Bromates: Human health tier II assessment

03 July 2015

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Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Bromic acid, potassium salt	7758-01-2
Bromic acid, sodium salt	7789-38-0

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 toxicological effects of these chemicals are mediated primarily through the bromate ion. Potassium bromate (CAS No. 7758-01-2) and sodium bromate (CAS No. 7789-38-0) produce similar effects and these chemicals are also roughly equivalent in the delivery of bromate ions, given the high molecular weight of the bromate anions compared with the cations. Following dissociation in water, sodium (Na^+) and potassium (K^+) cations are released, which are naturally occurring species and do not contribute to toxicity. Therefore, these chemicals are grouped together for assessment (NTP, 2007; Health Canada, 2010; REACHb).

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, Authorization and Restriction of Chemicals (REACH) dossiers;
- Galleria Chemica;
- the Substances and Preparations in Nordic countries (SPIN) database;
- the European Commission Cosmetic Ingredients and Substances (CosIng) database; and

- the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemicals have reported cosmetic use as oxidising agents.

There is currently no documented use of the chemicals in this group in cosmetic products in the United States of America (Personal Care Products Council, 2011).

Sodium bromate (CAS No. 7789-38-0) has reported domestic uses including in:

- cleaning/washing agents;
- surface treatments; and
- paints, lacquers and varnishes.

The US Household Products Database did not indicate any cosmetic or domestic use of these chemicals.

Potassium bromate (CAS No. 7758-01-2) has reported commercial uses including in:

- packaging material; and
- industrial cleaning agent.

These chemicals have reported site-limited uses including in complexing and flocculating agents.

These chemicals have reported non-industrial uses internationally as:

- non-agricultural pesticides and preservatives; and
- an agent for malting of barley.

Restrictions

Australian

These chemicals are listed in the *Poisons standard—the Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP) in Schedule 6 (SUSMP, 2015) as follows:

'POTASSIUM BROMATE **except** in preparations containing 0.5 per cent or less of potassium bromate'.

'SODIUM BROMATE **except** in preparations containing 0.5 per cent or less of sodium bromate'.

Schedule 6 chemicals are described as 'Substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label'.

International

Potassium bromate (CAS No. 7758-01-2) is listed on the following (Galleria Chemica):

- EU Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain - Table 1;
- The Association of Southeast Asian Nations (ASEAN) Cosmetic Directive Annex II Part 1: List of substances which must not form part of the composition of cosmetic products; and

- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient 'Hotlist').

Potassium bromate (CAS No. 7758-01-2) is also restricted by Annex XVII to the REACH Regulations (Appendix 2). The chemical cannot be used in substances and preparations placed on the market for sale to the general public in individual concentrations ≥0.5 %.

Sodium bromate (CAS No. 7789-38-0) is listed on the following (Galleria Chemica):

- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient 'Hotlist').

Existing Worker Health and Safety Controls

Hazard Classification

Potassium bromate (CAS No. 7758-01-2) is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

- T; R25 (acute toxicity)
- T; R45 Carc. Cat 2 (carcinogenicity)

Exposure Standards

Australian

No specific exposure standards are available.

International

Potassium bromate (CAS No. 7758-01-2) has a Workplace Environmental Exposure Level (WEEL) of 0.1 mg/m^3 time weighted average (TWA) in the United States of America (USA).

Health Hazard Information

The toxicological effects of these chemicals are mediated primarily through the bromate ion. Potassium bromate and sodium bromate produce similar effects and are also roughly equivalent in the delivery of bromate ions. Therefore, in the absence of information on sodium bromate, data from potassium bromate has been used (see **Grouping Ratioanle**) (NTP, 2007; Health Canada, 2010).

