Rocky Mountain Analytical®
Changing lives, one test at a time

Iodine Plus

Clinical Information for Professionals

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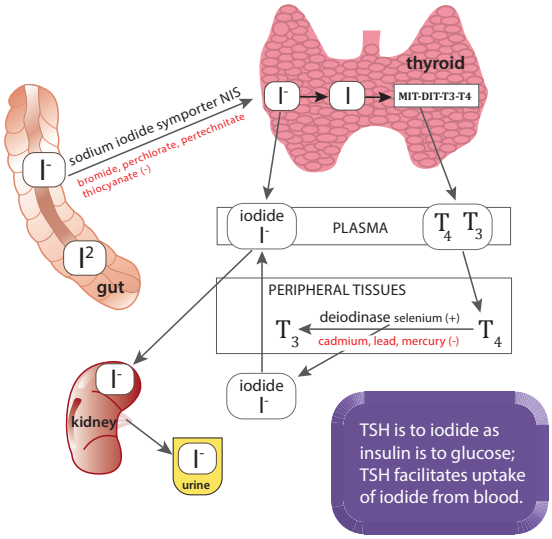
Iodine Plus measures levels of iodine, selenium, bromine and cadmium in a first morning or 24-hour urine collection. Iodine and selenium are key elements in thyroid function, and bromine and cadmium can interfere with the actions of iodine and selenium respectively.

Iodine Metabolism & Physiology

Taken orally, iodine is rapidly converted from iodine to iodide. The thyroid gland actively absorbs iodide from blood and incorporates it into thyroxine (T4) and triiodothyronine (T3). In the presence of an iodine deficiency, iodine concentrates within the thyroid to ensure adequate production of thyroid hormones.[1]

Toxic chemicals may impair iodine transport and adversely affect thyroid function. For example: chemicals that interfere with the sodium-iodide symporter (see diagram at right) may inhibit delivery of iodine to thyroid and other iodine dependent tissues. Toxic chemicals include: bromine, nitrate, thiocyanate (found in cigarette smoke) and perchlorate (used in fireworks, propellants, flares, and automobile airbags.[2]

The primary physiological role of iodine is to be incorporated into thyroid hormones by the thyroid gland. However, in an iodine sufficient adult, only 30% of total body iodine is concentrated in the thyroid tissue and thyroid hormones.[3] The remaining iodine is distributed to other iodine-dependent tissues including: breast, prostate, cervix, eye, salivary gland, and gastric mucosa. In non-thyroid tissues, iodide can act as an electron donor and protect tissues from reactive oxygen species and tissue damage. Glands lacking in iodine have increased levels of malondialdehyde, a product of lipid peroxidation and known risk factor for cancer and degenerative tissue damage.[4] Adequate iodine intake during gestational development is critical for proper maturation of the fetal central nervous system. Iodolipids regulate cell cycling in estrogen-sensitive tissues. Iodine is bound into antiproliferative iodolipids in the thyroid, producing iodolactones, which may contribute to proliferative control of mammary glands. [5]



Measuring Iodine

The Canadian Health Measures Survey (CHMS) conducted from 2009 to 2011 found that 22% of Canadians had a mild iodine deficiency, 7% had a moderate deficiency and 15% had excessive levels.[6] The fact that nearly half of Canadians have out-of-range iodine levels suggests that routine measurement of iodine levels may be worthwhile.

According to the CHMS, over 90% of iodine ingested in food and beverages is excreted in urine. Thus, iodine levels in urine reflect the amount of iodine consumed and present in the body 24 to 96 hours prior to ingestion. If dietary intake changes significantly during that time, iodine levels will not reflect habitual intake. [6]

To maintain quality results, Rocky Mountain Analytical (RMA) participates in Ensuring the Quality of Urinary Iodine Procedures (EQUIP), an international proficiency testing program managed by the American Centre for Disease Control and Prevention. RMA also participates in the Quebec Multielement External Quality Assessment Scheme (QMEQAS) managed by the Centre de Toxicologie du Quebec.

Iodine Plus

Measures iodine, selenium, bromine, and cadmium in first morning or 24-hour urine.

Importance of Selenium

Selenium is essential to the following selenoproteins (selenium-containing proteins): deiodinases, thioredoxin reductases, and glutathione peroxidases.[7]

Deiodinases are required to convert thyroxine (T4) to triiodothyronine (T3) in target tissues, which may include: breast, prostate, thyroid, brain, muscle, heart, and ovary. Animal models have shown that impaired hepatic deiodination is associated with cadmium, mercury, and lead toxicity.[8]

Other selenoproteins are essential for fertility and fetal development, mitochondrial function, calcium homeostasis, liver and kidney health, plus general antioxidant status. Selenium deficiency has been implicated in cardiovascular disease, various cancers, myopathies, and is currently under investigation for its role in insulin resistance and diabetes. [7]

Excess Bromine

Neurotoxicity and thyroid dysfunction are major clinical and research concerns associated with excess bromine exposure. Polybrominated diphenylethers (PBDEs) used as flame retardants are common brominated toxins, with significant effects:

- Elevated PBDE in breast milk correlates with lower birth weight and length, decreased BMI and reduced head and chest circumference.[9]
- PBDE levels in breast milk correlate with increased incidence of undescended testicles in newborns.[10]
- PBDEs affect neuron activity on several levels including: presynaptic neurotransmitter homeostasis, intracellular signaling, and neurotransmitter release. [11,12] The toxic effect of PBDEs on nervous system development involves changes in the cholinergic system and may also be related to altered thyroid homeostasis.
- PBDEs may compete with thyroxine (T4) for the serum transport protein, transthyretin.[13]
- PBDEs can disrupt calcium homeostasis by causing mitochondria and endoplasmic reticulum stores to release calcium. This in turn alters both calcium channel activity in cell membranes and neurotransmitter release. [12]
- Hydroxylated PBDEs have been shown to inhibit the aromatase enzyme.[11]
- PBDEs cause oxidative stress.[11]

