Iodic acid and selected salts: Human health tier II assessment

27 October 2017

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Iodic acid (HIO3), sodium salt	7681-55-2
Iodic acid (HIO3), potassium salt	7758-05-6
Iodic acid (HIO3)	7782-68-5
Iodic acid (HIO3), calcium salt	7789-80-2
Iodic acid (HIO3), magnesium salt	7790-32-1
Iodic acid (HIO3), calcium salt, hexahydrate	10031-33-1
Iodic acid (HIO3), potassium salt (2:1)	13455-24-8

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:**www.nicnas.gov.au**

Disclaimer

ACRONYMS & ABBREVIATIONS

Grouping Rationale

This group of chemicals include iodic acid (CAS No. 7782-68-5) and its respective salts: sodium iodate (CAS No. 7681-55-2), potassium iodate (CAS No. 7758-05-6), calcium iodate (CAS No. 7789-80-2), magnesium diiodate (CAS No. 7790-32-1), calcium iodate hexahydrate (CAS No. 10031-33-1) and potassium hydrogen diiodate (CAS No. 13455-24-8).

These chemicals share similar physicochemical properties and reported uses, and have been shown to have similar systemic toxicological profiles (see **Hazards** section); therefore, these chemicals are qualified to be assessed as a group. The cations in the salts are considered to have low toxicity (NICNAS, 2013).

Import, Manufacture and Use

Australian

No specific Australian use, import, or manufacturing information has been identified.

International

The following international uses have been identified through the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers; Galleria Chemica; the Substances in Preparations in Nordic countries (SPIN) database; the United States (US) Personal Care Products Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary; the US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR); the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); Environment Canada Domestic Substances List (DSL); and various material safety data sheets (MSDSs).

Sodium iodate (CAS No. 7681-55-2) has a reported cosmetic use as an oxidising agent; and domestic use as a disinfectant. Information submitted to the Food and Drug Administration (FDA) in 1993 indicated that sodium iodate was not contained in any cosmetic formulations (CIR, 1995). The absence of use in cosmetic products was confirmed in 2011 (Personal Care Products Council, 2011). Available North American databases do not give evidence for use of this chemical in consumer products, indicating that the chemical is not likely to be widely available for domestic uses.

Potassium iodate (CAS No. 7758-05-6) has a site-limited use as an industrial chemical during formulation and end-use stages.

Iodic acid (CAS No. 7782-68-5) has a commercial use as a component in stencil remover.

Calcium iodate (CAS No. 7789-80-2) has a site-limited use as an industrial chemical during the end-use stage.

The chemicals in this group have non-industrial uses as food additives and in the pharmaceutical industry.

Restrictions

Australian

No known restrictions have been identified.

International

No known restrictions have been identified.

Existing Worker Health and Safety Controls

Hazard Classification

The chemicals are not listed on the Hazardous Chemical Information System (HCIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

The following exposure standards are identified (Galleria Chemica):

US Department of Energy Temporary Emergency Exposure Limits (TEELs):

Sodium iodate:

TEEL-1: 0.83 mg/m^3

TEEL-2: 9.1 mg/m^3

TEEL-3: 55 mg/m^3

Potassium iodate:

TEEL-1: 0.45 mg/m^3

TEEL-2: 4.9 mg/m^3

TEEL-3: 29 mg/m^3

Iodic acid:

TEEL-1: 0.15 mg/m^3

TEEL-2: 1.7 mg/m^3

TEEL-3: 10 mg/m^3

Magnesium diiodate:

TEEL-1: 30 mg/m^3

TEEL-2: 330 mg/m^3

TEEL-3: 2000 mg/m^3

Potassium hydrogen diiodate:

TEEL-1: 1.1 mg/m^3

TEEL-2: 13 mg/m^3

TEEL-3: 76 mg/m^3

Health Hazard Information

The chemicals are reduced to iodides following absorption (see **Toxicokinetics** section). In the absence of study information, data from iodide salts (CAS Nos. 7681-11-0 and 7681-82-5) and iodine will be considered applicable to evaluate potential systemic effects of these chemicals. Both potassium and sodium iodide are assessed by NICNAS as chemicals not considered to pose an unreasonable risk to the health of workers and public health under the relevant conditions of use (NICNASa).