Toxicokinetics

These chemicals dissociate in water and bromate ion is rapidly absorbed from the gastrointestinal tract, at least in part unchanged. It is distributed throughout the body appearing in plasma and urine unchanged and in other tissues as bromide. Bromate is reduced to bromide in several body tissues, probably by GSH or other sulfhydryl-containing compounds. Most bromate is excreted in the urine, either as bromate or bromide, but some may leave the body in the faeces. Bromine has been detected in adipose tissue of mice following long-term treatment with bromate (US EPA, 2001; REACHb).

Acute Toxicity

Oral

Potassium bromate is classified as hazardous with the risk phrase ‘Toxic if swallowed’ (T; R25) in the Hazardous Substances Information System (HSIS) (Safe Work Australia). The available data (median lethal dose—LD50—157 mg/kg bw) support this classification (REACHa).

Data are not available for sodium bromate. Considering that both chemicals will produce similar effects through bromate ions, a classification similar to the above is also recommended for sodium bromate (NTP, 2007; Health Canada, 2010; REACHb).

Dermal

No data are available.

Inhalation

No data are available.

Observation in humans

A number of cases of acute bromate toxicity have been reported in humans following accidental or intentional ingestion of permanent hair wave neutralising solution. These products usually contain either 2 % potassium bromate or 10 % sodium bromate. Bromate intoxication leads to gastrointestinal symptoms (abdominal pain, nausea, vomiting, diarrhoea), central nervous system depression, renal failure, and hearing loss. Although these effects are usually reversible, death from renal failure may ensue if medical intervention is not successful. Hearing loss is usually irreversible (US EPA, 2001; NTP, 2007; HSDB; REACHb).

Corrosion / Irritation

Skin Irritation

Although limited data are available, the available information indicates that these chemicals are not likely to be corrosive.

The purpose of the available study was to identify potential of potassium bromate for skin corrosion using an in vitro method. The study was conducted according to Organisation for Economic Co-operation and Development (OECD) Test Guideline (TG) 431, using a human skin model. The study consisted of a topical exposure of potassium bromate to a human reconstructed model followed by a cell viability test. Potassium bromate was not considered to possess a corrosive potential (REACHa).

Eye Irritation

Although limited data are available, the available information indicates that these chemicals are not likely to be eye irritants.

An eye irritation study was conducted according to OECD TG 437: Bovine Corneal Opacity and Permeability Test Method for Identifying Ocular Corrosives and Severe Irritants. In this test, the damage is assessed by quantitative measurements of changes in corneal opacity and permeability with an opacitometer and a visible light spectrophotometer, respectively. Potassium bromate caused weak opacity but no permeability of the cornea compared with the results of the negative control group. The chemical was considered to be a mild eye irritant (REACHa).

Sensitisation

Skin Sensitisation

The available data on potassium bromate indicate that these chemicals are not likely to be skin sensitisers.

In a skin sensitisation study conducted according to OECD TG 429 (local lymph node assay—LLNA), potassium bromate (CAS No. 7758-01-2) at 1.25 %, 2.5 %, and 7.5 % (w/v) concentration was applied topically at the dorsum of each ear of female CBA mice once daily on three consecutive days. A further group of mice was treated with the positive control item and a control group of mice was also treated with the vehicle only. Stimulation Indices (S.I.) of 0.90, 0.53, and 0.64 were determined with the test item at concentrations of 1.25, 2.5, and 7.5 % (w/v), respectively. The EC3 value could not be calculated, since none of the tested concentrations induced an S.I. of greater than three. Potassium bromate was not considered to be a skin sensitiser (REACHa).

Repeated Dose Toxicity

Oral

A number of repeated dose oral toxicity studies in animals indicate that the kidney is the major target organ of bromate-associated toxicity, leading to carcinogenicity. Specific non-cancer effects included degenerative, necrotic, nephropathic, and regenerative changes in the kidney (see **Carcinogenicity**) (US EAP, 2001; WHO, 2005; Health Canada, 2010).