Excess Cadmium

Cadmium is considered an endocrine/neuroendocrine disrupter, and tends to concentrate in the kidney and thyroid.[14] The biological half-life of cadmium is more than 5 years, and exposure typically occurs through inhalation.[15] There are several means by which cadmium can interfere with biological functions:

- Cadmium induces reactive oxygen species (ROS).[14]
- Cadmium acts on the pituitary glands, resulting in secretory changes in prolactin, growth hormone, ACTH and TSH that can negatively impact sperm production, follicular development and other gonadal functions. [14]
- Cadmium toxicity impairs deiodination, leading to lower plasma T3 and T4 while the effects of cadmium on the pituitary may prevent compensatory increases in TSH in response to low T3 and T4.[14]
- Cadmium may interfere with steroid hormone synthesis. Cholesterol is transported into cell membranes for steroidogenesis via a T3 dependent transport protein. Because cadmium impairs deodination and T3 levels, the availability of transport proteins for steroid synthesis may be affected.[14]
- Cadmium is also known to displace other elements (i.e. zinc, iron, magnesium, calcium, and copper) from biological systems and tissues. [14]

Effects at a Glance

Iodine

- thyroid hormone production
- fertility/fetal development
- protection against breast and prostate cancer
- protection against autoimmune disease

Selenium

- conversion of T4 to T3
- fertility/fetal development
- mitochondrial function
- calcium homeostasis
- antioxidant

Excess Bromine

- neurotoxicity
- thyroid disruption
- calcium homeostasis disruption

Excess Cadmium

- neuroendocrine disruption
- endocrine disruption
- impaired conversion of T4 to T3
- formation of reactive oxygen species & malondialdehyde
- glutathione decrease
- mitochondrial damage

Conditions & Symptoms	Nutrient	Toxin	Literature
Fibrocystic breast disease	Iodine		Iodine adminstered at a dose of 3 to 6 mg/day over 6 to 18 months resulted in statistically significant decreases in pain and fibrosis compared to placebo or 1.5mg daily.[16]
Breast Cancer			Tissue iodine levels were found to be lower in patients with breast cancer compared to those with normal breast tissue or benign breast tumors.[17] In rats, administration of Lugol's solution or wakame seaweed suppressed the development of mammary tumors. Combined iodine and selenium deficiency may facilitate development of breast cancer. Iodine is bound into antiproliferative iodolipids in the thyroid, producing iodolactones, which may contribute to proliferative control of mammary glands. [18]
Hypothyroidism	Iodine, Selenium		Selenium in biological samples of patients with hypothyroidism can play an important role in determining the severity of hypothyroidism associated with iodine deficiency.[19]
Neurologic Disease	Iodine, Selenium	Bromine (PBDEs), Cadmium	Thyroid hormones are involved in neuronal proliferation, migration synaptogenesis, synaptic plasticity, and myelination, all of which have been shown to be directly affected by PBDEs.[12]
Childhood IQ & Development	Iodine		Adequate iodine in breast milk is essential for neonatal neurological development.[20] Children born to be iodine deficient mothers show reduced performance in spelling, grammar, and general English literacy. [21]
Autoimmune disease	Iodine, Selenium	Tobacco smoke (cadmium), PBDEs (bromine)	Excess iodine may enhance the activity of lymphocytes and macrophages associated with chronic thyroiditis.[21] Environmental pollutants interfere with iodine transport and induce oxidative stress.
Celiac Disease	Selenium		Impaired selenium absorption due to celiac disease may modulate selenoprotein (SeP) gene expression, thus promoting both intestinal mucosal damage and complications like autoimmune thyroid disease.[21]

Iodine

Food Sources:

Sea vegetables, including,
- wakame seaweed
- kelp, dulse
- iodized salt
- eggs

Herbal Sources:

- Bladderwrack (a seaweed)

Selenium

Food Sources:
- brazil nuts
- oysters
- seafood
- sunflower seeds
- meats
- mushrooms
- whole grains (rye, brown rice)

Bromine

Avoid exposure found in:

- fire retardants on new furniture and clothes (polybrominated diphenylethers, PBDEs)
- water systems using bromine disinfectants (e.g. hot tubs)
- medications with bromine salts (e.g. ipratropium bromide)
- potassium bromate (U.S. bread dough conditioner, banned in Canada)

Cadmium

Avoid exposure found in:

- cigarette smoke
- mining and smelting
- steel production, machining
- leaking or wrongly disposed batteries
- heating or burning of plastics
- contaminated herbs imported from outside North America
- imported children's toys, jewellery
- yellow pigmented pottery glaze

Who to Test

Workers in hazardous occupations like:

- Metal plating (cadmium exposure)
- Battery production (cadmium exposure)
- Plastics and other synthetics (cadmium exposure)
- Plumbing and indoor water treatment facilities (bromine exposure)
- Furniture and clothing production/distribution (bromine exposure)
- Agriculture workers exposed to conventional pesticides (bromine exposure)

Patients with:

- Personal or family history of thyroid disease
- Personal or family history of breast or prostate disease
- Bromine-containing medications i.e. ipratropium bromide inhalers
- Plans to conceive
- Patients who are currently pregnant
- Hypertension
- Smoking history

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