

# 1,2-Benzenedicarboxylic acid, bis(2-ethylhexyl) ester: Human health tier II assessment

28 June 2013

## CAS Number: 117-81-7



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## Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted

and published separately, using information available at the time, and may be undertaken at different tiers.

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

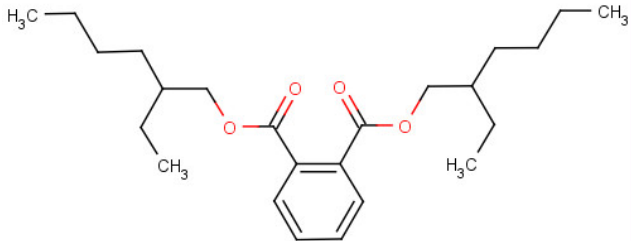
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### Acronyms & Abbreviations

## Chemical Identity

|  |  |
|--|--|
| Synonyms                               | Bis(2-ethylhexyl) phthalate<br>Dioctylphthalate<br>Di(2-ethylhexyl)phthalate<br>DEHP<br>Diethylhexyl phthalate |
| Structural Formula                     |                            |
| Molecular Formula                      | C <sub>24</sub> H <sub>38</sub> O <sub>4</sub>   |
| Molecular Weight (g/mol)               | 390.56   |
| Appearance and Odour (where available) | Oily colourless liquid at room temperature, with slight odour.   |
| SMILES                                 | C(=O)<br>(c1c(C(=O)OCC(CCCC)CC)cccc1)OCC(CCCC)C<br>C   |

## Import, Manufacture and Use

### Australian

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

The chemical was previously reported to have cosmetic use as:

- an ingredient in perfumery and cosmetic products.

The chemical has reported commercial use including:

- as a plasticiser in PVC and in other polymers for coatings, adhesives and resins for applications including:
  - flooring and waterproofing;
  - surface repair resin moulds;
  - epoxy and polyurethane products;
  - rubber components in brake assemblies;
  - hot melt adhesives for automotive assembly and repair; and
  - cable sheathing/insulation.

The following non-industrial uses have also been identified in Australia:

- as an ingredient in articles including toys and childcare articles.

The chemical is listed on the 2006 High Volume Industrial Chemicals List (HVICL) with a total reported volume between 10,000 and 99,000 tonnes per annum.

## International

The following international uses have been identified via European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) dossiers, the Organisation for Economic Cooperation and Development (OECD), European Union Risk Assessment Reports (EU RAR), Galleria Chemica, the Substances and Preparations In the Nordic countries (SPIN) database, the Household Products Database of the US National Library of Medicine, and the European Commission Cosmetic Ingredients and Substances (CosIng) database:

Cosmetic uses as:

- a masking ingredient;
- a perfuming ingredient; and
- as a solvent.

Domestic uses including:

- in lacquers, paints and printing inks; and
- in fillers and adhesives.

Commercial uses including:

- in dielectric fluids in capacitors.

The following non-industrial uses in articles have also been identified internationally:

- coatings and leather imitations (car seats, home furniture);
- shoes and boots;
- outdoor and rainwear;

- toys and child-care articles including pacifiers, teething rings and squeeze toys; and
- in medical products.

## Restrictions

### Australian

This chemical is listed in the Poisons Standard (the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)) (SUSMP, 2012) in Appendix C for cosmetic use.

Appendix C substances are poisons prohibited from sale, supply or use because of their known potential for harm to human and/or animal health.

The Australian Competition and Consumer Commission (ACCC) has banned the use of this chemical in children's products at > 1 % (ACCC, 2011). This ban prohibits supply of certain plastic products that:

- are intended for use by children up to and including 36 months of age;
- contain or have an accessible component containing > 1 % by weight of the chemical; and
- are products that children up to and including 36 months of age can readily chew and/or suck.

This ban only applies to toys, childcare articles, and eating vessels and utensils that meet each of the above criteria.

### International

EU Cosmetic Directive 76/768/EEC Annex II: List of Substances which must not form part of the Composition of Cosmetic Products.

Canada List of Prohibited and Restricted Cosmetic Ingredients (The Cosmetic Ingredient "Hotlist").