In a repeated dose toxicity study, potassium bromate was administered in the drinking water at concentrations of 0, 150, 300, 600, 1250, 2500, 5000, or 10000 mg/L to male and female Fischer 344 (F344) rats (10/sex/group) for 13 weeks. All animals exposed to >1250 mg/L died within seven weeks. Significant inhibition of body weight gain was observed in males exposed to 600 or 1250 mg/L. Various-sized droplets and regenerative changes were observed in the renal tubules of treated males. A no observed adverse effect level (NOAEL) of 300 mg/L was determined (US EPA, 2001; NTP, 2007; REACHb).

In a chronic toxicity/carcinogenicity study, potassium bromate was administered at 0, 250, and 500 ppm concentrations to F344 rats (53/sex/group) for 110 weeks. Daily intake of potassium bromate was equivalent to 12.5 and 27.5 mg/kg bw/day in males and 12.5 and 25.5 mg/kg bw/day in females, respectively. As the growth of males in the high dose group was severely inhibited, the concentration in this group was reduced to 400 ppm at week 60. Body weight gain was significantly reduced in high-dose males, but not in the other treated groups. Survival was reduced in high-dose males by about week 60 and in low-dose males by about week 100. No effect on survival was observed in treated female rats. A variety of non-cancer effects were reported, including: degenerative, necrotic, and regenerative changes in renal tubules; formation of hyaline droplets; thickening of transitional epithelium of the renal pelvis; papillary hyperplasia; and papillary growth. It was noted that the lesions were more extensive in degree and distribution in treated rats compared with controls, especially males. However, in the absence of information on the incidence of these lesions or on the statistical significance of these findings, a NOAEL for non-cancer effects could not be determined (US EPA, 2001; Health Canada, 2010).

In another chronic study, potassium bromate was administered to male F344 rats and male B6C3F1 mice in drinking water at concentrations of 0, 0.02, 0.1, 0.2, and 0.4 g/L and 0, 0.08, 0.4, and 0.8 g/L, respectively, for 100 weeks. The doses were equal to 0, 1.5, 7.9, 16.9, and 37.5 mg/kg bw/day and 0, 9.1, 42.4, and 77.8 mg/kg bw/day, respectively, for rats and mice. In male rats, a statistically significant decrease in the mean body weight and survival was noted at the termination of the study at 0.4 g/L. The decrease in survival and body weight was attributed to an excessive mesothelioma burden. The effects on survival and body weight in rats indicate that the maximum tolerated dose (MTD) was reached in this study. A significant dose-dependent increase in the incidence of urothelial hyperplasia was noted in rats in the 0.1 g/L and higher dose groups. Foci of mineralisation of the renal papilla and eosinophilic droplets in the proximal tubule epithelium were also noted, without any information on dose levels. There were no other treatment-related non-neoplastic effects observed in any other tissue examined. On the basis of kidney effects in male rats, a NOAEL of 0.02 g/L (20 ppm; 1.5 mg/kg bw/day) was determined (US EPA, 2001; Health Canada, 2010).

These results also indicate that male B6C3F1 mice may be less sensitive to the effects of bromate exposure than rats. Bromate in drinking water had no effect on the body weights and survival of male mice. There was no increased incidence of non-neoplastic lesion in any tissue examined. Therefore, the highest tested dose of 0.8 g/L (77.8 mg/kg bw/day) is a NOAEL for male mice (US EPA, 2001; Health Canada, 2010).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

Although potassium bromate has been found to be genotoxic in a variety of assays (in vitro, in vivo), results were not sufficient to support its classification. The genotoxicity of potassium bromate has recently been linked to oxidative stress (US EPA, 2001; Health Canada, 2001; REACHa; REACHb).

Potassium bromate has been reported to be positive in Ames tests, with and without metabolic activation, with *Salmonella typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 (REACHa).

In assays using V79 Chinese hamster ovary cells, potassium bromate increased the frequency of cells with micronuclei, the number of chromosomal aberrations and the number of DNA strand breaks and induced gene mutations at the HPRT locus. The majority of chromosome aberrations observed were chromatid breaks and chromatid exchanges. Significantly increased levels of 8-oxodeoxyguanosine were also detected (Health Canada, 2010; REACHb). The result of a chromosomal aberration assay (Chinese hamster fibroblasts) using potassium bromate indicated a dose-related increase in the frequency of exchange-type aberrations (including gaps) (REACHb).

