Iodine

Atomic number 53
Atomic weight 126.9

Collection

Serum/Plasma	5 mL	Lithium heparin, sodium heparin or plain tubes (vacutainers acceptable, but avoid gel separators). Send separated plasma or serum.
Urine	20 mL	Sterile Universal
Breast milk	20 mL	Sterile Universal

Reference ranges

			Reference
Serum/plasma			
Blood			
Urine	µmol/L	See below	1-3
	µmol/24 h	0.60 - 4.30	1-3
	µmol/mmol creatinine	0.05 - 0.36	1-3

Urine Iodine (µmol/L)- Interpretation	
severe reduction in iodine intake	less than 0.16
moderate reduction in iodine intake	0.16 – 0.39
mild reduction in iodine intake	0.39 – 0.78
adequate iodine intake	0.79 – 1.57
iodine intake above requirements	1.58 – 2.36
excessive iodine intake	greater than 2.36
Pregnancy and lactation (µmol/L)	
insufficient iodine intake	less than 1.18
adequate iodine intake	1.18 – 196
iodine intake above requirements	1.97 – 3.93
excessive iodine intake	greater than 3.93

Notes
Concentrations in serum and whole blood provide no useful information relevant to nutritional status [2,3]

References

1. Konig F, Andersson M, Hotz K et al. Ten Repeat Collections for Urinary Iodine from Spot Samples or 24-Hour Samples Are Needed to Reliably Estimate Individual Iodine Status in Women. J Nutr 2011; 141 (11): 2049-2054.
2. Zimmermann B, Jooste P, Pandav C. Iodine-deficiency disorders. Lancet 2008; 372: 1251-1262.
3. World Health Organisation/International Council for the Control of the Iodine Deficiency Disorders/United Nations Childrens Fund (WHO/ICCIDD/UNICEF). Assessment of the iodine deficiency disorders and monitoring their elimination. WHO Geneva: World Health Organisation, 2008.

Clinical

Sources:

Sources of iodine include soil, sea water, food (e.g. marine fish, seaweed, eggs and milk), food supplements, medicines such as amiodarone, wound disinfectants, radio-contrast material and photographic solutions.

Biological importance:

Iodine is a constituent of the thyroid hormones, thyroxine (T4) and tri-iodoothyronine (T3), which are essential for growth and development – particularly of the central nervous system.

Biology:

Iodine is present as iodide (I^-) in body fluids. It is absorbed rapidly from the intestinal tract and largely confined to the extra-cellular fluid with some in red blood cells. However, it is concentrated in the thyroid, stomach, salivary and mammary glands by a sodium-iodide transporter which is also expressed in the placenta. Overall >90% of absorbed iodine is excreted in the urine. Under normal circumstances plasma iodine has a half-life of about 10 hours.

In the thyroid, through the action of thyroid peroxidise, I^- is oxidised and incorporated into tyrosine residues of thyroglobulin (organification) forming mono- and di-iodotyrosine. These are then coupled to produce T3 and T4. Selenium-containing de-iodinases convert T4 to T3 in the thyroid gland, peripheral tissues and brain releasing iodine for re-use.

Recommended intakes (ug/day):

- Children 0-5 years 90 µg
- Children 6 to 12 years 120 ug
- Children > 12 years 150 ug
- Adults 150 ug
- Pregnancy 250 ug
- Lactation 250 ug

Deficiency:

World-wide, deficiency is a major health problem. It occurs in regions where the soil and groundwater are deficient in iodine. Crops grown in these soils will be low in iodine concentration and hence man and animals consuming food grown in these soils become iodine deficient.

The thyroid adapts to low dietary iodine by increasing secretion of TSH by the pituitary gland. TSH stimulates iodide uptake by the thyroid gland, which leads to reduced renal clearance and hence reduced urinary iodine excretion. TSH also stimulates thyroglobulin breakdown and increases T3 production relative to T4. In chronic iodine deficiency the total thyroid iodine content is depleted which results in hyperplasia of thyroid epithelial cells which may be clinically evident (goitre).

Iodine deficiency can affect human beings at all stages of the life cycle. The most serious adverse effect of iodine deficiency is damage to the fetus. Normal amounts of thyroid hormones are required for neuronal migration and myelination of the fetal brain. Severe iodine deficiency during pregnancy increases the risk of stillbirths and congenital abnormalities. Children who are iodine deficient can lose up to 13.5 IQ points.

Toxicity:

Iodine is a strong oxidising agent and it acts as an acid corrosive. Iodine vapour irritates the eyes, skin and mucous membranes.

There is a high tolerance to ingested iodine with a wide safety margin. Iodine intakes of up to 1000 ug/day are well tolerated by most adults although in children chronic intakes of 500 ug/day or more are associated with mild thyroid enlargement.

Causes of iodine toxicity include accidental or deliberate ingestion which causes corrosive damage to the gastrointestinal, cardiovascular collapse and renal failure. In the presence of starch blue I^- salts form - blue emesis is characteristic of iodine poisoning. Commoner causes of iodine toxicity are heavy applications of iodine to open wounds, large injections of iodine-containing radio-contrast media and amiodarone treatment for cardiac arrhythmias.

Iodine toxicity causes a rapid decrease in release of T3 and T4 from thyroglobulin; decreased I^- uptake by the thyroid gland; decreased organification of iodine and hence decreased T3 and T4 synthesis. This culminates in acute hypothyroidism with low plasma T3 and T4 and high TSH.

Laboratory indices:

Urine is the matrix of choice for suspected iodine toxicity and for the assessment of recent iodine *intake*.

For epidemiological studies, such as school-based surveys, the *median* urinary iodine concentration can be used to classify that ***population's*** iodine status. Adequate iodine status is indicated by a population median urinary iodine concentration >100 ug/l.

However, the urine iodine test is not an appropriate test to diagnose iodine *deficiency* in individuals. A common mistake is to assume that all subjects with a random urinary iodine concentration of <100 ug/l are iodine deficient. Urine iodine levels have a low predictive value in an individual because urinary iodine concentration varies substantially between days and seasons, as well as within a day (up to threefold) as a consequence of circadian rhythm and due to differences in fluid intake. Because the thyroid gland can store large amounts of iodine (12-16 mg in an iodine sufficient individual) a 'low' urine iodine concentration no more indicates iodine deficiency than a low urinary sodium indicates sodium deficiency

Recently it has been suggested that iodine status can be reliably estimated if 10 repeat random urine samples are collected, but clearly the large number of samples required is a major limitation.

References:

1. Zimmermann B, Jooste P, Pandav C. Iodine-deficiency disorders. *Lancet* 2008; **372**: 1251-1262.
2. World Health Organisation/International Council for the Control of the Iodine Deficiency Disorders/United Nations Childrens Fund (WHO/ICCIDD/UNICEF). Assessment of the iodine deficiency disorders and monitoring their elimination. WHO Geneva: World Health Organisation, 2008.
3. Konig F, Andersson M, Hotz K *et al.* Ten Repeat Collections for UrinaryIodine from Spot Samples or 24-Hour Samples Are Needed to Reliably Estimate Individual Iodine Status in Women. *J Nutr* 2011; **141 (11)**: 2049-2054.