Periodic acid and its salts: Human health tier II assessment

27 October 2017

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Periodic acid (HIO4), potassium salt	7790-21-8
Periodic acid (HIO4), sodium salt	7790-28-5
Periodic acid (H5IO6)	10450-60-9
Periodic acid (HIO4)	13444-71-8
Periodic acid (H5IO6), trisodium salt	13940-38-0

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

The chemicals in this group are orthoperiodic (CAS No. 10450-60-9) and metaperiodic (CAS No. 13444-71-8) forms of periodic acid, and their respective salts: sodium paraperiodate (CAS No. 13940-38-0), potassium metaperiodate (CAS No. 7790-21-8) and sodium metaperiodate (CAS No. 7790-28-5). Orthoperiodic acid can be dehydrated to metaperiodic acid by heating.

These chemicals share similar physicochemical properties and reported uses, and have been shown to have similar systemic toxicological profiles (see **Hazards**); therefore, the chemicals are qualified to be assessed as a group. The cations 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; United States (US) Environmental Protection Agency (EPA); Environment Canada Domestic Substances List (DSL); and various material safety data sheets (MSDSs):

Orthoperiodic acid (H_5IO_6) (CAS No. 10450-60-9) has reported commercial uses as:

- a component in stencil remover; and
- a reclamation agent in the screen printing industry.

Sodium paraperiodate (CAS No. 13940-38-0) has a reported commercial use as an additive to paper and cigarettes.

Metaperiodic acid (HIO_4) (CAS No. 13444-71-8) has a reported commercial use as a component in emulsion removers for screen reclamation.

Potassium metaperiodate (CAS No. 7790-21-8) has a reported site-limited use as a component in silica-based slurries for polishing of ruthenium and copper surface.

Sodium metaperiodate (CAS No. 7790-28-5) has a potential domestic use as a component in bleach. Available North American databases do not provide evidence for use of this chemical in consumer products, indicating that the chemical is not likely to be widely available for domestic uses. The chemical also has reported commercial uses as:

- a component in textile treatment products, dyes, washing and cleaning products, biocides (e.g. disinfectants, pest control products), coating products, non-metal-surface treatment products and inks and toners; and
- a component in emulsion removers for screen reclamation.

The chemicals in this group share the following reported site-limited uses:

- as oxidising agents used in industry settings and military pyrotechnic devices; and
- as intermediates for the manufacture of other substances.

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):

Orthoperiodic acid:

TEEL-1: 0.18 mg/m^3

TEEL-2: 2.1 mg/m^3

TEEL-3: 12 mg/m^3

Potassium metaperiodate:

TEEL-1: 1.2 mg/m^3

TEEL-2: 13 mg/m^3

TEEL-3: 79 mg/m^3

Sodium metaperiodate:

TEEL-1: 0.19 mg/m^3

TEEL-2: 2.1 mg/m^3

TEEL-3: 13 mg/m^3

No exposure standards are available for sodium paraperiodate and metaperiodic acid.

Health Hazard Information

Periodic acid and periodates exist as solid crystals under normal conditions, with the melting points of all chemicals being above 100 °C. In commercial and domestic settings, these chemicals are mainly available as components of liquid products.

In the absence of data, the information for iodate and iodide salts (CAS Nos. 7758-05-6, 7681-11-0, 7681-82-5, 7681-55-2) will be considered applicable to evaluate potential systemic effects of these chemicals due to the reduction of periodates in biological systems (see **Toxicokinetics**). Both potassium and sodium iodide were assessed by NICNAS as chemicals not considered to pose an unreasonable risk to the health of workers and public health under these relevant conditions of use (NICNASa).

Toxicokinetics

Limited data are available for sodium and potassium metaperiodate. No data are available for sodium paraperiodate, or for meta- and orthoperiodic acids. Based on the available data, the chemicals are expected to be metabolised to iodates and iodides. Iodides are extensively distributed in the body. The thyroid actively absorbs iodine from the blood to make and release thyroid hormones back into the blood (WHO, 2009; NICNASb).

Two hydrolysis studies based on the Organisation for Economic Co-operation and Development (OECD) Test Guideline (TG) 111 using sodium metaperiodate are available.