Potassium bromate induced: deoxyribonucleic acid (DNA) damage in cultured mammalian cells and primary human thyroid, white blood and kidney cells as measured by the *in vitro* comet assay; micronuclei in cultured mammalian cells and primary human lymphocytes and kidney cells; chromosomal aberrations, DNA repair, sister chromatid exchange, and DNA modifications (increased oxidation of DNA) in mammalian cell lines, primary human cultured cells and cell-free systems; and weak chromosomal aberration induction in cultured mammalian cells (Health Canada, 2010).

Induction of oxidative DNA modifications in isolated perfused kidneys or calf thymus DNA was not observed after potassium bromate administration. Dose-dependent increases in the number of aberrant metaphase cells in rat bone marrow cells were reported in all treated animals as acute cytogenetic effects of potassium bromate.

Potassium bromate induced micronuclei in vivo in multiple organs in rats and mice: micronucleated reticulocytes in CD-1 mice following intraperitoneal (IP) injection; peripheral blood cell micronuclei (micronuclei reticulocytes) in male F344 rats following IP injection; micronuclei in femoral bone marrow cells of mice following intraperitoneal injections; and micronucleated polychromatic erythrocytes in two strains of mice following gavage administration (US EPA, 2001; Health Canada, 2001).

Potassium bromate was negative with respect to in vivo genotoxicity assays: induction of micronuclei was not observed in spermatids, and no induction of DNA damage was observed in the lung, spleen or bone marrow of mice (Health Canada, 2010).

Carcinogenicity

Potassium bromate is currently classified as hazardous as a Category 2 carcinogen with the risk phrase ‘May cause cancer’ (T; R45) in the HSIS (Safe Work Australia). The available data support this classification (Health Canada, 1999; US EPA, 2001; WHO, 2005; REACHa).

Considering that potassium bromate and sodium bromate will produce similar effects through bromate ions, a classification similar to the above is also recommended for sodium bromate. This is supported by the classification of 'bromate moiety' as a carcinogen by other regulatory agencies (Health Canada, 1999; US EPA, 2001; WHO, 2005).

The International Agency for Research on Cancer (IARC) has concluded that there is inadequate evidence in humans for the carcinogenicity of potassium bromate. However, there is sufficient evidence in experimental animals for its carcinogenicity and it

is classified as possibly carcinogenic to humans (Group 2B) (IARC, 1999).

Health Canada has classified the bromate moiety as 'probably carcinogenic to humans, based on sufficient evidence in animals and no data in humans' (Health Canada, 1999). The US EPA has also classified the bromate moiety as a 'probable human carcinogen based on no evidence in humans, but adequate evidence of carcinogenicity in male and female rats' (Group B2 carcinogen) under previous guidelines and as a 'likely human carcinogen by the oral route of exposure, insufficient data for evaluation by the inhalation route' under current guidelines (US EPA, 2001).

Recently, the World Health Organization (WHO) evaluated the bromate moiety under the WHO Guidelines for Drinking-water Quality and stated that 'the weight of evidence from rat bioassays clearly indicates that bromate has the potential to be a human carcinogen' (WHO, 2005).

Several studies have been conducted in animals by oral administration to evaluate the carcinogenic effects of potassium bromate. The kidney is the major target organ of bromate-associated toxicity, rats are more sensitive than mice to bromate treatment and specific non-cancer effects include degeneration, necrosis, nephropathic, and regenerative changes in kidneys. The chemical produced tumours in kidneys (renal tubular tumours - adenomas and carcinomas) and the thyroid (follicular cell adenomas and carcinomas) and peritoneal mesotheliomas in males rats. However, only kidney tumours were developed in female rats and these were observed in the absence of the significant toxicity observed in the male rats. The chemical also produced a low incidence of renal cell tumours in male mice and the incidence of renal tubular tumours was marginally increased in male Syrian hamsters (IRIS, 2001; US EAP, 2001; WHO, 2005; Health Canada, 2010).