Toxicokinetics

The toxicokinetics of orally absorbed iodate is similar to that of inorganic iodine, as the chemicals are both reduced to iodide and almost completely absorbed by the small intestine and released into the bloodstream (Chapman and Maloof, 1955; WHO, 2009; REACHb).

Iodide has been detected in the urine and tissues in rabbits injected intraperitoneally with iodate, and in dogs fed with potassium iodate in the form of gelatin capsules (REACHa; REACHb). Inhaled sodium iodide aerosols were reported to be rapidly absorbed in mice and sheep, and it was also determined that, for comparable air concentrations, dermal absorption was 1–2 % of the inhalation uptake (WHO, 2009).

In rabbits injected intraperitoneally with iodate, radioiodine was reported to be extensively distributed and found in the liver, kidney, brain, heart, muscle, small intestine, stomach, testes, submaxillary gland, skin, hair and thyroid. Tissue distribution was the same for both iodine and iodate (REACHa; REACHb).

The distribution of absorbed iodide is considered similar across all routes of exposure. The thyroid actively absorbs iodide from the blood to make thyroid hormones. Iodide is eventually excreted from the human body primarily via urinary excretion (>97 %), with faecal excretion accounting for another 1–2 % (WHO, 2009).

Acute Toxicity

Oral

The available data indicate that sodium and potassium iodate have moderate acute toxicity based on results from animal tests following oral exposure, warranting hazard classification (see **Recommendation** section). Given the chemical and toxicological similarities of these chemicals, in the absence of more comprehensive information, the chemicals in this group are expected to share similar acute effects.

The acute oral toxicities of sodium and potassium iodate were examined in female Swiss mice. Sodium and potassium iodate (6 % solution in water) were administered by gavage. Three study groups of mice were included for potassium iodate: mice fasted on wire screens overnight (n=110, dosage 316–891 mg/kg bw); mice fasted on sawdust overnight (n=140, dosage 47–1240 mg/kg bw); and mice not fasted (n=90, dosage 737–1750 mg/kg bw). For sodium iodate, only mice fasted on wire screens (n=140, dosage 261–824 mg/kg bw) were used for the study. The study reported the following median lethal dose (LD50) values for potassium iodate: mice fasted on wire screens–531 mg/kg bw; mice fasted on sawdust–815 mg/kg bw; and mice not fasted–1177 mg/kg bw. The LD50 value for sodium iodate in the mice fasted on wire screens group was 505 mg/kg bw (Webster et al., 1956). Sublethal effects from potassium iodate ingestion included diarrhoea, alternate hyperactivity and lassitude, followed by weakness, prostration and dyspnoea (CIR, 1995; REACHb).

Potassium iodate has a reported oral lowest published lethal dose (LDLo) value of 200 mg/kg bw in dogs (Galleria Chemica), and a reported oral LDLo value of 400 mg/kg bw in guinea pigs (Sigma-Aldrich MSDS). However, supplemental information for deriving these values are not available.

Dermal

No data are available.

Inhalation

No data are available.

Observation in humans

There have been a few reports of retinal damage in humans following oral exposure to iodates (REACHb). This is consistent with retinotoxic effects observed in rats and rabbits dosed by intravenous injection (CIR 1995; Burgi et al., 2001). Classification is considered warranted (see **Recommendation** section).

Corrosion / Irritation

Corrosivity

Iodic acid is a strong acid with a low pKa (0.75–0.78), which warrants hazard classification. Potassium hydrogen diiodate (which is effectively a mixture of iodic acid and potassium iodate) should also be considered corrosive on the grounds of pKa.

No reliable data are available for the other salts. Given that iodic acid is a monoprotic acid, corrosive effects for these simple salts are not expected on the basis of pH. Therefore, this classification should not apply to the other iodates in this group. However, oxidants may be irritant or corrosive depending on their reactivity.

Sensitisation

Skin Sensitisation

No data are available.

Repeated Dose Toxicity

Oral

Based on the available data, repeated oral exposure to the chemicals is not considered to cause serious damage to health. The target organs at higher doses include the thyroid, kidney and retina.

In a non-guideline study, Swiss mice were exposed to potassium iodate in drinking water on a daily basis at dose concentrations of 0, 0.05, 0.10, 0.25, 0.50 or 0.75 % per day for 104 days. Increased in body weight was observed in all dose groups at the end of the study. Effects in the blood and deposition of haemosiderin in the kidney were observed at dose groups of 0.25 % and higher (REACHa; REACHb). Similar effects were observed in a four week drinking water study in mice (Burgi et al., 2001). This is a strong evidence of increased haemolysis due to the iodate treatment (REACHa). Mice seemed to be more sensitive to effects compared with guinea pigs (Burgi et al., 2001).

