2-Propanol, 1-chloro-, 2,2',2''-phosphate: Human health tier II assessment

05 February 2016

CAS Number: 13674-84-5

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.

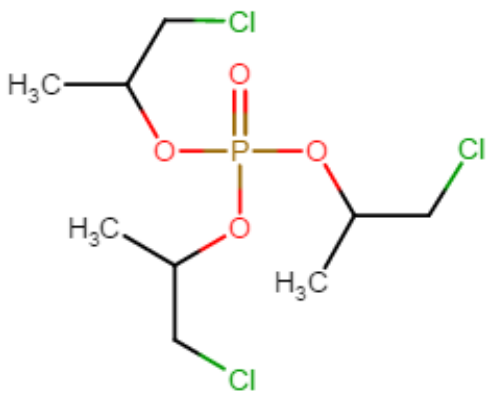
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	tri-(2-chloroisopropyl)phosphate tris (1-chloro-2-propyl) phosphate TCPP
Structural Formula	
Molecular Formula	C9H18Cl3O4P
Molecular Weight (g/mol)	327.57
Appearance and Odour (where available)	Clear, colourless liquid.
SMILES	C(C)(CCl)OP(=O)(OC(C)CCl)OC(C)CCl

Import, Manufacture and Use

Australian

The following Australian industrial uses were reported under previous mandatory and/or voluntary calls for information.

The chemical has reported commercial and site-limited use as a flame retardant that is incorporated into the material during the production and manufacture of:

- polyurethane flexible and rigid foams;
- polyurethane elastomers; and
- surface coating and fibreglass resins.

The total volume introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was 100–1000 tonnes per annum (tpa). It was reported to be imported at 290 tpa in 2000 (NICNAS, 2001).

International

The chemical has reported international commercial uses, including as a flame retardant that is incorporated into the material during the production and manufacture of polyurethane flexible and rigid foams, polyurethane elastomers and surface coating and fibreglass resins, as identified through:

- the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers;
- the Organisation for Economic Co-operation and Development Screening information data set International Assessment Report (OECD SIAR), under the OECD High Production Volume (HPV) program;
- Galleria Chemica;
- the Substances and Preparations in Nordic countries (SPIN) database;
- the US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR);
- the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and
- various international assessments (OECD, 2009; EU RAR, 2008).

Restrictions

Australian

No known restrictions have been identified.

International

No known restrictions have been identified.

Existing Work Health and Safety Controls

Hazard Classification

The chemical is not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

A preliminary Priority Exsting Chemical (PEC) assessment has been conducted by NICNAS on a group of chemicals known as trisphosphates, specifically chlorinated trisphosphates, including the chemical discussed in this report (TCPP; CAS RN 13674-84-5) (NICNAS, 2001). This preliminary PEC assessment did not involve a full risk assessment. In addition, since its publication, a substantial amount of data has been generated internationally on this chemical and is provided in this report. Information contained in the PEC assessment has been included in this report where still applicable and relevant.

The substance TCPP is manufactured from the reaction of phosphorous oxytrichloride (commonly oxychloride) with propylene oxide in the presence of a catalyst (Government of Canada, 2016). The final product is a reaction product that mainly consists of four trismonochloropropyl phosphate isomers (including CAS No’s 13674-84-5, 76025-08-6, 76649-15-5, 6145-73-9).

The composition of commercial TCPP is dominated (57-85 %) by the chemical discussed in this report, tris(1-chloro-2-propyl) phosphate (CAS No 13674-84-5). The CAS No 13674-84-7 is commonly used for commercial TCPP. The relative abundances of the other isomers vary depending on the manufacturing process (National Research Council, 2000; EU RAR, 2008).

Since individual isomers of the commercial substance are not produced individually or separated and marketed as such (NTP; EU RAR, 2008; Government of Canada, 2016), toxicity testing is normally expected to have been carried out on the commercial substance containing variable amounts of isomers (NICNAS, 2001).

The only TCPP product reported to be marketed is the commercial mixture, generally described as tris(1-chloro-2-propyl) phosphate. The other isomers are only available in this mixture and are not separately marketed. These isomers are therefore not considered commercially available. CAS No's 76025-08-6 and 76649-15-5 are not listed on the inventory. As the contribution of these isomers to the overall toxicity of TCPP is covered in the toxicological studies in this report, the remaining isomer CAS No 6145-73-9 is considered low priority for assessment at Tier II.