In the first gastric hydrolysis study, sodium metaperiodate was dissolved in either fasted state simulated gastric fluid (FaSSGF), or fed state simulated gastric fluid (FeSSGF), in the dark at body temperature. The quantities and nature of the decomposition products were investigated. Sodium metaperiodate was reported to be quickly degraded in both forms of stimulated gastric fluids (SGFs). The half-lives of sodium metaperiodate in both SGFs were reported to be less than 1 hour at 38.0 ± 0.5°C. The study group concluded that the periodate ion in both solutions were significantly reduced, potentially all of the way down to iodide (REACHa).

In a second gastric hydrolysis study, sodium metaperiodate was dissolved in either FaSSGF or FeSSGF at body temperature. The concentration of sodium metaperiodate and known decomposition products were determined at 0.5, 1, 2, 4, 6 and 24 hours. The procedure was repeated using the reference substance, potassium iodate, with incubation periods of 1, 2, 4 and 6 hours. Periodate (IO^{4-}) was confirmed to undergo a chemical reaction to form iodate (IO^{3-}) in the test condition (REACHa).

In an in vivo study, ^{131}I-labelled potassium metaperiodate (10 µC) dissolved in 0.1 mL distilled water was intravenously injected into adult Wistar rats (n=30; 18 males and 12 females). Groups of five animals (three males and two females) were sacrificed at different intervals (from 1 hour to 15 days) and the radioactivity in organs and tissues was determined with a scintillation counter. The study reported that metaperiodate seemed to be reduced to iodate as soon as it was injected (Anghileri, 1965).

Acute Toxicity

Oral

Based on the limited data available, the periodate salts have moderate acute toxicity based on results from animal tests following oral exposure. 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, except to the extent acute toxicity results from local acidity-related effects.

The acute toxicity of sodium periodate and potassium periodate was assessed using the sequential stage-wise probit (SSWP) method. The following clinical signs were observed in rats administered potassium metaperiodate (560 mg/kg bw and greater) and sodium metaperiodate (175 mg/kg bw and greater): lethargy, rough coat, laboured breathing, prostration, squinting, dark eyes, hunched posture, chromodacryorrhoea, diarrhoea, bloody urine, and red discharge from nose. Gross pathology included effects in the kidney and stomach (Lent et al., 2017).

The median lethal doses (LD50s) for potassium metaperiodate were 732 mg/kg bw and 685 mg/kg bw for females and males, respectively. The LD50s for sodium metaperiodate were 318 mg/kg bw and 741 mg/kg bw for females and males, respectively (Lent et al., 2017).

Sodium metaperiodate also has a reported LD50 of 264 mg/kg bw for acute oral toxicity from various MSDSs (Ricca Chemical Company, 2015; Spectrum Chemical, 2015); however, supplemental information for deriving this value is currently not available.

Iodates have moderate acute toxicity by the oral route (NICNASb). Effects are likely related to the corrosive properties of the chemical rather than systemic toxicity.

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 (the expected metabolite) (NICNASb). Whilst effects in humans have not been reported for periodates, sodium periodate injected intravenously into a rabbit (single dose of 40 mg/kg bw) resulted in retinal damages that were similar to those produced by iodate. Effects included oedema below the disk, discrete pigmentary changes in a broad zone below the disk, and extensive degeneration of the rods with marked proliferative changes in the pigment epithelium (Sorsby and Harding, 1962). Therefore, classification is considered warranted (see **Recommendation** section).

Corrosion / Irritation

Corrosivity

Orthoperiodic acid has a number of acid dissociation constants. In its original form (H_5IO_6), it has a pKa of 3.29 (Burgot, 2012). The acids of this group are expected to be particularly corrosive considering the combination of oxidising potential and acidity.

Based on an available study, classification for this corrosivity is also warranted for sodium metaperiodate. In the absence of more comprehensive information, the recommended classification will also be applicable for the members of this group due to the similar toxicological and chemical profiles of these chemicals. The chemicals are strong oxidising agents which will contribute to their corrosive effects.

The corrosivity of sodium metaperiodate was investigated in accordance with the OECD TG 431. Reconstructed human epidermis was treated with the chemical (20 mg) for exposure periods of 3, 60 and 240 mins. The relative mean viability of the tissues treated with the chemical were: 3 min exposure: 129.3 %; 60 min exposure: 55.7 %; and 240 min exposure: 12.9 %. Given that the chemical induced tissue viability of less than 35 % upon exposure for 240 min, the chemical was classified as corrosive to the skin (REACHa).