In a 90 day non-guideline drinking water study, female Wistar rats were exposed to 3000, 6000, 12,000, 24,000, 48,000, 96,000 or 192,000 µg/L of potassium iodate. The NOAEL was reported to be 3000 µg/L. Significant increase in total cholesterol and number of white blood cells were observed in groups dosed with ≥6000 µg/L of the chemical. At higher doses, lower thyroxine level in serum and retinal dysfunction were observed. However, the actual doses given to the rats per day and per treatment were unspecified (REACHb).

In a 100 day non-guideline drinking study in mice (species unspecified), increased thyroid weight and effects on the thyroid follicles were observed at concentrations of 250 µg/L potassium iodate and above (WHO, 2009).

In dogs exposed orally for 66–192 days with doses of 6–100 mg/kg bw/day of iodate, there was some evidence of gastric toxicity and other minor abnormalities (indicative of haemolysis). Retinal damage was not observed (Burgi et al., 2001).

Dermal

No data are available.

Inhalation

No data are available.

Observation in humans

Iodate salts may be used as dietary supplements to prevent iodine deficiency, and are rapidly metabolised and absorbed as iodide (see **Toxicokinetics** section). Excessive intake of iodide has been associated with both hyperthyroidism and hypothyroidism (WHO, 2009). The recommended daily intake of iodine in Australia is 90–110 µg/day for infants less than one year old, 90–150 µg/day for infants and children (1–18 years), and 150 µg/day for adults. The upper intake level (UL) is 200–900 µg/day in children and adolescents depending on age, and 1100 µg/day in adults. These ULs are not considered as thresholds for toxicity, but rather intake limits that provide comfortable margins of safety for the Australian population. In young children that are estimated to exceed the UL for iodine, the overall potential for adverse effects is considered low, with small additional intakes are unlikely to represent a health and safety risk to young children (FSANZ, 2007).

Genotoxicity

The limited data do not indicate that the chemicals are genotoxic. If further data becomes available, this hazard endpoint can be revised.

Sodium iodate was negative in a bacterial reverse mutation assay using *Salmonella typhimurium* strains TA 100 and TA 98. The chemical was reported to exhibit anti-mutagenic activity towards Aflatoxin B1 when tested using strain TA 100 (REACHb).

A comet assay in Chinese hamster ovary (CHO) cultures did not show the presence of DNA damage after a treatment of cells by potassium iodate at concentrations of up to 10 mM. This absence of primary DNA damage was confirmed in the cytokinesis-block micronucleus assay (REACHb).

The chemical did not increase the frequencies of micronuclei in mouse bone marrow cells or in recessive lethal mutations in *Drosophila melanogaster* (CIR, 1995; Burgi et al., 2001; WHO, 2009).

Whilst bromate and other halogenates have been found to be genotoxic in a variety of assays (NICNASb), iodate has a lower oxidative potential than bromate, and it did not induce the formation of oxidised bases in DNA (Burgi et al., 2001). In contract to bromate, iodate was not reported to produce oxidative damage to calf thymus DNA as measured by the appearance of 8-hydroxy-deoxyguanosine. Similar to bromate; however, iodate induced DNA strand breakage in the rat epithelial kidney cell comet assay. For iodate this effect was apparent at the first time

point measured (i.e. 15 mins after the start of the in vitro treatment). The DNA strand breakage then decreased at 4 hours and remained at that level at 24 hours. The DNA breakage induced by bromate showed a second activity peak at 24 hours (Burgi et al., 2001).

Carcinogenicity

No data are available. Whilst bromate salts are carcinogenic (NICNASb), there are notable differences in the interaction of iodate with DNA compared to bromate (Burgi et al., 2001).

Reproductive and Developmental Toxicity

Limited data are available for iodates and the potential metabolite iodide. The available information indicates that any reproductive and developmental effects would be secondary to maternal toxicity.

Sodium iodate at 1 mg/kg bw given twice weekly to pregnant dams and to offspring over 4 months was reported to have no toxic effect in the rabbit; however, doses that were retinotoxic to the rabbit dam may also cause irreversible damage to the retina of offspring (Burgi et al., 2001).