Toxicokinetics

In an oral toxicokinetic study in rats, a single 16.38 mg/kg dose of the chemical was orally administered to five animals that were monitored for seven days. The majority of the chemical (~90 %) was excreted through urine and faeces within the seven day observation period, with ~30 % excreted within the first three hours (EU RAR, 2008). The chemical was not considered to bioaccumulate. Highest levels of the chemical were detected in liver, kidney, lung and adipose tissues, with peak tissue concentrations detected at 5.7 hours after administration of the chemical. Only 0.7 % of the administered dose was detected in the carcass of animals after the seven day observation period.

In another oral toxicokinetic study in rats, a single oral dose of the chemical was administered to male and female rats at 200 mg/kg bw, and to male rats only at 20 mg/kg bw. Maximum serum concentrations of the chemical were detected at 0.5 and two hours after administration for the 20 and 200 mg/kg bw groups, respectively (OECD, 2009; EU RAR, 2008). Within the first 72 hours after administration, excretion levels were relatively similar across both dose groups (~89 %). At the end of the eight day observation period, excretion of the administered dose was 48 % and 70 % through urine and 40 % and 22 % through the

faeces for the 20 and 200 mg/kg bw group animals, respectively. Less than 1 % of the administered dose was detectable in tissue samples from both groups at the end of the eight day study period. Major metabolites detected were O,O-[bis(1-chloro-2-propyl)]-O-(2-propionic acid)phosphate (>50 %) and bis(1-chloro-2-propyl)monophosphoric acid (~12 %). Absorption from the gastrointestinal (GI) tract was calculated to be 68 % based on comparative results from a parallel intravenous dose levels of the chemical. GI absorption was therefore considered to be at least 75 % following ingestion (EU RAR, 2008).

An in vitro dermal absorption study was conducted by exposing human skin membranes to the chemical for eight hours (approximately equivalent to a standard working day) at concentrations of 0.002, 01 or 1 mg/cm^2. Resulting mean total absorption values were 22.7 %, 13.6 % and 3.7 %, respectively (EU RAR, 2008).

In another in vitro dermal absorption study, the amount of chemical potentially absorbed across the skin from handling polyurethane foams containing the chemical was assessed using skin membranes applied with artificial sweat containing the chemical for eight hours (EU RAR, 2008). Mean total absorption values were 33.3-38.1 %.

No toxicokinetics data are available following inhalation exposure.

Acute Toxicity

Oral

Based on the available experimental data in animals, the chemical has low to moderate acute oral toxicity.

Several acute oral toxicity studies on the chemical are available, with reported median lethal dose (LD50) values ranging from 632–4200 mg/kg bw across the studies. However, as the majority of results from well conducted studies report LD50 values <2000 mg/kg bw, hazard classification is recommended (REACH; OECD, 2009; EU RAR, 2008). Signs of toxicity included ataxia (unsteady or irregular movement), hunched posture, lethargy, laboured respiration, increased salivation, partially closed eyelids, body tremors, piloerection (raised fur), ptosis (drooping eyelids) and loss of righting reflex.

Dermal

The chemical has low acute toxicity based on results from animal tests following dermal exposure. The LD50 value (in both rats and rabbits) is reported to be >2000 mg/kg bw (OECD, 2009; EU RAR. 2008).

Inhalation

Based on the available experimental data in animals, the chemical is considered to have low to moderate acute toxicity following inhalation exposure.

Several acute inhalation toxicity studies (rats) on the chemical are available, with reported median lethal concentration (LC50) values ranging from 5 mg/L to >7 mg/L across the studies. Signs of toxicity included mild lethargy, matted fur, acute loss in bodyweight and in some cases, convulsions. The only deaths reported across the studies were in female animals; one study also reported reddened lungs observed in females only. However, due to the limited study details available, there is insuffucient information to warrant hazard classification (REACH; OECD, 2009; EU RAR, 2008).

Corrosion / Irritation

Skin Irritation

The chemical was reported to cause slight skin irritation in several experimental studies in animals (New Zealand White —NZW — rabbits) conducted according to OECD Test Guideline (TG) 404 (EU RAR, 2008). Slight erythema (redness of the skin) was

the main effect reported across the studies. However, all signs of irritation were reversible within the study observation periods; the reported effects are not sufficient to warrant hazard classification.