New Zealand Cosmetic Products Group Standard - Schedule 4: Components Cosmetic Products Must Not Contain.

## Existing Work Health and Safety Controls

### Hazard Classification

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

Repr. Cat. 2; R60-61 (Toxic to Reproduction).

### Exposure Standards

#### Australian

Time Weighted Average (TWA) = 5 mg/m<sup>3</sup>

Short Term Exposure Limit (STEL) = 10 mg/m<sup>3</sup>

## International

USA National Institute for Occupational Safety and Health (NIOSH):

TWA = 5 mg/m<sup>3</sup>

STEL = 10 mg/m<sup>3</sup>

United Kingdom Workplace Exposure Limits:

TWA = 5 mg/m<sup>3</sup>

STEL = 10 mg/m<sup>3</sup>

Canada (Quebec) Occupational Exposure Limits:

TWA = 5 mg/m<sup>3</sup>

STEL = 10 mg/m<sup>3</sup>

Canada (Ontario) Occupational Exposure Limits:

TWA = 3 mg/m<sup>3</sup>

STEL = 5 mg/m<sup>3</sup>

## Health Hazard Information

### Toxicokinetics

The chemical is rapidly and almost completely absorbed following oral or inhalation exposure. A bioavailability of 100 % is assumed for these routes. In contrast, bioavailability via dermal absorption is not likely to exceed 5 % (NICNAS, 2010).

Once ingested, the chemical is rapidly hydrolysed to monoethylhexyl phthalate (MEHP) and 2-ethylhexanol (2-EH). The chemical is also excreted mainly as these metabolites, along with a small amount of the parent compound, via urine and faeces.

It is reported that the chemical, most likely via some of its metabolites, can cross the placental barrier in pregnant rats and mice following oral administration.

The liver, kidney, testes and blood have been reported as the main sites of distribution of the chemical following oral administration in rats and monkeys. In mice intravenously injected with the chemical, rapid distribution was detected in the gall bladder, intestine, urinary bladder, liver, kidney and brown fat. No evidence of accumulation of the chemical or its metabolites in animal tissues was reported.

### Acute Toxicity

#### Oral

The chemical was reported to have low acute toxicity via the oral route. The median lethal dose (LD50) in rats and mice is > 20000 mg/kg bw (NICNAS, 2010).

#### Dermal

The chemical was reported to have low acute toxicity via the dermal route. The LD50 in rabbits is 24750 mg/kg bw (NICNAS, 2010).

## Inhalation

The chemical was reported to have low acute toxicity via inhalation, The median lethal concentration (LC50) in rats is > 10.62 mg/L (NICNAS, 2010).

## Corrosion / Irritation

### Respiratory Irritation

Data are insufficient to determine the respiratory irritation potential of the chemical (NICNAS, 2010). No studies specifically addressing this issue have been found. One acute toxicity study included examination of the lungs. Exposure to 10 mg/L of the chemical for four hours induced dark red foci and patches in the lungs of 19 out of 31 rats. It is unknown if these effects were reversible.

### Skin Irritation

The chemical is reported to cause minimal skin irritation in rabbits (NICNAS, 2010).

Two skin irritation studies in rabbits were performed according to OECD TG 404 and another irritation study in rabbits was performed according to the US Food and Drug Authority (FDA) recommended methods. In the first study, no erythema or oedema was observed. In the second study, very slight erythema were observed in all rabbits that persisted for 48 hours. In one rabbit, this progressed to a well-defined erythema. All reactions were reversible. The report concluded that the chemical was a slight skin irritant. The third study reported that the chemical caused mild to moderate skin irritation at 24 hours after application in an unknown number of animals. Reactions were reversible.

### Eye Irritation

The chemical is reported to cause minimal eye irritation in rabbits (NICNAS, 2010).

Two eye irritation studies in rabbits were performed according to OECD TG 405 and another eye irritation study in rabbits was performed according to US FDA recommended methods. No reaction was observed in the cornea or iris in any of the studies. All three studies reported mild conjunctival redness after one hour. In the earlier study, mild conjunctival redness was observed in five eyes, one hour after dosing and in three eyes, 24 hours after application. Where reported, all reactions resolved at later timepoints.

