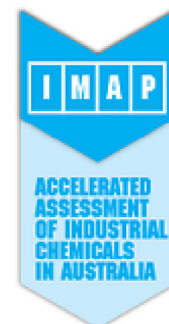


Propane, 1-bromo-: Human health tier II assessment

22 March 2013

CAS Number: 106-94-5



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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

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Acronyms & Abbreviations

Chemical Identity

Synonyms	1-Bromopropane Propyl bromide n-Propyl bromide 1-Propyl bromide
Structural Formula	
Molecular Formula	C3H7Br
Molecular Weight (g/mol)	123.0
Appearance and Odour (where available)	Colourless liquid
SMILES	C(Br)CC

Import, Manufacture and Use

Australian

The following Australian uses were reported under previous NICNAS calls for information (NICNAS Calls For Information 1991–2008).

The chemical has reported commercial use including:

- producing lubricants and fluid for gyroscopes; and
- as a heat transfer liquid and a component in hydraulic fluids.

The chemical has reported site-limited use including:

- as an intermediate in the manufacture of rubber compounds.

International

The following international uses have been identified through European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) Dossiers, Chemicalland21, the Substances and Preparations In the Nordic countries (SPIN), eChemPortal, and Hazardous Substances Data Bank (HSDB):

The chemical has reported domestic use including:

- in cleaning and washing agents [10 ppm limit by the American Conference of Governmental Industrial Hygienists (ACGIH) (Finkel, 2010)].

The chemical has reported commercial use including:

- manufacturing bulk/large scale chemicals, including petroleum products;
- as an industrial solvent for vapour degreasing (solvent generally for fats, waxes or resins); and
- in adhesive applications.

The chemical has reported site-limited use including:

- as an intermediate in the synthesis of pharmaceuticals, insecticides and other substances including quaternary ammonium compounds, flavours and fragrances.

Restrictions

Australian

No known restrictions have been identified.

International

The chemical is listed in the New Zealand Cosmetic Products Group Standard – Schedule 4: Components of cosmetic products must not contain (Galleria Chemica).

The chemical is also listed in the EU Cosmetic Directive 76/768/EEC Annex II and ASEAN Cosmetic Directive Annex II Part 1 List of Substances: The chemical must not form part of the composition of cosmetic products (ref no. 1139) (Galleria Chemica).

Existing Work Health and Safety Controls

Hazard Classification

The chemical is classified as hazardous with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

T; R60 (Repr. Cat. 2)

Xn; R63 (Repr. Cat. 3)

Xn; R48/20 (severe effects)

Xi; R36/37/38 (irritation)

Xi; R67 (other).

Exposure Standards

Australian

No specific exposure standards are available.

International

The following are identified (Galleria Chemica):

US DOE Temporary Emergency Exposure Limits (TEELs) = 10 ppm (TEEL-0), 30 ppm (TEEL-1) and 2500 ppm (TEEL-2 and TEEL-3);

Belgium Occupational Exposure Limits = 51 mg/m³ (10 ppm);

Canada (British Columbia, Nova Scotia and Prince Edward Island), Colombia, Italy, Nicaragua, Portugal, Spain, US ACGIH, Occupational Exposure Limits = 10 ppm (TWA);

Canada - Saskatchewan Occupational Health and Safety Regulations: 8-hour average Contamination Limits = 10 ppm and 15-minute average Contamination Limit = 20 ppm;

Finland Occupational Exposure Levels - Concentrations Known to be Harmful: HTP Value (8h) = 50 mg/m³ (10 ppm) and HTP Value (15min) = 250 mg/m³ (50 ppm);

Korea (South) Occupational Exposure Standards = 125 mg/m³ (25 ppm);

Lithuania Maximum Permissible Concentrations of Chemicals (Pollutants) in Air in Living Environment - Threshold limit value (TLV) = 0.03 mg/m³ (single) and 0.01 mg/m³ (daily);

Poland Workplace Maximum Allowable Concentration - NDS 8h/d - 40h/w = 42 mg/m³;

US - California Permissible Exposure Limits for Chemical Contaminants = 25 mg/m³ (5 ppm); and

US TSCA New Chemical Exposure Limits (NCEL) = 14.5 mg/m³.

