Methanone, diphenyl-: Human health tier II assessment

01 September 2015

CAS Number: 119-61-9

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.

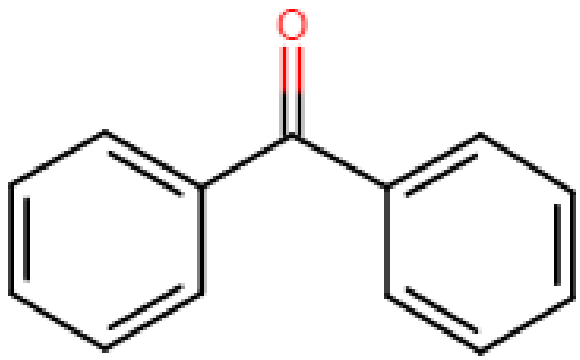
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	benzophenone diphenyl ketone benzoylbenzene
Structural Formula	
Molecular Formula	C13H10O
Molecular Weight (g/mol)	182.221
Appearance and Odour (where available)	White solid with a flowery odour
SMILES	c1(C(=O)c2ccccc2)ccccc1

Import, Manufacture and Use

Australian

No specific Australian use, import, or manufacturing information has been identified.

International

The following international uses have been identified through: the European Union (EU) Registration, Evaluation, Authorization and Restriction of Chemicals (REACH) dossiers; Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (CosIng) database; the United States (US) Personal Care Products Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary, the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and various international assessments (National Toxicology Program (NTP), 2006 and the International Agency for Research on Cancer (IARC), 2012).

The chemical has reported cosmetic uses as:

- a fragrance ingredient; and
- light stabiliser (UV absorber) in cosmetics and personal care products.

The chemical is included in the list of fragrance ingredients used in consumer goods published by the International Fragrance Association (IFRA) and has reported use in cosmetics in the United States of America (Personal Care Products Council, 2011).

The chemical has reported domestic uses, including:

- in cleaning and washing agents; and
- in paints, lacquers and varnishes.

The chemical has reported commercial uses, including as:

- a UV-curing agent in sunglasses and ink;
- an additive in plastics, coatings and adhesive formulations;
- a photo-initiator in the printing industry;
- a UV-blocker in the plastic packaging industry; and
- a process regulator.

The chemical has reported site-limited uses, including:

- in organic synthesis; and
- as a polymerisation inhibitor for styrene

The chemical has reported non-industrial uses in pharmaceuticals and insecticides.

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

The following exposure standard is identified (Galleria Chemica):

An exposure limit of 0.5 mg/m^3 time weighted average (TWA) in the United States of America.

Health Hazard Information

Toxicokinetics

The chemical can be absorbed following oral, dermal and inhalation exposure. Following oral administration, the chemical was rapidly absorbed from the gastrointestinal tract in rats. The percutaneous absorption (occluded) in rhesus monkeys was approximately 70 % within 24 hours (NTP, 2006; IARC, 2012).

In rabbits, the main metabolic pathway identified for the chemical following dietary administration is the reduction of the keto group to form benzhydrol. The metabolite is then excreted in the urine (41–61 % of the administered dose) as a labile glucuronide (NTP, 2006).

In a 24–hour oral gavage study in Sprague-Dawley (SD) rats, a small amount (1 %) of the administered dose (100 mg/kg bw) was detected as 4-hydroxybenzophenone in enzyme-treated urine samples. None was detected in the faeces. The peak levels of the chemical and its metabolites were reached around four hours after dosing. In isolated Fischer 344 (F344) rat hepatocytes, at least three metabolites of the chemical were identified: benzyhydrol, and 4-hydroxybenzophenone and its sulfate conjugate (IARC, 2012; REACH).

The elimination half-life of the chemical (parent compound) is approximately 19 hours (gavage) in SD rats, 4 hours (intravenous (i.v.) injection) and 8 hours (gavage) in F344 rats, and ~one hour (i.v. injection) and 1.5 hours (gavage) in mice (NTP, 2006).

Following exposure of an aqueous solution of the chemical to UV or sunlight irradiation, 3-hydroxybenzophenone, 4-hydroxybenzophenone were observed, together with concomitant production of hydrogen peroxide. This suggests 'that benzophenone might act as a photosensitiser' to generate reactive oxygen species 'which can cause aromatic ring hydroxylation' (IARC, 2012).

Acute Toxicity

Oral

The chemical has low acute toxicity in animal tests following oral exposure.