Potassium iodide was fed to male and female Sprague Dawley (SD) rats for 14 days before mating and for I–14 days during breeding, and to females only during gestation (22 days) and lactation (21 days). After weaning, the offspring were given dietary potassium iodide, at the same level as the parents, for the remainder of the study (up to 90 days of age for most offspring). The dose levels were 0, 0.025, 0.05 or 0.1 % (w/w) in the diet. The results indicate that potassium iodide at dietary levels of up to 0.1 % of the diet (approximately 90 mg/kg bw/day), produced only minor effects on parental weight gain and food consumption, and no significant effects on parental mortality, fertility, pregnancy maintenance, or gestation length. Potassium iodide was reported to significantly increase the proportion of litters born with less than eight live offspring at the highest dose (0.1 %). This dose group also showed significant reductions in mean litter size as compared to the negative control group. However, birth weights and external morphology among those born alive were not significantly altered. Postnatal treatment of potassium iodide resulted in a significant reduction in offspring body weight in the two highest doses (0.05 and 0.1 %) at days 42 and 90.

In addition, the halogenate, perchlorate, did not cause specific reproductive or developmental toxicity. Any reproductive and developmental effects were only observed secondary to maternal toxicity (NICNASc).

Oral exposure to excess stable iodine may also cause disruption of reproductive function secondary to thyroid gland dysfunction (ATSDR, 2004).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation includes local effect (corrosivity) and harmful systemic effects following a single oral exposure. Exposure to single doses could cause effects to the retina.

The chemicals are metabolised to iodide. Excessive intake of iodine has been associated with both hyperthyroidism and hypothyroidism; however, the expected small additional exposures from use of these chemicals are unlikely to represent a risk.

Public Risk Characterisation

Given the uses identified for these chemicals, it is unlikely that the public will be significantly exposed. Hence, the public risk from these chemicals is not considered to be unreasonable.

Occupational Risk Characterisation

During product formulation, dermal, ocular and inhalation exposure might occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemicals at lower concentrations could also occur while using formulated products containing the chemicals. The level and route of exposure will vary depending on the method of application and work practices employed.

Oral exposure is also possible but can be prevented by good hygiene practices.

Given the critical systemic acute and local health effects, the chemicals could pose an unreasonable risk to workers unless adequate control measures to minimise dermal, ocular and inhalation exposure are implemented. The chemicals should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine the appropriate controls.

The data available support an amendment to the hazard classification in the Hazardous Chemical Information System (HCIS) (Safe Work Australia) (see **Recommendation** section).

NICNAS Recommendation

The assessment of these chemicals is considered to be sufficient, provided that the recommended amendment to the classification is adopted, and labelling and all other requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory.

Regulatory Control

Public Health

Products containing the chemicals should be labelled in accordance with state and territory legislation (SUSMP, 2017).

Work Health and Safety

These chemicals are recommended for classification and labelling aligned with the Globally Harmonized System of Classification and Labelling of Chemicals (GHS) as below. This does not consider classification of physical hazards and environmental hazards.

The corrosivity classification applies to iodic acid (7782-68-5) and potassium hydrogen diiodate (13455-24-8) only.

From 1 January 2017, under the model Work Health and Safety Regulations, chemicals are no longer to be classified under the Approved Criteria for Classifying Hazardous Substances system.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Not Applicable	Harmful if swallowed - Cat. 4 (H302)
Irritation / Corrosivity	Not Applicable	Causes severe skin burns and eye damage - Cat. 1 (H314)
Other Health Effects	Not Applicable	May cause damage to organs - Specific target organ tox, single exp Cat. 2 (H371)

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

[*] Existing Hazard Classification. No change recommended to this classification

Advice for consumers

Products containing the chemicals should be used according to the instructions on the label.

Advice for industry

Control measures

Control measures to minimise the risk from oral, dermal, ocular, and inhalation exposure to the chemicals should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemicals are used. Examples of control measures that could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemicals from entering the breathing zone of any worker;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemicals.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemicals are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of these chemicals has not been undertaken as part of this assessment. According to the classification provided by companies to ECHA in REACH registrations this substance may intensify fire (oxidiser).