## Sensitisation

### Respiratory Sensitisation

Data are insufficient to determine the respiratory sensitisation potential of the chemical (NICNAS, 2010).

An in vitro study provided limited support linking the chemical with respiratory hyperresponsiveness. The chemical's metabolite, MEHP, provoked reversible hyperresponsiveness to methacholine in rat tracheal tissue, which was not observed with the chemical. The authors concluded that continuous exposure to the chemical might cause bronchial hyperresponsiveness.

### Skin Sensitisation

The chemical is not a skin sensitiser in guinea pigs (NICNAS, 2010).

Two skin sensitisation studies in guinea pigs, one using the Magnusson-Kligman maximisation test protocol and another using the Buehler test protocol, reported no positive reactions.

## Repeated Dose Toxicity

### Oral

The chemical has been tested for repeated dose effects via the oral route in many studies particularly in the rat but also in the mouse and marmoset monkey. Adverse effects were reported in the liver, testes and kidney (NICNAS, 2010).

Liver hypertrophy, increased liver weights and peroxisome proliferation were noted in most of the repeated dose studies. However, it is noted that liver effects due to peroxisome proliferation in rodents are not considered relevant to humans.

In a 104-week dietary study F344 rats (70/sex/group) were fed the chemical at dose levels up to 12500 ppm (789 mg/kg bw/day and 938.5 mg/kg bw/day, for males and females respectively). Hepatotoxicity (significant increases in serum albumin, absolute and/or relative liver weights and peroxisome proliferation) was observed in both sexes at  $\geq 2500$  ppm (146.6/181.7 mg/kg bw/day calculated for male/female body weight). The no observed adverse effect level (NOAEL) was reported to be 28.9 mg/kg bw/day for males and 36.1 mg/kgbw/day for females.

Testicular effects such as decreased weights, testicular atrophy, increased bilateral aspermatogenesis, immature or abnormal sperm forms, seminiferous tubular degeneration, Sertoli cell vacuolation or complete loss of spermatogenesis were also evident in most of the repeated dose studies.

Effects on the kidneys following repeated treatment with the chemical included increases in kidney weights, mineralisation of renal papillae, tubule cell pigments and chronic progressive nephropathy. The majority of these changes were seen in both sexes across different species in studies of varying durations.

### Dermal

In the only available dermal study, 0.2 mL of 10, 30, 50, or 100% of the chemical in olive oil was administered percutaneously to mice for one month (NICNAS, 2010). Macroscopically, the liver was greatly enlarged. Inflammatory signs were observed in the peritoneum in the two highest dose groups. Hepatic cells showed atrophied nuclei and frequently contained fat droplets. The authors concluded that the chemical is absorbed and accumulates in the liver. This study was considered to have several limitations.

### Inhalation

A number of inhalation studies in experimental animals were identified (NICNAS, 2010).

In one study, rats were exposed to the chemical at doses of up to 1000 mg/m<sup>3</sup> for six hours per day, five days per week for four weeks. In the highest dose group, there was a significant increase in relative lung weights in male rats accompanied by foam cell (macrophage) proliferation and thickening of the alveolar septa. Liver weights were slightly increased but not accompanied by peroxisome proliferation that had been reported to be observed in a similar range-finding study. No testicular toxicity was detected histologically.

In another study, four week old male Wistar rats (four per group) were exposed to the chemical at doses of 0, 5 or 25 mg/m<sup>3</sup> for six hours per day, for four or eight weeks. There were no differences in body or testes weights. Seminal vesicle weight was reduced in the eight week exposure groups but not in the four week exposure groups at both doses. Histological examination showed no significant pathological changes in the testes after four or eight week's exposure to either dose. The study did not show a dose-response relationship.

## Genotoxicity

The genotoxicity of the chemical has been reviewed extensively (NICNAS, 2010) and has been tested in a variety of short-term genotoxicity assays with predominantly negative results. Overall, the chemical is regarded as non-genotoxic.