Eye Irritation

The chemical was reported to cause slight skin irritation in several experimental studies in animals (NZW rabbits) conducted according to OECD TG 405 (EU RAR, 2008). Slight redness and discharge from the eyes were the only effects reported across the studies. However, all signs of irritation were reversible within the study observation periods; the reported effects are not sufficient to warrant hazard classification.

Sensitisation

Skin Sensitisation

The chemical was not found to induce dermal sensitisation when tested in mice according to OECD TG 429 (local lymph node assay) (EU RAR, 2008). Test concerntations of the chemical were 25, 50 and 100 % v/v, with resulting stimulation indices of 1.55-1.97 across the groups. No signs of toxicity were observed and bodyweights were reported to be comparable to control group animals.

Repeated Dose Toxicity

Oral

In a 13-week repeated oral dose toxicity study, the chemical was administered in diet to Sprague Dawley (SD) rats (20 animals/sex/group) at 800, 2500, 7500 or 20000 ppm (reported as equivalent to 52, 160, 481 and 1349 mg/kg bw/day for males and 62, 171, 570 and 1745 mg/kg bw/day for females) (EU RAR, 2008). Increased (statistically significant) absolute and relative liver weights in males at all dose levels and females administered >= 7500 ppm were observed compared to control group animals. Mild thyroid follicular cell hyperplasia (growth) was also reported in males at all dose levels, with an increased occurance noted in relation to increasing dose. This effect was also seen in females from the highest dose group, but not in any of the control group animals. An increase in relative kidney weights in males at the two highest doses was reported to be a male rat specific observation and not relevant for humans. However, histopathological changes were noted in the kidneys of both males (two highest dose groups) and females (highest dose group), although effects were also observed in one female from the control group. In relation to the liver, histopathological examination revealed swelling of periportal hepatocytes of treated male and female animals; this observation was noted in control female but not in control male animals. A lowest observed adverse effect level (LOAEL) of 52 mg/kg bw/day (equivalent to 800 ppm) for males and a no observed adverse effect level (NOAEL) of 171 mg/kg bw/day (equivalent to 2500 ppm) for females was reported for this study.

In a 28-day repeated dose oral toxicity study, Wistar rats (5 animals/sex/dose) were administered the chemical daily by oral gavage at dose levels of 10, 100 or 1000 mg/kg bw/day. A NOAEL of 100 mg/kg bw/day was reported for this study (EU RAR, 2008). Effects observed at the highest dose (1000 mg/kg bw/day) included statistically significant increases in relative liver weights, significant decreases in alanine aminotransferase (ALT) enzyme activity and increases in hepatocyte hypertrophy in males only.

In a two-week dietary study in rats, a NOAEL of 1015 mg/kg bw/day was reported for males based on a statistically significant reduction in weight gain at higher concentrations (1636 mg/kg bw/day). No adverse effects were reported in females at up to the highest dose tested (1517 mg/kg bw/day) (EU RAR, 2008).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

The chemical is not considered to be a mutagen based on weight of evidence from several in vitro and in vivo experimental studies. While the chemical demonstrated clastogenic (chromosomal disruption) potential in some in vitro studies, no clear evidence of this was observed in any of the in vivo studies.

In vitro

The chemical did not cause bacterial mutations (Ames test, reported as conducted according to OECD test guidelines) in *Salmonella typhimurium* strains TA 98, 100, 1535 and 1537 and *Escherichia coli* strain WP2uvrA- at test concentrations of 8-5000 μg/plate, both with and without metabolic activation (EU RAR, 2008). The chemical was also non-mutagenic in several other bacterial tests (EU RAR, 2008) including:

- an Ames test using *S. typhimurium* strains TA 97, 98, 100, 1535 and 1537 at test concentrations of 3.3-1000 μg/plate, both with and without metabolic activation;
- several studies using *S. typhimurium* strains TA 97a, 98, 100, 102, 104, 1535, 1537 and 1538 at varying test doses, both with and without metabolic activation; and
- *E. coli* strains W3110/po1A+ and p3478/polA- test concentrations of 2-20 μl/plate, both with and without metabolic activation.