Health Hazard Information

Toxicokinetics

Empirical evidence from rodent toxicity studies indicates that the chemical is absorbed via the inhalation route. Air partition coefficients in the blood of 7.08 for humans and 11.7 for rats indicate that the chemical is readily soluble in blood (air partition coefficient in fats for humans is 128 and for rats is 236) (NTP, 2003).

By four hours following inhalation exposure (800 ppm) or intravenous administration (5, 20, or 100 mg/kg), both rats and mice exhaled a majority of the co-administered [1,2,3-¹³C]-1-bromopropane and [14C]-1-bromopropane dose (rats, 50% to 71%; mice, 39% to 48%). The chemical was also excreted in urine (rats, 13% to 19%; mice, 13% to 23%) and faeces (rats, <2%; mice, 4%) or retained in tissues and carcass (rats, =6%; mice, <4%). *N*-acetyl-*S*-propyl-cysteine, *N*-acetyl-*S*-(2-hydroxypropyl)cysteine, *N*-acetyl-3-(propylsulfinyl) alanine, 1-bromo-2-hydroxypropane-*O*-glucuronide, *N*-acetyl-*S*-(2-oxopropyl)cysteine, and *N*-acetyl-3-[(2-oxopropyl)sulfinyl]alanine were the urinary metabolites characterised in rats and mice following both inhalation exposure and intravenous administration of the chemical. In rats, but not in mice, the route of elimination and the metabolite distribution changed significantly as the dose increased, with the percentage of dose excreted as unmetabolised chemical increasing significantly between the mid- and high-dose groups (NTP, 2011).

Studies on workers exposed to 1-bromopropane demonstrated that urinary elimination is an important excretion pathway for the chemical in humans (NTP, 2011).

Acute Toxicity

Oral

The chemical is reported to have low acute toxicity via the oral route.

Mouse oral LD50 = 4,700 mg/kg bw and rat oral LD50 = 3,600 mg/kg bw (Galleria Chemica).

Dermal

The chemical is reported to have low acute toxicity via the dermal route.

Rat dermal LD50 = 2,000 mg/kg bw and rabbit dermal LD50 = 10 mL/kg bw (Galleria Chemica).

Inhalation

The chemical is reported to have low acute toxicity via the inhalation route. However, the chemical is currently classified with the risk phrase 'Vapours may cause drowsiness and dizziness' (R67) in Australia. There is no information to support or remove this classification.

Rat inhalation LC50 = 253 g/m³/30 min (253,000 mg/m³) (ChemIDPlus).

Mouse inhalation LC50 = 7,100 mg/m³ and rat LC50 = 19700 mg/m³ (Galleria Chemica).

Corrosion / Irritation

Respiratory Irritation

No data are available.

The chemical is currently classified as a respiratory irritant (R37) in Australia. There is no information to confirm the validity of this classification and, therefore, the current classification was not amended.

Skin Irritation

No data are available.

The chemical is currently classified as a skin irritant (R38) in Australia. There are no data to confirm this classification, however, the chemical is reported as a skin irritant (Kosenko et al. 1973).

Eye Irritation

The chemical is currently classified with the risk phrase 'Irritating to eyes' (R36) in Australia. The data available support this classification.

An eye irritation study was performed on six male rabbits according to the scale of Draize. Since two out of the six animals tested exhibited a positive reaction, the test was repeated using a group of six different rabbits. The second test was considered positive since three animals out of six tested exhibited positive reactions. The chemical is considered to be an eye irritant (Galleria Chemica).

Sensitisation

Skin Sensitisation

No data are available.

Repeated Dose Toxicity

Oral

No data are available.

Dermal

No data are available.

Inhalation

The chemical is currently classified with the risk phrase 'Harmful: danger of serious damage to health by prolonged exposure through inhalation' (R48/20) in Australia. This classification applies when serious effects are likely to be caused by repeated or prolonged inhalation exposure at or below 0.25 mg/L (6 hours/day). The data available indicated that serious effects may occur above 1 mg/L (NOAEC) in rats with 13 weeks' exposure. Also, the chemical is reported to cause neurological effects in humans at very low concentrations (see **Other Health Effects**; Finkel, 2010). Considering the potential for neurotoxicity from inhalation, the current classification is supported.

