Phenol, 4-(1,1-dimethylethyl)-: Human health tier II assessment

01 July 2016

CAS Number: 98-54-4

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.

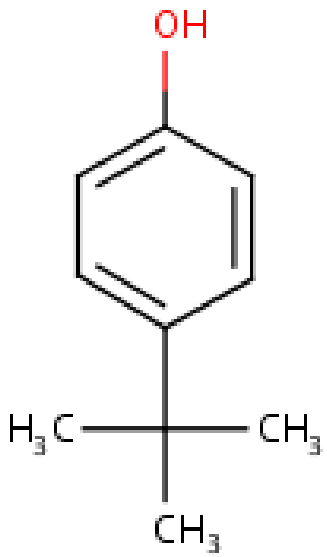
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	phenol, p-tert-butyl- p-tert-butylphenol
Structural Formula	
Molecular Formula	C10H14O
Molecular Weight (g/mol)	150.22
Appearance and Odour (where available)	white hygroscopic flakes, disinfectant-like odour.
SMILES	C(C)(C)(C)c1ccc(O)cc1

Import, Manufacture and Use

Australian

The chemical has reported potential domestic use in surface coatings.

Total volume of p-tert-butylphenol introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was in the range of 10–100 tonnes. No specific use information was identified.

International

The following international uses have been 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); Galleria Chemica; the US Household Products Database; the Substances and Preparations in Nordic countries (SPIN) database; and the EU Risk Assessment Report (EU RAR).

Although there is limited evidence that p-tert-butylphenol may have cosmetic use as a fragrance ingredient, it is unlikely given that its use in cosmetics is extensively restricted. It is listed as a prohibited ingredient in fragrances by the International Fragrance Association and it is not listed in the Compilation of Ingredients Used in Cosmetics in the United States (ed. Bailey, 2011).

The chemical has reported domestic uses, including:

- as a hardener in epoxy resins (adhesives and binding agents);
- in colouring agents;
- in fillers;
- in odour agents;
- in paints, lacquers and varnishes; and
- in surface treatment.

The chemical has reported commercial uses, including:

- in construction materials;
- in electromechanical components;
- as a corrosion inhibitor;
- in viscosity adjusters; and
- as a process regulator.

The chemical has reported site-limited uses, primarily as an intermediate in producing polycarbonates, phenolic and epoxy resins.

Restrictions

Australian

No known restrictions have been identified.

International

The chemical is listed on the following (Galleria Chemica):

- EU Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- ASEAN Cosmetic Directive Annex II Part 1: List of substances which must not form part of the composition of cosmetic products;
- International Fragrance Association (IFRA) Standards Prohibited:
- China List of Banned substances for use in Cosmetics;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain; and
- Health Canada List of Prohibited and Restricted Cosmetic Ingredients (The Cosmetic Ingredient 'Hotlist').

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

The following exposure standards are identified (Galleria Chemica).

An exposure limit of 0.5 mg/m^3 (0.08 ppm) time weighted average (TWA) in different countries such as Denmark, Germany, Iceland and Switzerland. Switzerland also has a 1.0 mg/m^3 (0.16 ppm) short-term exposure limit (STEL).

Health Hazard Information

Toxicokinetics

There are no data available from toxicokinetic studies conducted in accordance with OECD Test Guideline (TG) 417. However, the role of sulfonation and glucuronidation in the biotransformation of p-tert-butylphenol was assessed in vivo in rats as well as in vitro in rat and human systems. The urinary metabolite levels in workers handling this chemical were also measured (EU RAR, 2008).

In rats exposed to the radiolabelled chemical by the oral route, 26.7 % and 72.9 % of the applied dose were eliminated in faeces and urine, respectively. In another in vivo rat study, 65–71 % and 17–21 % of intravenously injected radiolabelled chemical were excreted as glucuronide and sulfate conjugates, respectively. The total recovery of radioactivity was 91–93 %. In vitro studies investigating the enzyme activity of the chemical and similar phenolic compounds in rat hepatocytes and the human liver supported the results of the in vivo rat study with intravenously injected p-tert-butylphenol. Retention of the chemical after seven days in rat studies was negligible (0.1 %) and the likelihood for bioaccumulation is low. This is further supported by the physicochemical properties of p-tert-butylphenol (water solubility (600 mg/L), log Pow value at 3.31 and low molecular weight 152) which indicate that the likelihood for bioaccumulation is low (EU RAR, 2008).