Sensitisation

Skin Sensitisation

No data are available.

Repeated Dose Toxicity

Oral

Available data are insufficient to support hazard classification. If further data becomes available, this hazard endpoint can be revised.

A 14 day oral subacute toxicity study was performed to determine the toxicity of sodium metaperiodate in SD rats (6/sex, approximately 10 weeks old). Test animals were given oral doses (via gavage) of sodium metaperiodate (suspended in deionised water) of 20, 40, 80, 159 and 318 mg/kg bw/day for females, and 46, 93, 185, 370 and 741 mg/kg bw/day for males. After 14 days, the rats were euthanised and tissues were histopathologically evaluated. Triiodothyronine and total thyroxine levels were also determined. The study reported that sodium metaperiodate did not produce any histopathological effects on the thyroid, and changes in thyroid hormones occurred only at high toxic doses (318 mg/kg bw/day (females), ≥370 mg/kg bw/day (males)) and did not include increases in thyroid-stimulating hormone (TSH). Clinical observations included lethargy, squinting, congested breathing, prostration, hunched posture, rough coat, bloody bedding in cage, bloody urination, dried red material on front paws, brown perianal staining, diarrhoea, red discharge from nose, and abnormal grooming behaviour in the female 318

mg/kg bw/day group and the male 185, 370, and 741 mg/kg bw/day groups. Pathology findings included damage in the gastrointestinal tract and stomach, red mesenteric lymph nodes, red outer rim of medulla of kidney, pale and/or dark kidney, mottled and/or dark liver, and fluid-filled cysts surrounding the adrenals. Effects observed were considered likely secondary to kidney toxicity and uremia (Lent et al., 2017).

Repeated oral exposure to iodates (the expected metabolites) are not considered to cause serious damage to health. Effects to the thyroid, kidney, blood and retina have been observed at high doses (NICNASb). Effects in the thyroid at high doses have been observed in multiple studies in animals exposed to iodine or iodide (WHO, 2009).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

No data are available. The limited data available for the potential metabolite iodate and other perhalogenates, does not indicate that the chemicals in this group are genotoxic.

Several tests, including alkaline comet assay, lymphoma assay and micronucleus assay, indicate that iodate salts do not pose significant mutagenic or cell transforming potential. Iodate did not produce oxidative damage to calf thymus DNA but did induce DNA strand breakage in the rat epithelial kidney cell comet assay (NICNASb).

The perhalogenate, perchlorate, gave negative results in several in vitro tests and was negative in bone marrow erythrocyte micronucleus test in rats and mice (NICNASc).

Carcinogenicity

No data are available. Other halogenates are carcinogenic, with bromate salts causing tumours in the kidney and thyroid and perchlorate salts causing tumours in the thyroid (NICNASc; NICNASd). However, there are notable differences in the interaction of periodate with DNA compared to bromate (NICNASc). Perchlorates cause rodent tumours by a specific mechanism (inhibition of iodide uptake). While limited data are available, periodates seem to be less toxic to the thyroid than perchlorates and did not induce increased levels of TSH in rats (Lent, 2017).

Reproductive and Developmental Toxicity

No data are available for these chemicals, and limited data are available for the possible metabolites, iodates and iodides. 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. Doses retinotoxic to the rabbit dam; however, may cause irreversible damage the retina of offspring (NICNASb).

Potassium iodide was fed to male and female Sprague Dawley (SD) rats for 14 days before mating and for l–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, until up 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 indicated 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 dose groups (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 effects (corrosivity) and harmful systemic effects following a single oral exposure. There are limited data available on the chronic systemic toxicity of the chemicals. Both the periodate and iodate ions (NICNASb) are strong oxidants and are not expected to persist in the systemic circulation.

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 controls expected to be in place due to the corrosivity classification are expected to be sufficient to protect workers from any potential chronic systemic toxicity effects.

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

Work Health and Safety

The 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.

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 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 the chemicals may cause fire or explosion (strong oxidiser).