Adverse effects in rats, including effects on reproductive organs/parameters and/or neurological damage, were reported in several studies (National Toxicology Program (NTP)) at doses 200 ppm or above. One 13-week study reported a NOAEC of 1 mg/L (200 ppm).

Groups of nine to 11 male Wistar rats were exposed via inhalation to the chemical at 200, 400, 800 and 1000 ppm for 8 hours/day, 7 days/week for up to 12 weeks. The effects on neurological function and reproductive organs were evaluated. Body weight gain and several organ weights (liver, brain, prostate, seminal vesicle, etc.) were significantly decreased at 1000 ppm. There was a time and concentration - dependent decrease in grip strength, motor nerve conduction velocity (MCV) of the tail and prolonged motor nerve distal latency. At 1000 ppm, statistically significant neurophysiological effects were evident by four weeks of exposure. There was evidence of myelin degeneration in the tibial nerve and neuroaxonal swelling in the medulla oblongata at the 1000 and 800 ppm exposure levels. The absolute weight of most reproductive organs and blood testosterone were significantly lower than controls at the 800 ppm level. The seminal vesicle was particularly affected by the chemical exposure with about a 50% reduction in weight in 800 ppm-exposed animals. This organ weight was still significantly reduced in the lowest exposure group (200 ppm). The chemical also caused decreases in epididymal sperm density and motility but did not effect spermatogonia development in the testis (NTP, 1999).

Male Wistar rats (nine per group) were exposed to 200, 400 and 800 ppm of the chemical for seven days. There was a significant decrease in body, prostate and seminal vesicle weights at 800 ppm. The exposure to the chemical significantly reduced the epididymal motile sperm rate and the percentage of abnormal sperm without affecting the sperm count. The reduction in motile sperm rate showed a concentration dependency and was significantly less than the control, even at 200 ppm. Minor histopathological changes in the tibial nerve were found at the 800 ppm level.

In a 13 week study, groups of 15 males and 15 female rats were exposed to 0, 0.5, 1, 2, 3 mg/L (0, 100, 200, 400, and 600 ppm) for 6 hours/day, 5 days/week. No significant treatment related clinical, functional or haematological effects were found. There was a significant increase in the relative liver weights in the male rats at the two highest doses. This was accompanied by mild centrilobular hepatocyte vacuolation in 6/15 male rats (statistically significant elevation) at 3 mg/L (600 ppm) and 3/15 rats (non-significant elevation) at 2 mg/L (400 ppm). The combination of liver weight increases with histopathological changes indicates slight to mild liver toxicity at the 2 mg/L (400 ppm) and 3 mg/L (600 ppm) exposures. No vacuolisation of brain tissue was reported at any exposure levels in this study. Based on these findings the authors reported a NOAEC of 1 mg/L (200 ppm) (NTP, 1999).

A Japanese group studied testicular toxicity in male Wistar rats exposed to 1500 ppm of the chemical, 6 hours/day, 5 days/week for three weeks followed by a two week recovery period. Exposure caused a time-dependent decrease in the number of spermatogonia followed by incomplete recovery during the post-exposure period (NTP, 1999).

In a 28 day study, groups of 10 male and 10 female Sprague Dawley rats were exposed to 0, 2, 5, and 8 mg/L (0, 400, 1000, or 1600 ppm) for 6 hours/day, 5 days/week. The high dose produced significant mortality in males and females by the end of the study period. Clinical signs of neurotoxicity (convulsions, incoordination, hunched postures etc.) were evident at the mid and high doses. This was confirmed by impairment in a modified functional observation battery (FOB). Several organ weights (liver, kidney, brain, lung) were marginally increased; haematologic parameters (red blood cells, haemoglobin etc.) were marginally decreased; and widespread histopathological damage was found in several tissues (testis, bone marrow, brain, spinal cord, kidney, bladder etc.) at the 8 mg/L (1600 ppm) exposure. Where examined, many of these changes were still present to a lesser extent at the 5 mg/L (1000 ppm) exposure. While there were no apparent clinical and haematological effects at the 2 mg/L (400 ppm) exposure, mild vacuolisation in the white matter of the brain was evident in almost half of the animals (5/10 males; 4/10 females) examined, indicating some neurological damage at this exposure level (NTP, 1999).