### *In vitro*

In a review of 15 published reverse mutation assays in bacteria, all results were reported as negative. The maximum concentration used was 14700 µg/plate. Two studies in yeast were negative, failing to show any evidence of mutation or recombination events. Primary DNA damage, mutation, sister chromatid exchange or chromosomal aberrations were not induced in most assays with cultured mammalian cells. Some of these *in vitro* systems are also sensitive to non-genotoxic substances which are tumour promoters and/or peroxisome proliferators.

### *In vivo*

Results were generally negative in *in vivo* studies (mouse, rat and *Drosophila melanogaster*) using the chemical and its main metabolites MEHP and 2-EH. Low levels of mutation but not DNA damage were induced in somatic cells of *Drosophila melanogaster*. Gene mutations were not induced *in vivo* in the liver of dosed mice and there was no evidence of chromosomal aberrations in mice or rats *in vivo*.

## Carcinogenicity

There is sufficient evidence to classify the chemical as a potential carcinogen.

Lifetime dietary exposures to the chemical were associated also with dose-dependent increases in the incidence of Leydig cell tumours in some rat studies (NICNAS, 2010). In mice and rats, the chemical also induced significant dose-dependent increases in the incidence of hepatocellular tumours. At low doses, there was no evidence of liver toxicity or increase in hepatocellular tumours, suggesting a threshold for this effect. The lowest observed adverse effect level (LOAEL) and the NOAEL for tumour induction in rats were established as 146.6 mg/kg bw/day and 28.9 mg/kg bw/day, respectively. In mice, the LOAEL and the NOAEL for induction of liver tumours were 292 mg/kg bw/day and 98 mg/kg bw/day, respectively. As per repeat dose toxicity, it is noted that liver effects associated with peroxisome proliferation in rodents are not considered relevant to humans.

In a single lifetime dietary study with Sprague Dawley (SD) rats, the chemical was associated with an increased incidence of Leydig cell tumours. In this study, the NOAEL for both hepatic tumours and testicular tumours was determined to be 95 mg/kg bw/day. However, the dose-related increase in Leydig cell tumours was observed commencing from the lowest dose of 30 mg/kg bw/day. Leydig cell tumours were not reported in other studies with F344 rats even at higher doses. Notably, spontaneous Leydig cell tumours are not common in SD rats in contrast to F344 rats. The chemical does not appear to induce testicular neoplasia in B6C3F1 mice.

The chemical has been classified by the International Agency for Research on Cancer as a Group 2B Carcinogen: 'Possibly carcinogenic to humans' (IARC, 2012) and by the United States National Toxicology Program's Report on Carcinogens as 'Reasonably anticipated to be a human carcinogen' (NTP RoC, 2011).

## Reproductive and Developmental Toxicity

The chemical is currently classified as hazardous as a Category 2 reproductive toxin with the risk phrases 'May impair fertility' (T; R60) and 'May cause harm to the unborn child' (T; R61) in HSIS (Safe Work Australia). The available data support these classifications.

Multigenerational studies with rodents reveal adverse reproductive effects of the chemical manifesting as decreased fertility and adverse developmental effects on progeny (NICNAS, 2010).

In a three-generational dietary study in rats, a LOAEL of 14 mg/kg bw/day was established for male developmental toxicity based on decreased testes weight and seminiferous tubule atrophy in F1 and F2 generations. The NOAEL in this study is 4.8 mg/kg bw/day. At higher doses, decreased *in utero* survival, reduced anogenital distance, undescended testes, retained



nipples/areolae, incomplete preputial separation and disruption of spermatogenesis were also observed in F1 and F2 generations.

Consistent observations of reproductive effects of the chemical have been demonstrated in both male and female rodents. Overall, studies support a NOAEL for fertility and developmental effects of the chemical in the dose range of 1–10 mg/kg bw/day. In addition, data on mode of action indicate the chemical affects steroidogenesis and expression of genes critical for reproductive system development common to both rodents and humans, suggesting that the reproductive toxicity effects of the chemical seen in rodents are relevant for humans.

## Other Health Effects

### Endocrine Disruption

Biochemical studies in rodents reveal association of exposure to the chemical with alterations in Leydig cell steroidogenesis, serum levels of testosterone and luteinizing hormone (LH), and expression of genes crucial for development of the male reproductive system (NICNAS, 2010).

