Benzene, 1,2-dimethoxy-4-(2-propenyl)-: Human health tier II assessment

03 July 2015

CAS Number: 93-15-2

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.

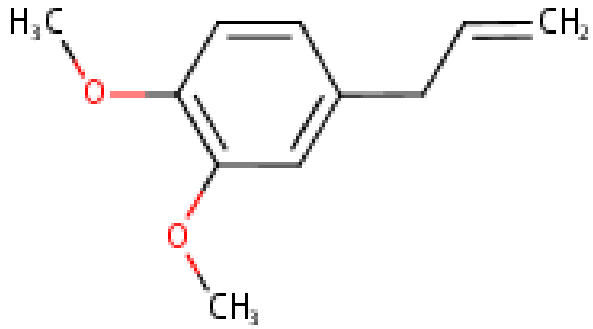
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	methyl eugenol 1,3,4-eugenol methyl ether 4-allyl-1,2-dimethoxybenzene 1,2-dimethoxy-4-(2-propenyl)benzene 1-allyl-3,4-dimethoxybenzene
Structural Formula	
Molecular Formula	C11H14O2
Molecular Weight (g/mol)	178.22
Appearance and Odour (where available)	Colourless to pale yellow liquid with a clove-carnation odour
SMILES	c1(OC)c(OC)cc(CC=C)cc1

Import, Manufacture and Use

Australian

The following non-industrial use has been identified in Australia: as an insect attractant in pesticides (Australian Pesticides and Veterinary Medicines Authority—APVMA).

International

The following international uses have been identified through: Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (CosIng) database; the United States (US) National Library of Medicine's Hazardous Substances Data Bank (HSDB); and various international assessments including from the International Agency for Research on Cancer (IARC), the National Toxicology Program (NTP) and Health Canada.

The chemical is a naturally occuring substance, present in essential oils, but it was reported to be also manufactured in small quantities. It has been used as a flavouring agent or fragrance ingredient in many sectors.

The chemical has reported cosmetic use as a fragrance ingredient.

The chemical has reported domestic use as a component of essential oils used in household cleaning products.

The chemical has also reported non-industrial uses as:

- a flavouring agent in food; and
- a component in insect repellent.

Restrictions

Australian

This chemical is listed in Schedule 6 of the *Poisons Standard—the Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP, 2015) as 'METHYLEUGENOL except in preparations containing 1 per cent or less of methyleugenol.'

Schedule 6 chemicals are described as 'Substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label'. Schedule 6 chemicals are labelled with 'Poison' (SUSMP, 2015).

International

In 2000, the Scientific Committee on Cosmetic Products and Non-Food Products (SCCNFP) concluded that the chemical should not be 'intentionally added as a cosmetic ingredient' (SCCNFP, 2000). However, the chemical is a fragrance raw material, and using it in cosmetics is subject to restrictions in many countries including the European Union (EU) (CosIng).

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

- the Association of South East Asian Nations (ASEAN) Cosmetic Directive Annex II Part 1—List of substances which must not form part of the composition of cosmetic products as 'Methyleugenol (CAS No 93-15-2) except for normal content in the natural essences used and provided that the concentration does not exceed: (a) 0.01 % in fine fragrance (b) 0.004 % in eau de toilette (c) 0.002 % in fragrance cream (d) 0.001 % in rinse-off products (e) 0.0002 % in other leave-on products and oral hygiene products';
- EU Cosmetics Regulation 1223/2009 Annex III—List of substances which cosmetic products must not contain except subject to the restrictions laid down: the maximum concentration in ready-for-use preparation are 0.01 % in fine fragrance, 0.004 % in eau de toilette, 0.002 % in a fragrance cream, 0.0002 % in other leave-on products and in oral hygiene products, and 0.001 % in rinse-off products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain as 'Methyleugenol except for normal content in the natural essences used and provided that the concentration does not exceed: (a) 0.01% in fine fragrance, (b) 0.004% in eau de toilette, (c) 0.002% in fragrance cream, d) 0.001% in rinse-off products and (e) 0.0002% in other leave-on products and oral hygiene products'; and
- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient ‘Hotlist’) as 'Methyl eugenol (93-15-2): Permitted as a naturally occurring component in botanical extracts at concentrations equal to or less than 0.01% in fine fragrances, 0.004% in eau de toilette, 0.002% in a fragrance cream, 0.0002% in other leave-on products and in oral hygiene products, and 0.001% in rinse-off products'.

