is positively correlated with dietary intake; faecal excretion of iodide is relatively low. According to World Health Organization criteria, UIE levels ≥100 µg/L are defined as iodine-replete, whereas UIE levels of 50–99 µg/L or 20–49 µg/L indicate mild or moderate iodine deficiency, respectively. Limitations of this method include variable losses of iodide in sweat especially in tropical regions; and the possible presence of dietary goitrogens, which prevent uptake of iodine into the thyroid gland, resulting in anomalously high circulating and urinary levels – which could mask reduced iodine status.

Serum TSH concentrations are raised early in iodine deficiency and provide a sensitive indicator of functional status. Iodine concentrations in the local drinking water provide an indication of content in the soil. In general, iodine-deficient areas have water iodine levels below 2 µg/L.

9. b.
Surveys to monitor iodine status in Australia have been conducted by the Australian Centre for Control of Iodine Deficiency Disorders, based on measurement of UIE. In the recent National Iodine Nutrition Study of 1709 Australian boys and girls from 88 schools across five states, about half (46.3%) of the children tested in mainland Australia had UIE levels consistent with mild (36.7%) to moderate (9.6%) iodine deficiency. Deficiency was greatest in children from south-eastern Australia, with the higher prevalence of moderate deficiency in Victoria (19%) and NSW (14%), and lower prevalence in Western Australia (6%) and South Australia or Queensland (both 3%). Tasmania and Northern Territory were excluded because of an iodine fortification program or for logistical reasons, respectively.

These results contrast with those of an earlier survey conducted in 1992 by the Australian Centre for Control of Iodine Deficiency Disorders, in which median UIE levels in the Australian population were almost twofold higher than in the recent survey (>200 µg/L vs 104 µg/L, respectively) and consistent with iodine sufficiency. Possible reasons for the decline in iodine status include reduction in use of iodophores to sanitise vessels within the dairy industry, and decrease in use of iodised salt. Because geographical deficiency of iodine is a major determinant of iodine deficiency, specific interventions and effective health education will be required to address the low iodine status in Australia and prevent development of overt iodine deficiency diseases. Risk groups include individuals restricting salt intake, those whose consumption of dairy products or fish is negligible (especially with vegan diets), pregnant and lactating women and infants.

REFERENCES
6 National Health and Medical Research Council, Department of Health and Ageing, Australian Government.