A LOAEL of 10 mg/kg bw/day was established based on increased serum LH and testosterone levels in rats exposed to the chemical for 28 days during postnatal days 21–48. The NOAEL for these biochemical alterations is 1 mg/kg bw/day.

## Risk Characterisation

### Critical Health Effects

The main critical effects to human health are reproductive and developmental toxicity, with a potential endocrine disruption mechanism, and carcinogenicity. The chemical may also cause adverse systemic effects following repeated exposure.

### Public Risk Characterisation

The risk to adults and children from direct exposure to the chemical in consumer applications (such as adult cosmetics, mouthing of children's toys and child care articles) has been evaluated by NICNAS (2010). There are controls in place on these uses which aim to reduce this exposure to acceptable levels.

The use of the chemical in cosmetics is restricted in Australia, as listed in the Poisons Standard (SUSMP, 2012). The chemical is also banned for use in children's products at > 1 % (ACCC, 2011).

Although there is no reported use of the chemical in domestic products in Australia, indirect exposure to the chemical can occur via dermal contact of consumer products containing the chemical (for example shower curtains and vinyl tiles), exposure to indoor air and/or dust containing the chemical, and also via food contact materials.

Direct dermal exposure, for example from a child crawling on floor tiles, is expected to be low; dermal contact was found to result in very low exposure compared with oral intake in the mouthing scenario, due to the low rate of transdermal flux from plastic articles (NICNAS, 2010).

Estimated exposure levels to the chemical have been determined for indoor air and dusts (mean value calculated to be 64 µg of the chemical ingested per day; equivalent to 0.91 µg/kg bw/day based on a 70 kg adult), and also food that has been in contact with the chemical (4.9 – 18 µg/kg bw/day) (Bornehag et al, 2004; Schettler, 2006); these levels are much lower than those calculated for direct exposure to chemical where the worst case estimates were 231.7 µg/kg bw/day from mouthing toys and child care articles, and 154.7 µg/kg bw/day from cosmetic use (NICNAS, 2010).

Assessments of Margin Of Exposure (MOE) comparing these estimated indirect exposure (ingestion) levels with doses of the chemical at which no adverse reproductive and/or development, and repeat dose effects were observed (4.8 and 28.9 mg/kg

bw/day, respectively), gives MOEs of much greater than 100 indicating a low risk of adverse health effects due to these sources of indirect exposure.

## Occupational Risk Characterisation

Given the critical systemic long-term health effects, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise exposure to the chemical are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine appropriate controls.

## NICNAS Recommendation

Assessment of the chemical is considered to be sufficient provided that the recommendation is adopted for the amendment of the classification and labelling of the chemical and all requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory.

## Regulatory Control

### Public Health

Current restrictions on the use of the chemical in cosmetic products are considered sufficient. Considering that the health risks to the public from indirect exposure to the chemical are lower than for direct exposure, no additional regulatory controls are recommended.

### Work Health and Safety

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

| Hazard                                  | Approved Criteria (HSIS) <sup>a</sup>  | GHS Classification (HCIS) <sup>b</sup>                      |
|---|--|---|
| Carcinogenicity                         | Carc. Cat 2 - May cause cancer (T; R45)  | May cause cancer - Cat. 1B (H350)                           |
| Reproductive and Developmental Toxicity | Repro. Cat 2 - May impair fertility (T; R60)*<br>Repro. Cat 2 - May cause harm to the unborn child (T; R61)* | May damage fertility or the unborn child - Cat. 1B (H360FD) |

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

<sup>b</sup> 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 label instructions.

## Advice for industry

### **Potential for substitution**

There are many alternative plasticisers available for specific uses, and there has been ongoing international research into alternatives to this chemical. Considering the health effects, it is recommended that companies evaluate safer, viable alternatives to the chemical in products where exposure is expected to be high (notably consumer products).

### **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 may 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;
- air monitoring to ensure control measures in place are working effectively and continue to do so;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the 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 assist with meeting obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((m)SDS) containing accurate information about the hazards (relating to both health 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.

## References

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