One early study (1983) in *S. typhimurium* reported that the chemical and some of its potential metabolites caused a dose-related responses at test concentrations ≤500 μg/plate. However, no records on the use of positive or negative controls were provided (EU, RAR, 2008).

The chemical did not cause mutations in two yeast cell studies in *Saccharomyces cerevisiae* strain D4 at test concentrations of 1-5000 nL/plate, both with and without metabolic activation.

In a mammalian cell mutagenicity study conducted according to OECD TG 476 (mouse lymphoma study), the chemical was shown to induce statistically significant increases in mutation (clastogenicity) frequency compared to controls, in the presence of metabolic activation, at test concentrations of 137.5-200 μg/mL. Higher concentrations (250 and 300 μg/mL) were reported to cause excessive toxicity and, therefore, were excluded from the main study. No significant mutagenic effects were observed in the absence of metabolic activation in this study (EU RAR, 2008). In one previously conducted mouse lymphoma study, the chemical did not induce mutations at the thymidine kinase (tk) locus in L5178Y cells (mouse lymphoma L5178Y/tk+/- assay) at test concentrations of 103-619 μg/mL, both with and without metabolic activation. However, in another mouse lymphoma L5178Y/tk+/- assay, while the chemical did not induce mutations without metabolic activation, it was reported to induce dose-related mutations with metabolic activation in one out of two test replicates, up to the highest dose of 472 μg/mL. A higher dose of 748 μg/mL was reported to cause almost complete cell death, although it was noted that only limited details are available for this study, including whether positive or negative controls were used (EU RAR, 2008).

The chemical did not cause genotoxic effects in two in vitro unscheduled DNA synthesis (UDS) assays in rat liver cells (one study conducted according to OECD guidelines) at test concentrations of 12.5-200 μg/mL (EU RAR, 2008). In a UDS assay in human embryonic lung WI-38 cells, the chemical, tested at concentrations of 6.45-129 μg/mL, was reported to induce genotoxicity both with and without metabolic activation. However, in addition to a non-standard cell line being used in this study, no clear dose-related response in the effects was observed (EU RAR, 2008).

In cell transformation studies using mouse embryonic fibroblast (BALB/3T3) cells, the chemical induced significant increases in the number of transformed foci at test concentrations of 50-400 μg/mL without metabolic activation. No dose-related response in effects was reported to be observed. No significant increase in transformed foci was observed in studies at lower doses of 3.22-51.6 μg/mL (EU RAR, 2008).

The chemical did not induce DNA strand breaks in a comet assay in Chinese hamster V79 cells at test concentrations of 1 or 1000 μM, with or without metabolic activation.

In vivo

The chemical did not induce clastogenic effetcs in an in vivo mouse micronucleus test. Adult NMRI mice (20 male and 20 female) were administered the chemical at 350 mg/kg bw by intraperitoneal (i.p.) injection. Signs of toxicity were observed in treated animals for up to 16 hours after dosing, with deaths occuring in two animals. Although the study was reported to be conducted according to OECD guidelines, the study methodology did not consistently align with the guideline, in that only one dose level was used and half the recommended number of polychromatic erythrocytes (PCEs; immature erythrocytes) were evaluated (EU RAR, 2008).

In a UDS assay in rats conducted according to OECD TG 486, the chemical was considered to not induce any biologically significant effects. The chemical was administered by oral gavage to female Wistar rats (8 animals/dose) at 750 or 1500 mg/kg bw. Signs of toxicity were noted in the high dose group animals. A statistically significant increase in the mean net nuclear grain (NNG) count (a measure of UDS) was reported. However, the NNG count was determined to be lower than the biologically significant level; therefore, the results from this study were considered to be equivocal (EU RAR, 2008).

In a cytogenetic assay rats, the chemical was reported to not induce increased chromosomal aberrations when orally administered at 14.2, 51.6 or 142 mg/kg bw (8 animals/dose). However, it was noted that this study was not conducted according to OECD test guideline recommendations (EU RAR, 2008).

Carcinogenicity

No carcinogenicity data are available for this chemical. Based on the available genotoxicity data (see **Genotoxicity** section), the chemical is not expected to be carcinogenic through a genotoxic mechanism. However, there is insufficient information to exclude carcinogenic potential through a non-genotoxic mechanism.