The exact mode of action for induction of tumours is not clear. However, considering the detection of 8-hydroxydeoxyguanosine in kidneys of rodents, the role of oxidative stress has been suggested in the formation of kidney tumours. The evidence is insufficient to establish lipid peroxidation and free radical production as key events responsible for the induction of kidney tumours. Even though the role of cell proliferation has also been proposed in the induction of tumours, the mechanism involving cell proliferation remains to be elucidated. Although bromate is mutagenic in bacteria and causes chromosomal aberrations, the role of mutation in the induction of tumours has also been questioned. The US EPA has suggested the predominant mode of action is DNA reactivity at low doses, considering the detection of tumours at relatively early time points and the positive response of bromate in a variety of genotoxicity assays (US EPA, 2001; WHO, 2005; Health Canada, 2010).

Reproductive and Developmental Toxicity

Limited data are available on the reproductive or developmental effects. However, the available information indicated that these chemicals are not likely to have specific reproductive or developmental effects.

In a short term reproductive and developmental toxicity study, sodium bromate was administered in the drinking water at concentrations of 0.25 ppm (2.6 mg/kg bw/day), 80 ppm (9.0 mg/kg bw/day), or 250 ppm (25.6 mg/kg bw/day) over a 35-day period. While group A females (10/group) were dosed from study day 1–34 to test for effects during conception and early gestation, group B females (13/group) were dosed from gestation day six to postnatal day one to test for effects during late gestation and birth. Male rats (10/group) were cohabited with Group B females for five days before dosing (study days 1–5) and were then dosed from study day six to days 34 and 35. Female reproductive function was not adversely affected and there were no treatment-related gross or microscopic changes in the kidney, liver, spleen, testes, or epididymis. The treated males in the 250-ppm dose group demonstrated a statistically significant decrease (18 %) in epididymal sperm density. Based on epididymal sperm density effect, a NOAEL of 80 ppm and a LOAEL of 250 ppm were derived (US EPA, 2001; NTP, 2007; Health Canada, 2010; REACHb).

In another study using the multigeneration, continuous-breeding paradigm, sodium bromate was administered to male and female Sprague-Dawley (SD) rats in drinking water at concentrations of 0, 30, 100, and 300 mg/L. The chemical produced general toxicity in male and female SD rats at 100 and 300 mg/L as noted by chronic progressive nephropathy and hyaline droplets in males and renal cell proliferative changes in females. Even though the chemical produced a 16 % decrease in sperm density in the F0 generation, the chemical is not considered a reproductive toxicant as no treatment-related changes were observed in the reproductive litter parameters. Although the sperm density was also decreased by 8 % in the F1 generation, the change was not statistically significant (NTP, 2007).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects of carcinogenicity and systemic acute effects from oral exposure to these chemicals.

Public Risk Characterisation

The use of these chemicals in cosmetic and domestic products in Australia is not known. Even though these chemicals have reported cosmetic and domestic uses overseas (see **Import, manufacture and use**), the available North American databases do not give evidence for the use of these chemicals in consumer (cosmetic and domestic) products. Considering the reported cosmetic use overseas (oxidising agents), the concentrations in the cosmetic products are not considered to be sufficiently high to cause any significant human health concerns.

Therefore, considering the limited consumer use, the risk to public health is not considered to be unreasonable and further risk management is not considered necessary for public safety.

Occupational Risk Characterisation

During product formulation, oral, dermal, ocular and inhalation exposure may 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.

Given the critical (systemic long-term/acute) health effects, the chemicals could pose an unreasonable risk to workers unless adequate control measures to minimise oral, 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.

NICNAS Recommendation

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 under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical and environmental hazards.