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

Toxicokinetics

Rat studies have shown that the chemical is rapidly absorbed through oral exposure, with peak plasma levels achieved within the first five minutes. About 72 hours after oral or intravenous administration in rats, the chemical is distributed primarily to the liver and, to a lesser extent, to the kidneys. It is rapidly metabolised and excreted via the urine. In one rat study, 85 % of the absorbed dose was found in the urine after 72 hours, either as the parent form or as a metabolite (NTP, 2000).

A study conducted in nine human volunteers who ingested the chemical, also showed that the chemical was rapidly absorbed, with peak blood levels reached 15 minutes after ingestion. The half-life for elimination was 90 minutes (Schecter et al., 2004).

According to the IARC, the metabolic enzymatic pathways of the chemical are similar for rodents and humans, supporting the relevance of animal data to humans, especially regarding carcinogenicity mechanisms. In particular, mutations and DNA damage observed in rodents and possibly responsible for the chemical-induced tumours in the liver, were also observed in human cells in vitro (see also **Genotoxicity**) (IARC, 2012).

Acute Toxicity

Oral

Based on results from a rat study, the chemical has moderate acute oral toxicity, warranting hazard classification.

A gavage study on Sprague Dawley (SD) rats (n = 2/sex/dose) given doses of the chemical at 600–3038 mg/kg bw reported a median lethal dose (LD50) of 1180 mg/kg bw. Observed sub-lethal effects included hypoactivity, ruffled fur, diuresis, slight muscular weakness, laboured breathing, haemorrhagic rhinitis and prostration (Beroza et al., 1975).

Dermal

The chemical has low dermal toxicity based on results following dermal exposure in a rat study.

In a study in SD rats (n = 2/sex/dose), a dermal LD50 of >2025 mg/kg bw (the maximum dose administered) was established. No signs of toxicity were reported, except for local skin reactions at the end of the 24-hour contact period (Beroza et al., 1975).

Inhalation

No data are available.

Corrosion / Irritation

Skin Irritation

The chemical is not a skin irritant.

In a primary skin irritation study, 0.5 mL of the undiluted chemical was applied to the intact and abraded skin of New Zealand White rabbits (n = 6) for 24 hours. No irritation was observed, with an average primary irritation score of 0.9, below the cut-off of 5 (Beroza et al., 1975).

Eye Irritation

The chemical is not an eye irritant.

In a primary eye irritation study, 0.1 mL of the undiluted chemical was instilled into the conjunctival sac of the right eye of six New Zealand White rabbits and the animals were observed for seven days. No eye irritation was observed (Beroza et al., 1975).

Sensitisation

Skin Sensitisation

The available information indicates that the chemical is not a skin sensitiser.

In a guinea pig sensitisation test, 10 female Hartley albino guinea pigs were exposed to the chemical at a 10 % concentration for 48 hours. This process was repeated three times a week for two weeks (induction phase). Two weeks after the end of induction, doses of 0.1 % and 1 % of the chemical were applied for 48 hours (challenge phase). The chemical did not elicit any sensitisation reaction (Itoh, 1982).

Observation in humans

At an 8 % concentration, the chemical is not considered to be a skin sensitiser.

A maximisation test conducted in 25 volunteers showed that the dermal application of the chemical at 8 % in petrolatum did not cause skin sensitisation, when observed 48 hours after exposure (HSDB).

A patch test conducted in 218 fragrance-sensitive volunteers resulted in 1.8 % positive reactions to the chemical at a 5 % concentration (Health Canada, 2010).

Repeated Dose Toxicity

Oral

Repeated oral exposure to the chemical is not considered to cause serious damage to health. However, studies in rats and mice indicated that the chemical was specifically toxic to the liver and the glandular stomach at high doses. This could be due to the high lipophilicity of the chemical and rapid absorption through the stomach (NTP, 2000).