The chemical is structurally similar to two other chlorinated trisphosphates, TCEP (CAS no. 115-96-8) and TDCPP (CAS no. 13674-87-8), both of which are considered to be non-genotoxic carcinogens (EU RAR, 2008) and classified as carcinogens in the HSIS (Safe Work Australia). However, the reported differences in metabolism, target organs and severity of effects indicate that a quantitative read-across (preferable for non-genotoxic, or threshold carcinogens) from either TDCPP or TCEP to this chemical may not be appropriate in this case (EU RAR, 2008).

While no experimental carcinogenicity data are available for this chemical, the systemic effects and resulting LOAEL of 52 mg/kg bw/day reported in a 13-week repeated oral dose toxicity study in rats (see **Repeat dose toxicity** section) suggest that a non-genotoxic mechanism for formation of tumours at high doses following long-term exposure cannot be ruled out.

Reproductive and Developmental Toxicity

Based on the available information, the chemical has the potential to cause reproductive or developmental toxicity.

In a two-generation reproductive and developmental toxicity study in Wistar rats (28 animals/sex/group), the chemical was administered in the diet at concentrations of 1500, 5000 or 15000 mg/kg, resulting in intake levels of 85, 293 and 925 mg/kg bw/day for males and 99, 330 and 988 mg/kg bw/day for females (EU RAR, 2008). Animals were exposed to the chemical throughout the entire study, including a premating period of at least 10 weeks. Successful mating of initial parental animals (F0) produced first generation pups (F1), which were weaned on post-natal day 21 (PN21), then selected at random (28 animals/sex/group) for mating to produce the next generation. A LOAEL of 99 mg/kg bw/day for fertility effects was established from this study based on the significant decrease in absolute and relative uterus weights in all treatment group F0 females, with statistically significant increases in the length of the longest oestrus cycle and the mean number of cycles per animal in high dose females of both generations. Significant decreases in absolute and relative uterus weights of F1 females were observed in the high dose group. Significant decreases in spleen and pituitary weights were also observed in the F0 and F1 females of the high dose group.

A LOAEL for developmental toxicity was established at 99 mg/kg bw/day in the above study based on the statistically significant increase in the mean number of runts (small or weak offspring; defined as a pup with a weight less than the mean pup weight of the control group minus 2 standard deviations) within the F1 generation across all dose groups. An increased number of runts was observed in the high dose group throughout the lactation period for both F0 and F1 generations.

A NOAEL of 85 mg/kg bw/day was established for male parental toxicity from this study based on decreased body weights, decreased food consumption and changes in relative organ weights (brain, adrenal, kidney, liver, spleen, thyroid, epididymis and testes) observed in mid and high dose group animals.

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects of reproductive and developmental toxicity. The chemical can also cause harmful systemic effects following a single oral exposure. The systemic long-term effect of carcinogenicity cannot be excluded.

Public Risk Characterisation

Given the uses identified for the chemical, there is a low likelihood that the public will be exposed. Although the public could come into contact with articles and coated surfaces containing the chemical, it is expected that the chemical will be bound within the article or coated surface and hence will not expected to be bioavailable.

It is reported that there are some products containing the chemical that are available for use by the general public, i.e. polyurethane spray foams for use in filling of cavities during home repair and maintenance (EU RAR, 2008). This is not considered to be a widespread or regular consumer use; although likely short-term exposure is anticipated, long-term exposure to the chemical likely is to be negligible.

Occupational Risk Characterisation

During product formulation, 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 chemical at lower concentrations could also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise dermal exposure are implemented. The chemical 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 the chemical 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 chemical should be labelled in accordance with state and territory legislation (SUSMP, 2015).

Work Health and Safety

The chemical is 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.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)	Harmful if swallowed - Cat. 4 (H302)
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of impaired fertility (Xn; R62) Repro. Cat 3 - Possible risk of harm to the unborn child (Xn; R63)	Suspected of damaging fertility or the unborn child - Cat. 2 (H361fd)

[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 chemical should be used according to the instructions on the label.

Advice for industry

Control measures

Control measures to minimise the risk from dermal and ocular exposure to the chemical 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 chemical is 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 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 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 chemical 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 the chemical has not been undertaken as part of this assessment.

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