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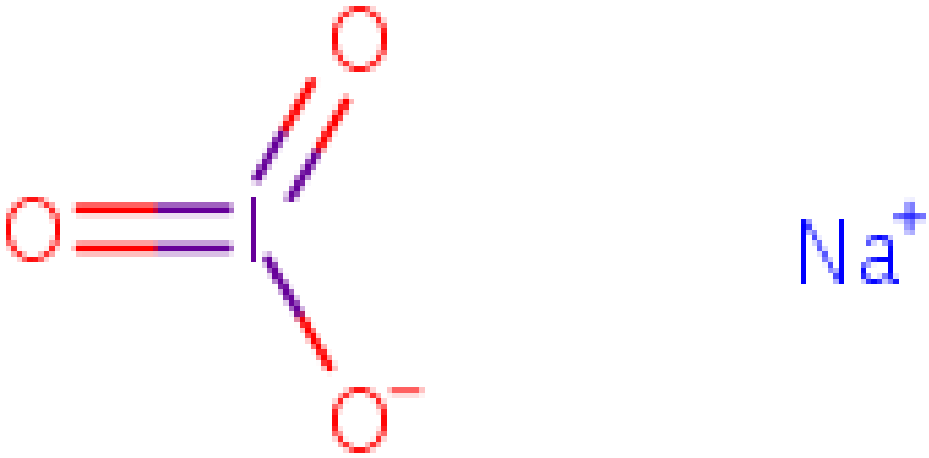
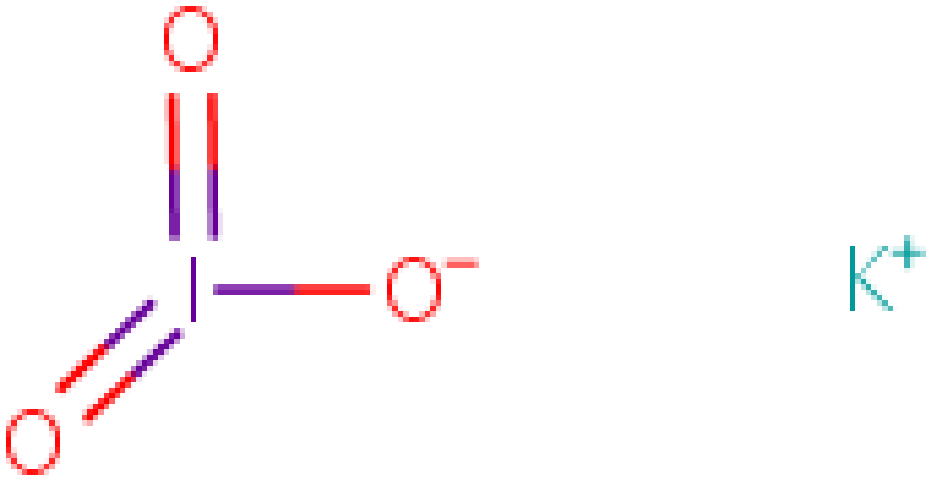
Last Update 27 October 2017

Chemical Identities

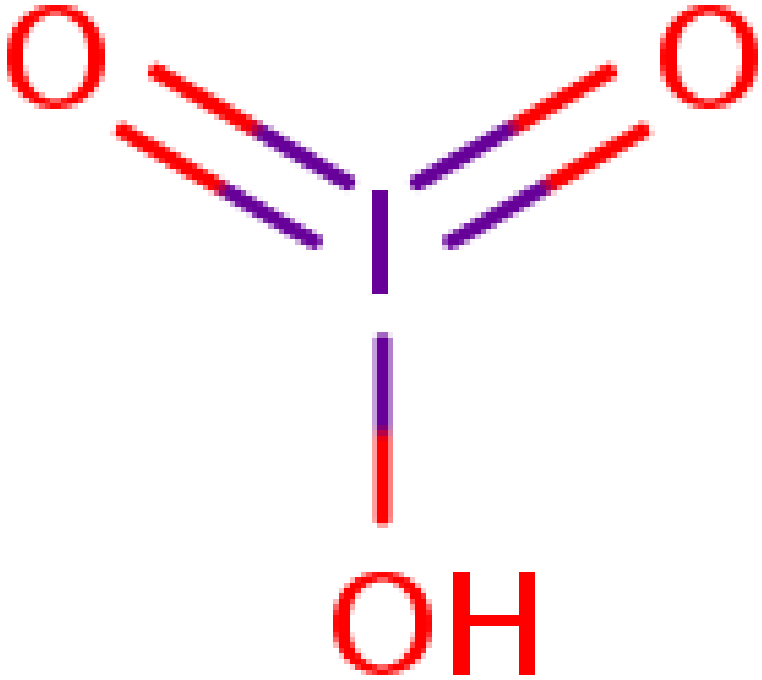
Chemical Name in the Inventory and Synonyms	**Iodic acid (HIO3), sodium salt** sodium iodate
CAS Number	7681-55-2
Structural Formula	