The levels of metabolites in the urine of workers handling p-tert-butylphenol increased with increasing exposure to the parent compound. Most of the p-tert-butylphenol was shown to be excreted within 24 hours. The studies indicated that skin penetration plays an important role as a route of entry in addition to vapour exposure (EU RAR, 2008).

Acute Toxicity

Oral

The chemical has low acute toxicity based on results from animal tests following oral exposure. The majority of the studies reported LD50 values above 2000 mg/kg bw (EU RAR, 2008).

In an oral acute toxicity study performed according to OECD TG 401 and good laboratory practice (GLP), five Sprague Dawley (SD) rats (5 animals/sex) received 2000 mg/kg bw of p-tert-butylphenol suspended in arachis oil and administered by gavage. No deaths and no signs of systemic toxicity were noted during a 14 day observation period. In the accompanying range-finding study, a male died following exposure to 5000 mg/kg bw, whereas a female survived. Observed sub-lethal effects in the male receiving the 5000 mg/kg bw dose included hunched posture, lethargy, ptosis, red/brown stains around the snout and ataxia (EU RAR, 2008).

Dermal

The chemical has low acute toxicity based on results from animal tests following dermal exposure. The LD50 in rabbits is >2000 mg/kg bw (EU RAR, 2008).

In a study similar to OECD TG 402, p-tert-butylphenol was moistened with distilled water and applied to the clipped skin of New Zealand White rabbits (5/sex/dose) at 2000, 8000, and 16000 mg/kg bw. The chemical remained in contact with the skin for 24 h under occlusive conditions. Signs of toxicity included reduced body weight in the mid and high dose groups and skin irritation. No animals died in this study. Severe skin irritation was observed in both sexes at all doses (REACH).

Inhalation

No data are available from studies compliant with the OECD TG for acute inhalation toxicity. However, a limit test indicates that p-tert-butylphenol has low acute, systemic toxicity following inhalation with a median lethal concentration (LC50) >5000 mg/m^3. A limit test was performed in which SD rats (five/sex) were exposed whole body for 4 h to the chemical as a dust aerosol of 5600 mg/m^3 (median particle diameter of 3.6 micrometres) with an additional vapour component of 30 mg/m^3. Clinical signs observed on the day of exposure and up to seven days after exposure included signs of mucosal irritation (perinasal, perioral, and periocular encrustation) and signs of respiratory distress (audible respiration, gasping, and decreased respiration rate). Within one to two days following exposure, one rat of each sex died. The dead animals showed dark red or purple discolouration of the lungs and/or kidneys. No macroscopic lesions were observed in the surviving animals (EU RAR, 2008).

Corrosion / Irritation

Skin Irritation

Based on the available data, the chemical is a skin irritant and warrants hazard classification (see **Recommendation** section). In addition, the chemical may induce partial depigmentation of the skin (EU RAR, 2008).

In a skin irritation study performed in accordance with OECD TG 404 and GLP, the chemical was applied to the intact skin of three New Zealand White rabbits, producing severe erythema and very slight to moderate oedema after 4 hours. Mean scores for erythema were 4 (24 h), 4 (48 h), 3.3 (72 h) and 0 (14 days). Mean scores for oedema were 2 (24 h), 1.3 (48 h), 1.7 (72 h) and 0 (14 days). After 14 days, the exposed areas were free of irritation (REACH).

Eye Irritation

Based on the available data, the chemical causes serious, irreversible damage to eyes and warrants hazard classification (see **Recommendation** section).

In a study similar to OECD TG 404, p-tert-butylphenol was placed in the eyes of six New Zealand White rabbits, producing severe corneal injury, iritis and severe conjunctival irritation. The following mean scores were reported: corneal opacity of grade 1 (1 h) to 3.2 (7 d), iris lesion grade 1, conjunctival redness of grade 1.8 (1 h) to 2.2 (72 h), and chemosis of grade 2.3 (1 h) to 3.8 (72 h). Due to corneal opacity, the scoring of iris lesions after 4 h was not possible in many animals and thus reversibility could not be established. The corneal opacity was still apparent 21 days after exposure (mean score 2.5; range 0– 4) (EU RAR, 2008).

