# Selenium

**Pseudonyms** – Se

Selenium is an essential cofactor for many proteins and enzymes, including the deiodinases involved in thyroxine metabolism. Deficiency is rare, but more frequent than toxicity.

Plants are the major source of selenium in the diet and their content depends on soil levels of selenium. Serum levels are therefore highly dependent on adequate dietary intake. Selenium is very efficiently absorbed as selenite or seleno-methionine in the duodenum and is metabolised by the liver so it can be incorporated into the selenoproteins. Excretion occurs predominantly in the urine. Direct measurement of serum (or whole blood) selenium reflects recent changes in dietary intake and is relatively straightforward compared to measuring activity of selenoproteins as alternative biomarkers of selenium status. However, activity of selenium-containing glutathione peroxidise may be a better indicator of total body levels.

Deficiency of selenium is associated with Keshan disease (cardiomyopathy with arrythmia) and Kashin-Beck disease (osteoarthropathy in young patients with severe enlargement and dysfunction of joints) but both of these are usually only ever seen in China.

Overt deficiency is rarely seen in European countries but it is thought sub-optimal levels are associated with an increased risk of a wide range of problems including increased susceptibility to viral infections, increased cancer risk and reduced male fertility. Thyroid function is not thought to be affected unless there is a concomitant iodine deficiency such as that seen in central Africa.

Selenium toxicity is referred to as selenosis and is not thought to be observed until serum levels have exceeded 12.7 µmol/L. The symptoms include gastrointestinal upsets, hair loss, white blotchy nails, garlic breath odour, fatigue, irritability and mild nerve damage. This is a rare event other than accidental industrial exposure.

A baseline selenium measurement is recommended in Nice Clinical guideline [CG32] primarily for adults having parenteral nutrition in the community, who are at risk of selenium depletion.

## General information

**Collection container:** The sample must be collected into a plain plastic bottle, which has been shown to be suitable for trace metals.

Adults (internal users): 4.9mL Gel-free Serum (Sarstedt white top)/LiHep Plasma (Sarstedt orange top)

Paediatrics (internal users): 1.2mL Serum (Sarstedt white top)/LiHep Plasma (sarstedt orange top)

External users: separated serum/plasma in a plain plastic tube, which has been shown to be suitable for trace metals

**Type and volume of sample:**

Whole blood: 1.0mL

Separated serum/plasma: minimum 150 µL

**Specimen transport/special precautions**: None

## Laboratory information

**Method principle:**

Inductively Coupled Plasma Mass Spectrometry (ICP-MS), Agilent 7800 series.

**Biological reference range or cut off:**

| | |
|---|---|
| Up to 2 years | 0.2 - 0.6 µmol/L |
| 2 to 16 years | 0.44 - 1.4 µmol/L |
| >16 years | 0.8 - 1.5 µmol/L |

**Turnaround times:**

1 week

## Clinical information

**Factors known to significantly affect the results**:

Selenium is a negative acute phase reactant and can be up to 60% lower in ICU patients due to redistribution throughout tissues, rather than increased excretion. Smaller decreases can be observed post-operatively and a CRP may be useful in interpreting the result.

**Clinical decision points**:

Refer to reference range information above.

**References:**

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