Genotoxicity

Considering all available data, the chemical is not considered genotoxic. Negative results were reported for all in vivo genotoxicity studies (micronucleus test and dominant lethal activity in male rats at 400 mg/kg bw and mice inhalation study with up to 500 ppm). Apart from one Ames study with two *Salmonella typhimurium* strains, all other Ames tests showed negative results for mutagenicity in many *Salmonella typhimurium* strains and in one *Escherichia coli* strain. One in vitro mouse lymphoma assay also reported mutagenic activity without metabolic activation.

The chemical showed mutagenic activity in the in vitro mouse lymphoma assay. It induced mutations at the TK (thymidine kinase) locus in L5179Y mouse lymphoma cells (OECD TG 476) with and without metabolic activation at test substance concentrations from 125 to 2500 µg/mL (Galleria Chemica).

The chemical was also reported to be mutagenic with and without metabolic activation in the Ames test using *S. typhimurium* strains TA1535 and TA100, but was negative in the reverse mutation assay using the Ames method. It was negative in the micronucleus test and negative for dominant lethal activity in male rats given 400 mg/kg (NTP, 1999).

The chemical was not mutagenic in either of two independent bacterial mutagenicity assays, each conducted with and without metabolic activation. Bacterial strains tested included *S. typhimurium* strains TA97, TA98, TA100, and TA1535, and *E. coli* strain WP2

uvrA/pKM101. In addition, no increases in the frequencies of micronucleated normochromatic erythrocytes were seen in male or female B6C3F1 mice exposed to 62.5 to 500 ppm 1-bromopropane via inhalation for 3 months (NTP, 2011).

The genotoxic potential of the chemical (100 to 10,000 µg/plate) was assessed by the Ames test on five *S. typhimurium* tester strains: TA1533, TA1537, TA1538, TA98 and TA100, both in the absence and presence of metabolic activation. A slight toxic effect was observed, mainly at 10,000 µg/plate on the five tester strains with metabolic activation, and in TA1535, TA 1538, TA98 and TA100 without metabolic activation. The assay was repeated. In both studies, whether in the presence or in the absence of metabolic activation, no increase was observed in the number of His⁺ revertant colonies/plate at any of the concentrations tested. The chemical was not genotoxic in the Ames test, with or without metabolic activation (Galleria Chemica).

Carcinogenicity

Based on the data available, the chemical is likely to be carcinogenic and a hazard classification is warranted according to the approved criteria and the GHS (see table under NICNAS Recommendation).

In a combined chronic toxicity/carcinogenicity study (OECD TG 453), groups of 50 male and 50 female F344/N rats were exposed to 1-bromopropane vapour at concentrations of 0, 125, 250, or 500 ppm, five days a week (six hours plus 10 minutes i.e. the theoretical value for the time to achieve 90% of the target concentration after the beginning of vapour generation (T90) per day), for 105 weeks (REACH Dossier, 2012). Survival of 500 ppm males was significantly less than that of the control group. Mean body weights of exposed groups were similar to those of the control groups. There was some evidence of carcinogenic activity in male rats, based on the occurrence of rare adenomas of the large intestine and increased incidences of neoplasms of the skin. Increased incidences of malignant mesothelioma and pancreatic islet adenoma may also have been related to the exposure. There was clear evidence of carcinogenic activity of the chemical in female rats based on increased incidences of adenoma of the large intestine. Increased incidences of neoplasms of the skin may also have been related to the exposure. Exposure to the chemical resulted in increased incidences of non-neoplastic lesions in the nose and larynx of rats, and the trachea of female rats. Suppurative inflammatory lesions with Splendore-Hoepli material were present, primarily in the nose and skin of male and female rats exposed to the chemical (REACH Dossier, 2012).