Sensitisation

Skin Sensitisation

The data for assessing the skin sensitisation potential of p-tert-butylphenol are limited and variable in quality. They are not sufficient to support classification of the chemical as a sensitiser.

The chemical was not found to induce dermal sensitisation in a maximisation test using ten male Dunkin Hartley/Pirbright White guinea pigs conducted according to OECD TG 406 and GLP. Concentrations used were: intradermal induction phase (0.5% in corn oil); epicutaneous induction phase (10% in Vaseline); and challenge phase (1% in Vaseline) (REACH).

The EU Risk Assessment Report (EU RAR, 2012) describes a number of sensitisation studies with conflicting results including a positive animal study and some human studies. The report and the European Chemicals Agency (ECHA) Risk Assessment Committee (RAC) noted that the positive animal study was conducted some time ago and the protocol is not well described. No firm conclusions can be drawn based on the animal studies as a whole. However, based on the scientific quality of the studies, it appears more likely that p-tert-butylphenol does not cause skin sensitisation in animals, particularly given the negative results from the OECD TG and GLP compliant study (described above). The human data are also of limited value since most of the studies show very few positive results and they are mainly performed on patients with former skin allergy or other skin diseases or there is limited information about exposure. The RAC deliberated about the available evidence and decided against classification for sensitisation (ECHA RAC, 2012).

Repeated Dose Toxicity

Oral

A combined repeated dose and reproductive/developmental toxicity screening test (OECD TG 422) using SD rats (13 animals/sex/dose) at 20, 60 and 200 mg/kg bw/day has been conducted. The no observed adverse effect level (NOAEL) was 60 mg/kg bw/day and the lowest observed adverse effect level (LOAEL) was 200 mg/kg bw/day based on respiratory distress in exposed females and effects on several blood parameters in males (REACH).

Long-term dietary exposure to high doses of p-tert-butylphenol induced moderate effects on relative kidney and liver weights in two carcinogenicity studies. In the first study, 7-week old male Syrian Golden hamsters (15 animals) were exposed to 1.5% of the chemical in the diet (approximately 1230 mg/kg bw/day) for 20 weeks. The average body weight was lower (5%) compared to the control group and the relative liver weight was greater by approximately 20%. In the second study, 20 Fischer 344 male rats were given 1.5% of the chemical in the diet for 51 weeks (approximately 600 mg/kg bw/day). Relative liver weights decreased by approximately 8% and relative kidney weights increased by approximately 13% (ECHA RAC, 2012). These studies are discussed further in the **Carcinogenicity** section.

Dermal

No data are available.

Inhalation

No data are available.

Observation in humans

The EU RAR report summarises the outcomes of depigmentation studies in workers following exposure to the chemical. Based on these studies, it is considered that the chemical can induce depigmentation of the skin in humans. This effect was likely to have been induced not only via direct contact with skin but also via inhalation or ingestion routes (EU RAR, 2008).

Genotoxicity

Based on the weight of evidence from the available in vitro genotoxicity studies, the chemical is not considered to be genotoxic. There are no available data from in vivo studies.

A bacterial reverse mutation test was conducted in accordance with OECD TG 471 with GLP using four *Salmonella typhimurium* strains (TA100, TA98, TA1535, TA1537) and *Escherichia coli* WP2 up to a maximum concentration of 5 mg/plate of the chemical with and without an exogenous metabolic activation system. Negative findings were reported in this study (REACH).

Other studies also indicate that this chemical did not induce gene mutation in *S. typhimurium* TA98, TA100, TA1535, TA1537 (maximum concentration of 1 mg/plate, -S9); *E. coli* WP2uvrA (maximum concentration of 0.5 mg/plate, -S9); *S. typhimurium* TA100, TA1535, TA1537 (maximum concentration of 0.5 mg/plate, +S9); and *E. coli* WP2 uvrA and TA98 (maximum concentration of 1 mg/plate, +S9) (REACH).