	
Molecular Formula	HIO3.Na
Molecular Weight	197.89

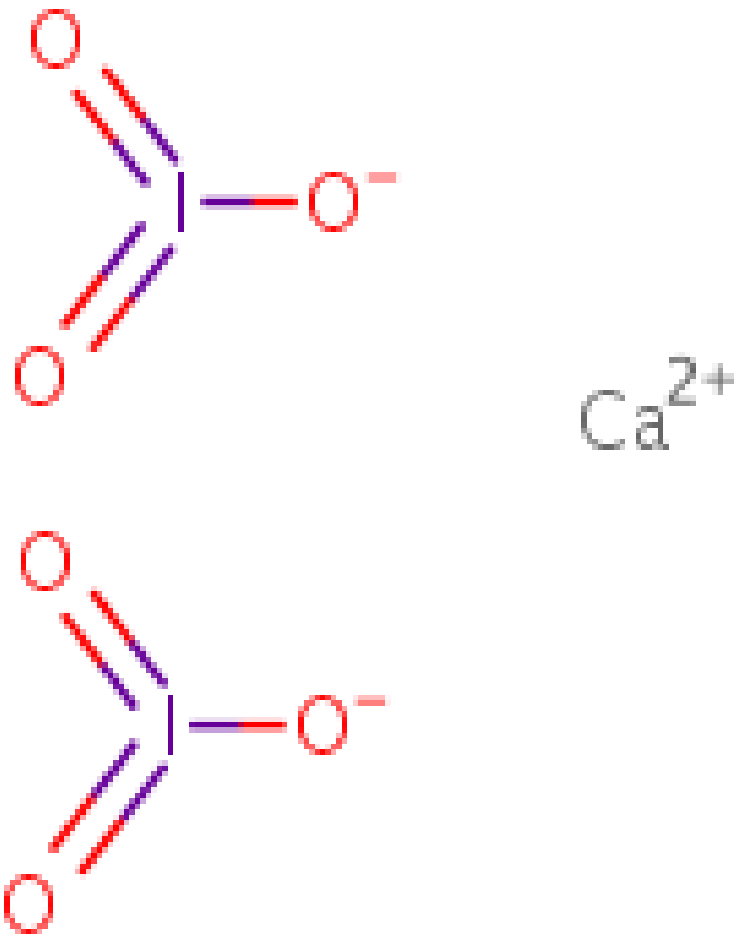
Chemical Name in the Inventory and Synonyms	**Iodic acid (HIO3), potassium salt** potassium iodate
CAS Number	7758-05-6
Structural Formula	