Note:

Whilst the existing classification is for potassium bromate (CAS No. 7758-01-2), this should apply to sodium bromate (CAS No. 7789-38-0).

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Toxic if swallowed (T; R25)*	Toxic if swallowed - Cat. 3 (H301)
Carcinogenicity	Carc. Cat 2 - May cause cancer (T; R45)*	May cause cancer - Cat. 1B (H350)

[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;
- health monitoring for any worker who is at risk of exposure to the chemicals, if valid techniques are available to monitor the effect on the worker's health;
- air monitoring to ensure control measures in place are working effectively and continue to do so;
- 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.

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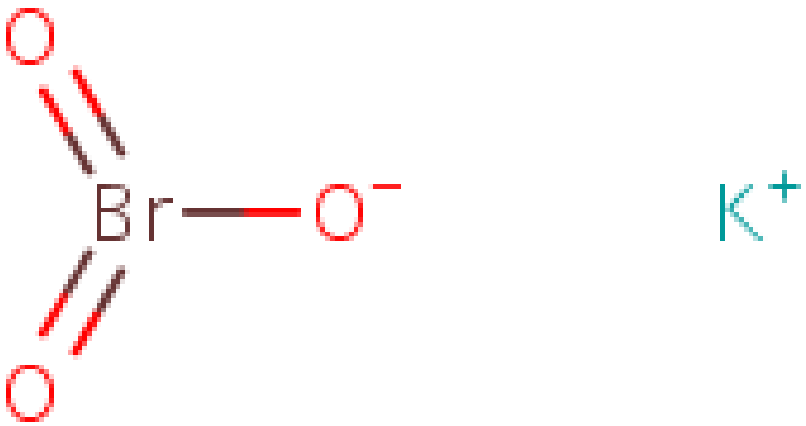
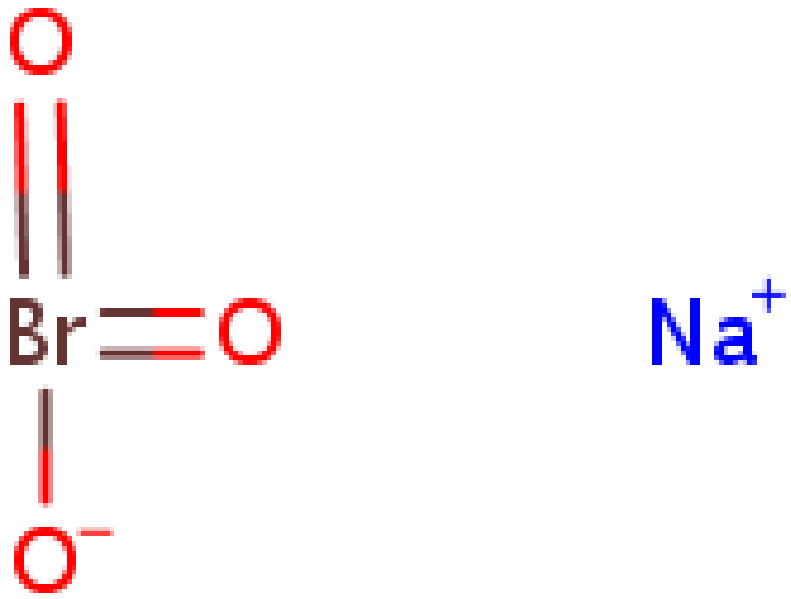
Last Update 03 July 2015

Chemical Identities

Chemical Name in the Inventory and Synonyms	**Bromic acid, potassium salt** potassium bromate bromic acid, potassium salt (1:1)
CAS Number	7758-01-2
Structural Formula	
Molecular Formula	BrHO3.K
Molecular Weight	167.00

Chemical Name in the Inventory and Synonyms	**Bromic acid, sodium salt** sodium bromate
CAS Number	7789-38-0

Structural Formula	
Molecular Formula	BrHO3.Na
Molecular Weight	150.90

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