The chemical induced structural chromosome aberration in CHL/IU cells with exogenous metabolic activation after short term treatment. Chromosome aberrations were seen in 6.5–12.0 % of cells at all concentrations in a study conducted in accordance with OECD TG 473. This chemical also induced polyploidy with and without exogenous metabolic activation system. However, negative results were obtained in two other mammalian chromosomal aberration tests conducted using the same OECD Test Guideline. In one study conducted in accordance with GLP, this chemical induced neither clastogenicity nor polyploidy in rat lymphocytes following 20 or 30 hours' treatment with and without exogenous metabolic activation. In another study, this chemical did not induce chromosomal aberration in rat liver epithelial-type cells following 24 hours' treatment without exogenous metabolic activation at higher concentrations than in the above studies. Polyploidy was not included as an outcome (SIAR, 2000).

This chemical was not mutagenic to L5178Y mouse lymphoma cells at the thymidine kinase TK +/- locus in a study conducted in accordance with OECD TG 476 with and without exogenous metabolic activation (REACH).

Given the positive results observed in one of the chromosome aberration tests, the possibility of *in vivo* genotoxicity remains. However, the study reporting positive results had methodological limitations compared with the studies obtaining negative results for chromosome aberrations. The ECHA RAC considered that, based upon the weight of evidence and the quality of the information provided, the evidence does not support classification (ECHA RAC, 2012).

Carcinogenicity

The available carcinogenicity studies are not sufficient to assess the carcinogenic potential of p-tert-butylphenol. However, given the available data and the uncertain mutagenic effects, it is considered unlikely that the chemical is a human carcinogen.

In high dose dietary studies conducted in male rats and Syrian Golden hamsters (described in the **Repeat Dose** section), the only relevant effects were observations of hyperplasia in the forestomach of rats and hamsters. Papillomatous lesions were also induced in the stomach of hamsters. In addition, the chemical induced forestomach tumours in an initiation-promotion study in rats following initiation with N-methyl-N'-nitro-N-nitrosoguanidine (MNNG).

It is not possible to draw any absolute conclusions in relation to the possible carcinogenic potential of the chemical alone. The chemical probably acts as a promoter in the induction of rodent forestomach tumours. The evidence pertaining to the location of the tumours (the rodent forestomach) and the fact that the mechanism is likely to involve p-tert-butylphenol acting as a promoter does not support classification as a carcinogen (ECHA RAR, 2012).

Reproductive and Developmental Toxicity

Based on the available data, the chemical showed specific reproductive effects, warranting a hazard classification for reproductive toxicity (see **Recommendations** section). Developmental effects were only observed secondary to maternal toxicity.

Reproductive Toxicity

In a two-generation reproduction study performed according to OECD TG 416 in compliance with GLP, SD rats were exposed to p-tert-butylphenol. The chemical was given orally in the diet at 0, 800, 2500 and 7500 mg/kg, corresponding approximately to 0, 70, 200 and 600 mg/kg bw/day. In the parental (F0) generation 28 rats/sex/group were mated to produce the first group of offspring (F1). Animals from the F1 generation (24/sex/group) were subsequently mated to produce the second generation of pups (F2).

The following results were reported: at 2500 ppm, statistically significant decreases in weight gain were reported in F0 and F1 animals prior to mating. During gestation and lactation, statistically significant reductions in body weight gain were reported at 7500 ppm. Statistically significant reductions in food consumption were reported at 2500 ppm in F0 and F1 animals prior to mating. During gestation and lactation, statistically significant reductions in food consumption were reported at 7500 ppm. At concentrations up to 7500 ppm, no effects on mating performance, fertility or duration of gestation were reported. However, at 7500 ppm, slight decreases in the number of implantation sites, live pups born and viability of the pups were reported. Furthermore, decreases in pup body weights and litter weights in the F1 and F2 generation from 2500 ppm were reported on lactation day 14, as well as a smaller litter size. Pup survival was reduced, particularly over days 1–4 of lactation when six different litters had more than 3 pups dying and in two of these litters all pups died. Delays in vaginal opening and preputial separation in the F1 generation were reported at 7500 ppm. In the F0 and F1 female generations, marked increases in atrophy of the vaginal epithelium were reported from 2500 ppm. The severity in the vaginal epithelial atrophy in the F1 generation was greater compared to the F0 generation. Increases in the incidence of primordial follicles with concurrent decreases in the incidence of growing follicles were reported in the F0 and F1 females at 7500 ppm. This effect was also more pronounced in the F1 generation. From 2500 ppm, statistically significant decreases in the weight of the ovaries were reported in the F0 generation but this was only seen at 7500 ppm in the F1 generation. A NOAEL at 800 ppm (corresponding to 70 mg/kg bw/day) was derived for effects on reproduction and development from the 2-generation study.