	
Molecular Formula	HIO3.K
Molecular Weight	214.00

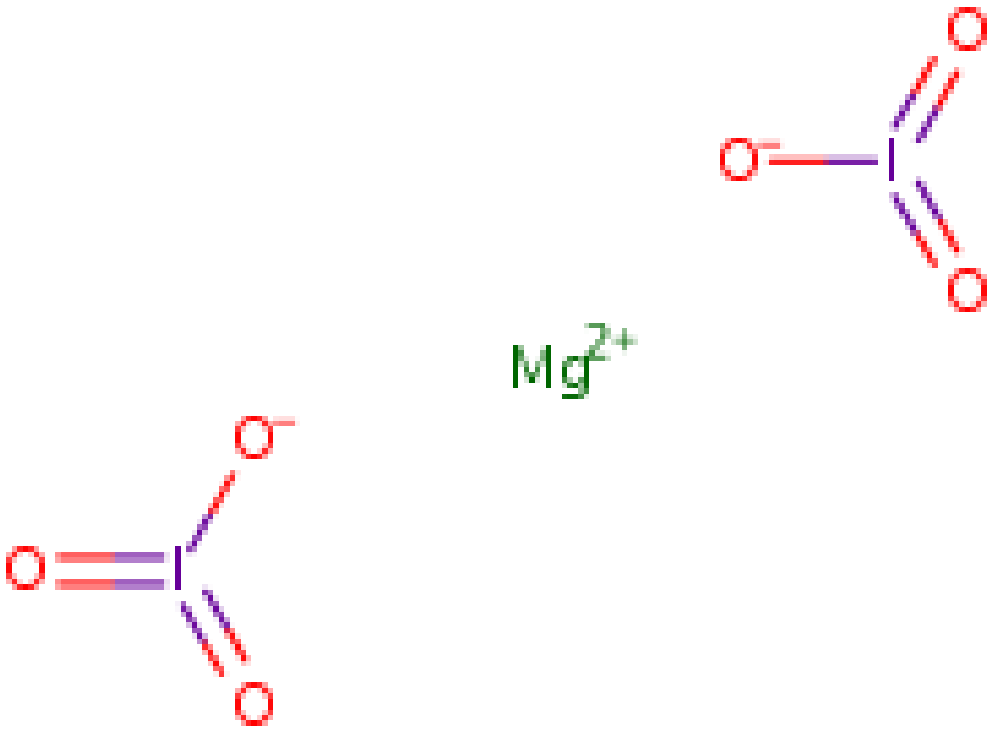
Chemical Name in the Inventory and Synonyms	**Iodic acid (HIO3)**
CAS Number	7782-68-5
Structural Formula	

	
Molecular Formula	HIO3
Molecular Weight	175.91

Chemical Name in the Inventory and Synonyms	**Iodic acid (HIO3), calcium salt** calcium iodate
CAS Number	7789-80-2
Structural Formula	

	
Molecular Formula	Ca.2HIO3
Molecular Weight	389.87

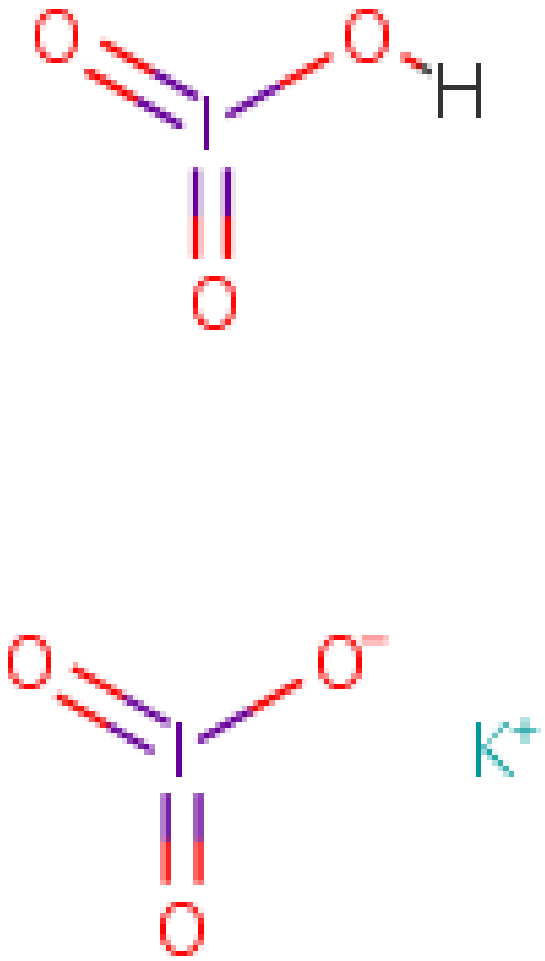
Chemical Name in the Inventory and Synonyms	**Iodic acid (HIO3), magnesium salt** magnesium diiodate
CAS Number	7790-32-1
Structural Formula	



Molecular Formula	HIO3.1/2Mg
Molecular Weight	374.10

Chemical Name in the Inventory and Synonyms	**Iodic acid (HIO3), calcium salt, hexahydrate** calcium iodate hexahydrate
CAS Number	10031-33-1
Structural Formula	**No Structural Diagram Available**

Molecular Formula	Ca.6H2O.2HIO3
Molecular Weight	497.98

Chemical Name in the Inventory and Synonyms	**Iodic acid (HIO3), potassium salt (2:1)** potassium hydrogen diiodate
CAS Number	13455-24-8
Structural Formula	
Molecular Formula	HIO3.1/2K
Molecular Weight	390.00

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