In a 14-week study conducted by the NTP, groups of F344/N rats (n = 10/sex/dose) received the chemical in 0.5 % methylcellulose by gavage at 0, 10, 30, 100, 300 or 1000 mg/kg bw/day, five days/week. Another group of 20 rats received only deionised water. All rats survived until the end of the study. Final mean body weights and body weight gains were significantly decreased (compared with vehicle control groups) in males at 300 and 1000 mg/kg bw/day and females in all dosed groups. Liver weights of males at the 100, 300, and 1000 mg/kg bw/day doses, females at 300 and 1000 mg/kg bw/day and testis weights of males at 1000 mg/kg bw/day were significantly increased compared with controls. At the highest dose, the chemical induced lesions in the liver (male and female rats), the testes of male rats, and the uterus of female rats. The incidence of hepatic lesions, including cytologic alteration, cytomegaly, Kupffer cell pigmentation, bile duct hyperplasia, and foci of cellular alteration, were significantly increased in rats at the 300 and 1000 mg/kg bw/day doses, and the lesions were generally more severe in males than in females. At these two doses, the incidences of atrophy (decrease in the thickness of the gastric mucosa) and chronic inflammation of the mucosa of the glandular stomach (consisting of fibrosis and a diffuse infiltration of the lamina propria by lymphocytes, neutrophils and macrophages) were also significantly increased. The incidence of cortical hypertrophy of the adrenal cortex was significantly increased in males at the 100, 300, and 1000 mg/kg bw/day dose and in females at the 1000 mg/kg bw/day dose. No NOEL (no observed effect level) was identified (NTP, 2000).

In a 14-week study in B6C3F1 mice (n = 10/sex/dose), the chemical in 0.5 % methylcellulose was administered by gavage at doses of 0, 10, 30, 100, 300 or 1000 mg/kg bw/day, five days/week. A group of 20 mice received only deionised water. At the highest dose, all mice except one male died before the end of the study. The mean body weight gains of males and females in the 300 mg/kg bw/day groups were significantly less than those of the vehicle controls. The liver weights of males at the 30, 100, and 300 mg/kg bw/day doses and females at the 300 mg/kg bw/day dose were significantly increased compared with the controls. Hepatic lesions, including cytologic alteration, necrosis, bile duct hyperplasia, and focal subacute inflammation were reported in females at 300 mg/kg bw/day and were significantly increased in males and females at 1000 mg/kg bw/day. The incidences of atrophy, degeneration, necrosis, oedema, mitotic alteration and cystic glands of the glandular stomach were significantly increased in one or more groups of male and female mice administered the chemical at 30 mg/kg bw/day or greater, compared with the controls (NTP, 2000).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

The available data indicate that the chemical can damage DNA, warranting hazard classification.

The following in vitro genotoxicity data were available for the chemical without further details (NTP, 2000; Health Canada, 2010; IARC, 2012):

- negative results in several bacterial gene mutation tests, including in strains of *Salmonella typhimurium*, with or without metabolic activation and *Escherichia coli* WP2uvrA, with metabolic activation;
- DNA damage induced in a rec assay in *Bacillus subtilis*, without metabolic activation;

- negative results in a chromosome aberration test using Chinese hamster ovary (CHO) cells;
- positive results in a sister chromatid exchange (SCE) assay using CHO cells with metabolic activation;
- positive results in an unscheduled DNA synthesis (UDS) test in rat hepatocytes; and
- positive results for formation of DNA adducts in rat and human hepatocytes.

The following in vivo genotoxicity data were available for the chemical without further details (NTP, 2000; Health Canada, 2010):

- increased mutations on the *lacI* gene in the liver of female Big Blue® rats which received the chemical at 1000 mg/kg bw/day for 90 days in a gene mutation test;
- no induction of mutations on the *lacI* gene in the liver of male Big Blue® rats administered the chemical at 300 mg/kg bw/day for 90 days;
- no micronuclei formation in the bone marrow cells of B6C3F1 mice treated with the chemical;
- induction of DNA adducts in the liver of CD-1 female mice treated with the chemical by intraperitoneal (i.p.) injection at 100 or 500 mg/kg bw;
- induction of DNA adducts in the liver of newborn male B6C3F1 mice treated with i.p. doses of the chemical at 0.25 to 3.0 µmol on days 1, 8, 15 and 22 after birth.