In a combined repeated dose and reproductive/developmental toxicity screening test conducted according to OECD TG 422 and GLP, p-tert-butylphenol was administered to SD (Crj: CD) rats (13/sex/dose) at 0 (vehicle), 20, 60, 200 mg/kg bw/day by gavage for 44 days in males and from 14 days before mating to day 3 of lactation in females. No treatment-related changes were observed except noisy respiratory sounds in a few females exposed at the highest dose (REACH). The NOAEL for effect on fertility was = 200 mg/kg bw/day and the NOAEL was 60 mg/kg bw/day for systemic toxicity in the parental animals (EU RAR, 2008).

It was noted that the effects seen at the high dose in the two-generation study were seen at doses which, over parts of the study, exceeded the limit dose for reproductive toxicity classification at 1000 mg/kg bw/day (ECHA RAC, 2012).

Developmental Toxicity

The ECHA RAC concluded that the available data are not sufficient to warrant classification for developmental toxicity (ECHA RAC, 2012). This is based on the fact that no embryotoxicity or teratogenicity was induced at the tested doses in the combined repeated dose and reproductive/developmental study and the doses causing significant fertility effects in the 2-generation reproduction study did not cause significant developmental toxicity effects in the absence of maternal toxicity (ECHA RAC, 2012).

Other Health Effects

Endocrine Disruption

The EU Risk Assessment Report (EU RAR, 2008) described a number of in vitro studies indicating that the chemical had the potential to induce proliferation of the oestrogen-dependent human breast cancer cell line MCF-7 through binding to the oestrogen receptor and inducing oestrogen-regulated proteins. However, p-tert-butylphenol has a binding affinity that is 10000-fold less than 17β−oestradiol. Utilising in vitro assays such as this oestrogen-binding test for predicting in vivo endocrine activity may generate false-negative as well as false-positive results. More emphasis should be put on in vivo adverse outcomes. The two-generation study (OECD TG 416) is currently the most complete study available for endocrine activity via the oestrogenic and androgenic pathways (see the **Reproductive and Developmental Toxicity** section).

A study evaluated the effects of the chemical on the prenatal testicular testosterone surge in SD rats in utero. Subcutaneous injection of p-tert-butylphenol into dams at doses of 1.0, 10 and 100 mg/kg bw on embryonic day 13.5, 15.5 and 17.5 did not decrease testicular testosterone content, whereas exposure to diethylstilboestrol (DES) (18 dams, 0.01, 0.1, or 0.2 mg/kg bw) caused a significant depression in testosterone content and secretion. The authors of the study concluded that alkylphenols have less severe and even opposite in utero effects on the prenatal rat testicular testosterone steroidogenesis when compared with the effect of DES, which induced a significant depression of testosterone content (EU RAR, 2008).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (reproductive toxicity). The chemical can also cause skin and eye irritation.

Public Risk Characterisation

Provided that normal precautions are taken to avoid prolonged skin and eye contact, the risk to public health posed by adhesives, paints, lacquers and similar products containing the chemical is not considered to be unreasonable.

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 systemic long-term and local health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise dermal, ocular and inhalation 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.

The data available support hazard classification in the HSIS (Safe Work Australia) (refer to **Recommendation** section).

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

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]
Irritation / Corrosivity	Risk of serious eye damage (Xi; R41) Irritating to skin (Xi; R38)	Causes serious eye damage - Cat. 1 (H318) Causes skin irritation - Cat. 2 (H315)
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of impaired fertility (Xn; R62)	Suspected of damaging fertility - Cat. 2 (H361f)

[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, ocular and inhalational 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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