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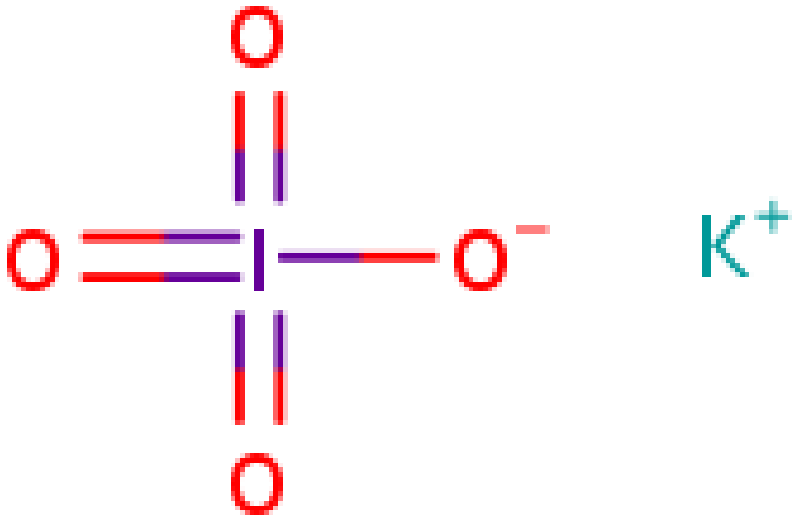
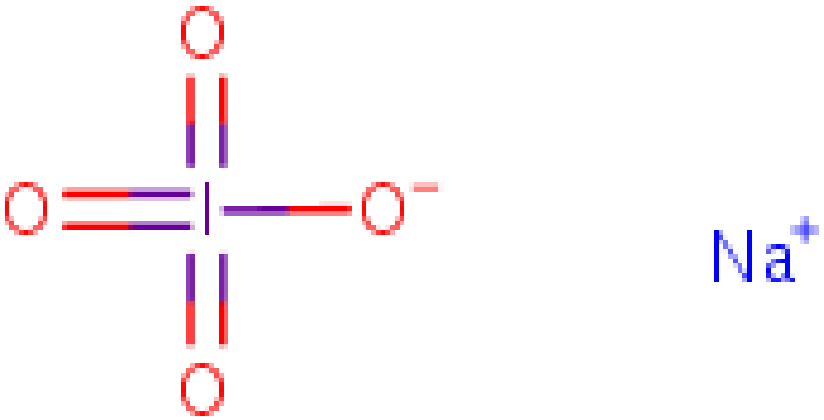
Last Update 27 October 2017

Chemical Identities

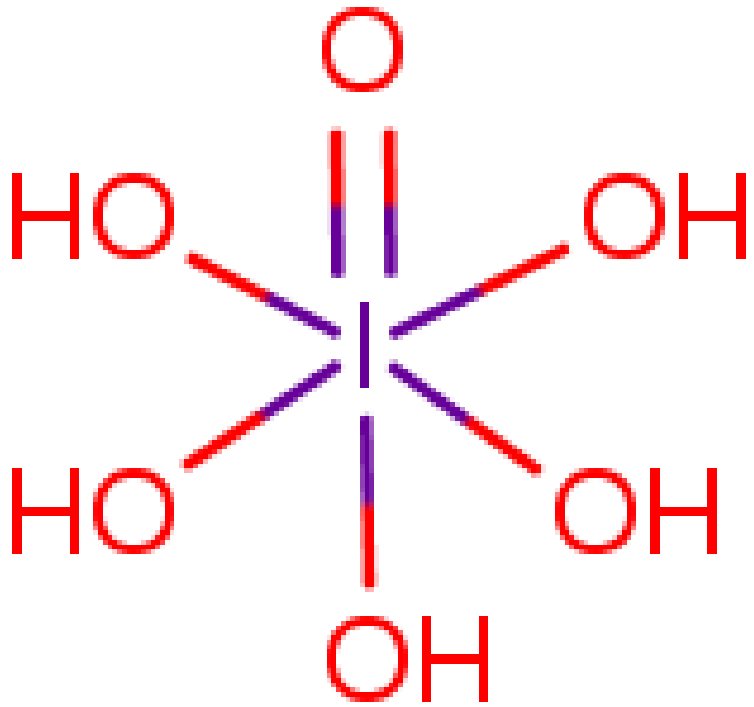
Chemical Name in the Inventory and Synonyms	**Periodic acid (HIO4), potassium salt** potassium metaperiodate
CAS Number	7790-21-8
Structural Formula	