In another study (OECD TG 453), groups of 50 male and 50 female B6C3F1 mice were exposed to 1-bromopropane vapour at concentrations of 0, 62.5, 125, or 250 ppm, six hours plus T90 per day, five days a week for 105 weeks (REACH Dossier, 2012). Survival of exposed groups was similar to that of the control groups. Mean body weights of all exposed groups were similar to those of the control groups throughout the study. There was no evidence of carcinogenicity in male mice exposed to concentrations of 62.5, 125, or 250 ppm. There was, however, clear evidence of carcinogenicity in female mice based on increased incidences of alveolar/bronchiolar neoplasms. In the females, there were increased incidences of alveolar/bronchiolar adenoma, alveolar/bronchiolar carcinoma, and alveolar/bronchiolar adenoma or carcinoma (combined). The incidences of alveolar/bronchiolar adenoma or carcinoma (combined) were significantly increased in all exposed groups of females. There were significantly increased incidences of cytoplasmic vacuolisation of the bronchiolar epithelium in all exposed male groups and regeneration of the bronchiolar epithelium in all exposed groups of males and females. In the nose, there were significantly increased incidences of cytoplasmic vacuolisation of the respiratory epithelium in all exposed groups of males and in 125 and 250 ppm females. There were significantly increased incidences of respiratory epithelial hyperplasia in all exposed female groups and in 62.5 and 250 ppm males. There were significantly increased incidences of respiratory metaplasia of olfactory epithelium in 62.5 and 125 ppm males and 125 and 250 ppm females. There were significantly increased incidences of cytoplasmic vacuolisation of respiratory epithelium in the larynx and trachea of all exposed male groups and in the trachea of 62.5 and 125 ppm females (REACH Dossier, 2012).

Studies in rodents have also demonstrated that several structurally related brominated hydrocarbons are potent multisite carcinogens (NTP, 2011).

Reproductive and Developmental Toxicity

The chemical is currently classified with the risk phrases 'May impair fertility' (R60) and 'Possible risk of harm to the unborn child' (R63) in Australia. The data available support these classifications.

The NTP Center for the Evaluation of Risks to Human Reproduction (CERHR) evaluated the potential for 1-bromopropane to produce adverse reproductive and developmental effects in humans (NTP, 2003). CERHR concluded that there was convincing evidence for reproductive and developmental toxicity in experimental animals including decreased sperm motility and percentage of normal sperm in males; increased ovarian follicular cysts, longer oestrous cycle, decreased litter sizes and implantation sites in females; decreased foetal weights and increased incidences of

skeletal variations in pups. Evidence in humans was limited, but in the monograph a new case was noted that was not available to the expert panel, indicating positive findings in women (altered menstruation) occupationally exposed to 1-bromopropane. The overall NTP conclusion was that 'there is serious concern for reproductive and developmental effects of 1-bromopropane at the upper end of the human occupational exposure range (18 to 381 ppm).' 'Serious concern' is the highest level of NTP conclusion regarding the possibilities that human development and reproduction might be adversely affected (Finkel, 2010).

Reproductive and developmental effects were reported in repeat dose inhalation toxicity studies in rats (NTP, 1999).

Other Health Effects

Neurotoxicity

The chemical is likely to cause neurotoxicity in humans.

The chemical was reported to produce severe and apparently irreversible neuropathy, affecting gait and cognition in humans at exposure range 0.3 to 49 ppm (Finkel, 2010). Some repeat dose inhalation toxicity studies also showed clinical signs of neurotoxicity in animals (NTP, 1999).

Risk Characterisation

Critical Health Effects

The main critical effects to human health are the potential for carcinogenicity, fertility and developmental effects and neurotoxicity. The chemical will cause irritation to skin, eyes and the respiratory tract. The vapours of the chemical may cause drowsiness and dizziness.

Public Risk Characterisation

The chemical is reported to be used in domestic products (cleaning and washing agents) overseas. The chemical was not reported to be used in domestic products or cosmetics in Australia. Hence, the public risk from this chemical is low.

Occupational Risk Characterisation

Given the critical health effects, the risk to workers from this chemical is considered high unless adequate control measures to minimise occupational exposure to the chemical are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU), or an employee at a workplace, has adequate information to determine appropriate controls. The existing hazard classification for worker health and safety needs amending to include the classification for carcinogenicity.

