Sodium selenate

CAS: 13410-01-0
MF: Na_2SeO_4
MW: 188.9
Solubility: water-soluble.

Se
MW: 78.96

Major uses
Sodium selenate is used as a chemical reagent and as insecticide for certain horticultural applications on inedible plants [1].

Human toxicity
Elemental selenium metal has a relatively low order of toxicity. Acute poisoning with selenium and its salts are rare, however, in excessive amount toxic and even fatal action was documented.

Normal whole blood selenium levels in adults range from 0.085 to 0.13 mg/l [2].

All selenium salts can produce toxicity by ingestion, inhalation, and percutaneous absorption, although specific information on the possible dermal absorption of sodium selenate was not found. Sodium selenate is the least toxic inorganic selenium form; sodium selenite (Na_2SeO_3) is more toxic [3].

Several toxic symptoms are listed here [1]:

- cardiovascular: ECG changes indicating myocardial damage; hypotension; in severe cases – fatal cardiac arrest;
- respiratory: pulmonary edema, respiratory tract irritation, cough, and respiratory failure;
- neurologic: CNS depression, convulsions (at acute selenium poisoning), dizziness, irritability, restlessness, headache, and coma;
- gastrointestinal: nausea, vomiting, salivation, and gastrointestinal pain.

The ingestion of 22 mg/kg of selenium (in form of sodium selenate) produced minimal toxicity, despite a substantial rise in the blood level (3.1 mg/l, corresponding to 39.3 μM) [4]. In another case described in the literature, selenium blood concentration of 2.4 mg/l (30.9 μM) was fatal [2]. Average selenium blood concentration in 4 fatalities after ingestion of sodium selenite or selenious acid was 5.9 mg/l (range 0.5-18 mg/l) [5].

Kinetic data
There is little information in the literature about sodium selenate, but some data about selenium are listed here.

Kinetics: Triphasic ? (AK*) (there are three elimination phases after ingestion of therapeutic doses of selenite; see *Elimination*)

Absorption: elemental selenium is well absorbed from the lungs and gastrointestinal tract (57% and 50%, respectively) (animal data) [3]. The absorption of sodium selenate results from the action of sodium-mediated carrier transport shared with sulfate, whereas the absorption of sodium selenite occurs via passive diffusion [6].

Volume of distribution: unknown.

Distribution: highest concentration of selenium is found in kidney, liver and spleen [5].

Plasma half-life: for selenium 69-77 days [5].

Passage of blood-brain barrier: restricted (AK) {reviewed in [5]}.

Plasma half-life: unknown.

Time to peak blood concentration: unknown.

Plasma-protein binding: selenium is incorporated into serum proteins [5].

Elimination: for sodium selenite, the initial elimination phase is rapid (1 day), while the second phase is 8-20 days, and third phase is 65-116 days [1].

Metabolism and excretion

Selenium is an essential trace element in man, known to be a co-factor for red blood cell glutathione peroxidase (reviewed in [7]). Selenium is an active catalyst in peroxide metabolism [8]. Chronic deficiency of selenium can lead to cardiomyopathy (heart muscle inflammation).

In animals, the major metabolites of selenium are trimethylselenonium ion (up to 30% in exhaled air, largely as dimethylselenium) [5]. Trimethylselenonium ion is an inactive metabolite of selenium.

In humans, sodium selenate is converted to dimethyl selenide. The methylated forms are generally less toxic compared with the nonmethylated selenium compounds [6].

Excretion: nearly all of daily dietary intake of selenium can be accounted for in daily urine (20-50 μg) , feces (8-30 μg), and miscellaneous excreta (32-80 μg) [5].

Toxicological mechanisms

The mechanisms of selenium-induced toxicity are unknown; however, some hypothetical mechanisms were outlined (reviewed in [7]):

- disruption of cellular peroxide reactions of free radical generation or trapping
- interference with sulfhydryl-dependent enzyme activities and depletion of glutathione
- inhibition of protein synthesis.

As sodium selenate, selenium is highly toxic; it binds sulfhydryl enzymes and produces a clinical syndrome similar to that of arsenic [8].

Target organs (for selenium): CNS, heart, lung, liver, kidney [1, 5].

References

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