	
Molecular Formula	HIO4.K
Molecular Weight	229.99

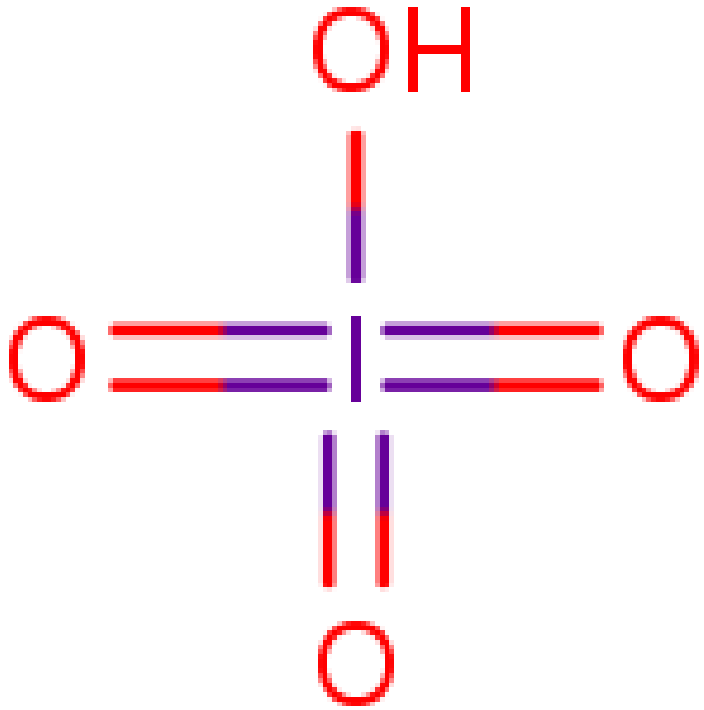
Chemical Name in the Inventory and Synonyms	**Periodic acid (HIO4), sodium salt** sodium metaperiodate
CAS Number	7790-28-5
Structural Formula	

	
Molecular Formula	HIO4.Na
Molecular Weight	213.89

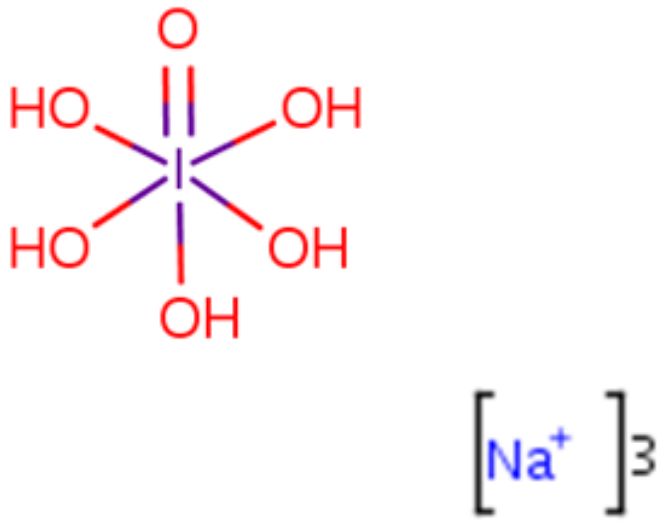
Chemical Name in the Inventory and Synonyms	**Periodic acid (H5IO6)** orthoperiodic acid
CAS Number	10450-60-9
Structural Formula	

	
Molecular Formula	H5IO6
Molecular Weight	227.93

Chemical Name in the Inventory and Synonyms	**Periodic acid (HIO4)** metaperiodic acid
CAS Number	13444-71-8
Structural Formula	

	
Molecular Formula	HIO4
Molecular Weight	191.90

Chemical Name in the Inventory and Synonyms	**Periodic acid (H5IO6), trisodium salt** sodium paraperiodate trisodium paraperiodate
CAS Number	13940-38-0
Structural Formula	

	
Molecular Formula	H5IO6.3Na
Molecular Weight	298.92

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