Specific mutations (base substitution) in the β-catenin gene in liver tumours of mice treated with the chemical were reported (69 % compared with only 9 % in spontaneous liver tumours of mice). However, 'a relatively high frequency of β-catenin gene mutations in mouse tumours induced by a variety of other chemicals' was also found and this gene was reported to be 'frequently mutated in human liver tumours' (IARC, 2012).

Carcinogenicity

Based on the available rodent data, the chemical is considered to be carcinogenic, warranting hazard classification. As the metabolic pathways for the chemical are considered similar in rats and humans, the chemical is expected to be a carcinogen in humans.

The IARC has classified the chemical as ‘Possibly carcinogenic to humans’ (Group 2B), based on sufficient evidence for carcinogenicity in animal testing (IARC, 2012). The NTP concluded that the chemical is 'reasonably anticipated to be a human carcinogen' (NTP, 2014).

In a 105-week gavage study conducted by the NTP, groups of F344/N rats (n = 50/sex/dose) received doses of the chemical in 0.5 % methylcellulose at 37, 75 or 150 mg/kg bw/day, five days a week. The control groups of 60 male and female rats received only the 0.5 % methylcellulose vehicle. Another group of 120 rats (n = 60/sex) (stop-exposure group) received the chemical at 300 mg/kg bw/day for 52 weeks and then the 0.5 % methylcellulose vehicle for another 53 weeks. The chemical significantly increased the incidence of rare benign and malignant neuroendocrine tumours of the glandular stomach in both sexes (at 75 and 150 mg/kg bw/day in females and at 150 mg/kg bw/day in males, and in the stop-exposure group). In the liver, incidences of hepatocholangioma and hepatocholangiocarcinoma were significantly increased in the stop-exposure group. In the kidneys of male rats, there was a significantly increased incidence of renal tubular adenoma reported for the doses of 75, 150 and 300 mg/kg bw/day. In the stop-exposure group, increased incidences of oval cell hyperplasia and hepatocellular atrophy were observed in rats aged six and 12 months, and persisted until the end of the study, suggesting that the chemical caused irreversible effects in the liver that continued to develop even after the exposure was discontinued (NTP, 2000).

The NTP also conducted a 105-week gavage study with groups of B6C3F1 mice (n = 50/sex/dose) receiving the chemical in 0.5 % methylcellulose at 0, 37, 75 or 150 mg/kg bw/day, five days a week (NTP, 2000). In all treated groups, the incidence of hepatocellular adenoma and/or combined carcinoma was significantly increased. Hepatocellular carcinoma was significantly increased in all treated females and in males at the 37 and 75 mg/kg bw/day doses. Hepatoblastoma incidence was increased in both sexes, but the increase was significant only in treated females. The chemical also induced tumours of the glandular stomach in both sexes at the highest dose, but this finding was not statistically significant (NTP, 2000; IARC, 2012).

The metabolism of the chemical leading to the formation of DNA-reactive intermediates was considered responsible for liver tumour formation (IARC, 2012; NTP, 2014). A study in male B6C3F1 mice receiving i.p. injections of 1'-hydroxymethyleugenol (a metabolite of the chemical) showed increased incidence of hepatomas (hepatocellular adenomas) (IARC, 2012).

The IARC (2012) stated that 'the doses used in the rodent studies result in the metabolism of methyleugenol by specific CYPs that leads to the formation of high levels of 1'-hydroxymethyleugenol, which can form a reactive carbonium ion. This could then result in DNA damage, as indicated by the DNA adducts detected in human hepatocytes *in vitro* and in the liver of rats *in vivo*. Mutations have been found in genes such as β-catenin, which alters expression of the Wnt pathway. These effects, together with altered expression of other genes involved in apoptosis and other pathways, could then result in the liver tumours observed in rodent studies. Alterations in these pathways also appear to occur in humans. Thus, there is moderate evidence that a mutational mechanism underlies the formation of methyleugenol-induced tumours in rodents'. As the enzymatic pathways for the metabolism of the chemical are similar in rodents and humans, 'the mechanistic data provide some additional support for the relevance of animal carcinogenicity data to humans' (IARC, 2012).