NICNAS Recommendation

Current risk management measures are considered adequate for the protection of workers provided that the hazard classification is revised as recommended. No further assessment is required.

Regulatory Control

Public Health

As the chemical is not expected to be used in consumer products in Australia, no regulatory controls are recommended.

Work Health and Safety

The chemical is recommended for classification and labelling under the current approved criteria and adopted Globally Harmonized System of Classification and Labelling of Chemicals (GHS) as below. This does not consider classification of physical hazards and environmental hazards.

Hazard	Approved Criteria (HSIS) ^a	GHS Classification (HCIS) ^b
Acute Toxicity	Vapours may cause drowsiness and dizziness (R67)*	May cause drowsiness or dizziness - Specific target organ tox, single exp Cat. 3 (H336)
Irritation / Corrosivity	Irritating to eyes (Xi; R36)* Irritating to skin (Xi; R38)* Irritating to respiratory system (Xi; R37)*	Causes serious eye irritation - Cat. 2A (H319) Causes skin irritation - Cat. 2 (H315) May cause respiratory irritation - Specific target organ tox, single exp Cat. 3 (H335)
Repeat Dose Toxicity	Danger of serious damage to health by prolonged exposure (Xn; R48)*	May cause damage to organs through prolonged or repeated exposure - Cat. 2 (H373)
Carcinogenicity	Carc. Cat 3 - Limited evidence of a carcinogenic effect (Xn; R40)	Suspected of causing cancer - Cat. 2 (H351)
Reproductive and Developmental Toxicity	Repro. Cat 2 - May impair fertility (T; R60)* Repro. Cat 3 - Possible risk of harm to the unborn child (Xn; R63)*	May damage fertility. Suspected of damaging the unborn child - Cat. 1B (H360Fd)

^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 dermal, ocular or inhalation exposure to the chemical should be implemented in accordance with the hierarchy of controls: substitution, isolation and engineering controls. Measures required to eliminate or minimise risk arising from storage, handling and use of a hazardous chemical are dependent on the physical form and the manner in which the chemical is used. Examples of control measures which may minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- health monitoring for any worker who is at risk of exposure to the chemical, if valid techniques are available to monitor the effect on the worker's health;

- 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 chemical.

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

Obligations under workplace health and safety legislation

Information in this report should be taken into account to assist with meeting 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 hazardous chemical are prepared; and
- managing risks arising from storage, 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 physical hazards of the chemical has not been undertaken as part of this assessment.

References

Approved Criteria for Classifying Hazardous Substances [NOHSC: 1008(2004)] Third edition. Accessed at http://www.nohsc.gov.au/pdf/Standards/approved_criteriaNOHSC1008_2004.pdf

Chemicaland21.com. Accessed October 2012 at <http://www.chemicaland21.com/industrialchem/solalc/1-BROMOPROPANE.htm>

eChemPortal, 1-bromopropane (106-94-5). Accessed October 2012 at <http://www.echemportal.org/echemportal/substancesearch/substancesearchlink.action>

Finkel AM, June 2010. Supplemental report to the city of Philadelphia, Department of Public Health/Air Management Services. Increased Toxicity and Carcinogenicity of n-Propyl bromide relative to Perchloroethylene.

Galleria Chemica. Accessed November 2012 at <http://jr.chemwatch.net/galleria/>

Globally Harmonised System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third edition. Accessed at http://www.unece.org/trans/danger/publi/ghs/ghs_rev03/03files_e.html

Kosenko AM et al. 1973 Chem. Abstr. 6th, 2, 36. Cited in Patty's Industrial Hygiene and Toxicology, 1994. 4th edition, Volume II, Part E. pp 4154-4156.

National Toxicology Program - Center for the Evaluation of Risks to Human Reproduction (NTP-CERHR), 2003. Monograph on the potential human reproductive and developmental effects of 1-Bromopropane, October 2003.

National Toxicology Program (NTP) 1999. Nomination of 1-Bromopropane (1-BP) and 2-Bromopropane (2-BP) for Testing by the