Reproductive and Developmental Toxicity

The available data indicate that the chemical is not expected to cause developmental toxicity. Only limited data are available on reproductive toxicity. In repeated dose toxicity studies in rats and mice, some significant effects on the histology and weight of reproductive organs were reported, but mostly at

high doses.

In a developmental toxicity study conducted for the NTP, female CD rats were administered the chemical by gavage (in 0.5 % aqueous methylcellulose) at doses of 80, 200 or 500 mg/kg bw/day during gestation days (GD) 6–19. No treatment-related maternal deaths were reported in the study. Treatment-related maternal effects included significantly increased liver weights at all doses and an aversion to dosing at the 80 mg/kg/day dose and above, leading to a lowest observed adverse effect level (LOAEL) of 80 mg/kg bw/day for maternal toxicity. Confirmed pregnancy rates were 72–92 % per dose. The average foetal body weight per litter was significantly reduced at the 500 mg/kg bw/day dose (86 % of the average control value). Foetal morphological anomalies, except for a significant increase in the incidence of unossified sternebrae at the highest dose, were similar between treated and control groups. Based on the results, an NOAEL of 200 mg/kg bw/day for developmental toxicity was established (NTP, 2004).

In a 14-week repeated dose toxicity study in F344/N rats (see **Repeat dose toxicity: Oral**), no significant differences in sperm motility or vaginal cytology parameters were observed between treated and control groups. The only effects related to fertility observed during the study were increased incidence of moderate dilatation of the seminiferous tubules and testicular degeneration (characterised by diffuse loss of spermatogenic cells within the seminiferous tubules) in male rats at the 1000 mg/kg bw/day dose and mild uterine atrophy in females at the 300 and 1000 mg/kg bw/day dose (NTP, 2000).

In another 14-week repeat dose toxicity study in B6C3F1 mice (see **Repeat dose toxicity: Oral**), no significant effects in sperm motility or vaginal cytology parameters were observed. However, male mice administered the chemical at 10 or 30 mg/kg bw/day had significantly lower weights of cauda epididymis, epididymis and testis, compared with the vehicle controls. Males had significantly decreased spermatozoal concentrations at the 100 mg/kg bw/day dose (NTP, 2000).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- systemic long-term effects (carcinogenicity and mutagenicity); and
- systemic acute effects from oral exposure.

Public Risk Characterisation

Given the use identified for the chemical, it is unlikely that the public will be exposed. However, as a naturally occuring substance in a number of plants, the chemical could be found in natural foodstuffs (NDPSC, 2005).

Although use in cosmetic or domestic products in Australia is not known, the chemical is permitted for use in cosmetic products overseas, as 'a component of plant extracts only' (Health Canada).

The chemical is listed on Schedule 6 of the SUSMP. A number of warning statements, first aid instructions and safety directions relating to the chemical apply for formulations containing $\geq$1 % of the chemical. The current controls are considered adequate to minimise the risk to public health posed by the use of the chemical. Therefore, the chemical is not considered to pose an unreasonable risk to public health.

Occupational Risk Characterisation

During product formulation, 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 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 of the chemical (refer to **Recommendation** section).

NICNAS Recommendation

Assessment of the chemical is considered to be sufficient, provided that the recommended 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

At present, the chemical falls within the scope of the listing of 'METHYLEUGENOL' in Schedule 6 of the SUSMP.

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)
Genotoxicity	Muta. Cat 3 - Possible risk of irreversible effects (Xn; R68)	Suspected of causing genetic defects - Cat. 2 (H341)
Carcinogenicity	Carc. Cat 2 - May cause cancer (T; R45)	May cause cancer - Cat. 1B (H350)

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

[*] Existing Hazard Classification. No change recommended to this classification

Advice for consumers

Products containing the chemical should be used according to the instructions on the label.

Advice for industry

Control measures

Control measures to minimise the risk from 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 which 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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