The median lethal dose (LD50) is >2000 mg/kg bw in mice and >10000 mg/kg bw in rats (REACH).

Observed sublethal signs in mice included neurological effects (sedation, progressive depression of motor activity, unstable gait, tremors), and respiratory impairment (REACH).

Dermal

The chemical has low acute toxicity in rabbits following dermal exposure.

A dermal LD50 of >2000 mg/kg bw in rabbits was reported. The chemical was 'practically non-toxic' (REACH).

Inhalation

No data are available.

Corrosion / Irritation

Skin Irritation

Based on the weight of evidence from the available studies, the chemical may be a slight skin irritant.

In a skin irritation study conducted according to the OECD Test Guideline (TG) 404, the chemical (0.5 mL) was applied (semi-occlusively) to the shaved skin of rabbits (n = 4) for four hours, followed by observation for up to 72 hours. No signs of skin irritation were observed at concentrations up to 100 % (REACH).

Guinea pigs treated with the chemical on the abdomen (occlusively) for 24 hours or on the back (uncovered) for 10 days displayed slight skin irritation (slight erythema and desquamation, and slight to moderate oedema). No other details were reported (NTP, 2006).

In a skin irritation study using the Draize method, the chemical was slightly irritating to rabbit skin (exposure time not reported), with a primary irritation index of 2.0. However, no skin irritation was observed in studies conducted in guinea pigs (NTP, 2006; REACH).

Eye Irritation

Based on the limited information available, the chemical could be a slight eye irritant.

In two poorly-documented eye irritation studies, the chemical caused little to no eye irritation in rabbits. The chemical was administered as solid crystals in one of the studies and caused only a slight reaction. No other details were available (REACH).

Sensitisation

Skin Sensitisation

Only limited data are available. Based on the available guinea pig test data, the chemical did not show skin sensitisation properties up to 10 % concentration. Some benzophenone derivatives are reported to induce skin allergy and photoallergy especially when used in sunscreens and other personal care products (see **Sensitisation – Observations in Humans**). Benzophenones as a group were named the Contact Allergen of the Year for 2014 by the American Contact Dermatitis Society to raise awareness of both allergy and photoallergy for this class of chemicals (Heurung et al., 2014). These concerns relate to a class of hydroxylated benzophenone derivatives and it is not clear that these concerns would relate to benzophenone itself.

In a modified skin sensitisation test, guinea pigs were intradermally exposed to the chemical (0.1 mL) at 1 % concentration (similar to the Draize test). No topical induction was performed. This was followed with a combined intradermal (0.25 %) and topical (20 %) challenge exposure on day 14. Challenging was repeated on days 35 and 42 due to absence of sensitisation on day 14. No skin sensitising effects were observed (REACH).

In a Magnusson-Kligman skin sensitisation test, guinea pigs were initially administered the chemical intradermally at 1 % concentration, followed by topical application at 10 % concentration for 48 hours. The skin was challenged two weeks after dermal induction with topical applications at concentrations of 1 and 5 %. The chemical was reported to be negative in this test (REACH).

Observation in humans

Some benzophenone derivatives in sunscreen products are reported to cause allergic skin reactions in humans (Heurung et al., 2014; Hanson & Warshaw, 2015). However, no available information indicates that the same effects result from exposure to benzophenone.

Repeated Dose Toxicity

Oral

Based on the available data, the chemical is not considered to cause severe effects following repeated oral exposure.

Animal studies showed that the liver and kidney were the primary target organs for the toxicity of the chemical. Rats were more sensitive than mice to the chemical, with effects observed at ≥75 mg/kg bw/day.

In a repeated dose toxicity study (OECD TG 408), rats (n = 10/sex/dose) were administered the chemical in the diet at concentrations of 0, 1250, 2500, 5000, 10000 or 20000 ppm (males: 75–850 mg/kg bw/day, females: 80–1000 mg/kg bw/day), for 14 weeks. The chemical was unpalatable at the highest dose (20000 ppm) and the rats in that dose group were terminated on days 39 or 40 due to significant weight loss. Body weights for both males (≥2500 ppm) and females (all treated groups) were significantly lower than the controls. Liver toxicity was observed as treatment-related increases in liver weights attributed to centrilobular hypertrophy and cytoplasmic vacuolisation (all treated groups), clinical chemistry changes (increased alanine aminotransferase and bile salt concentrations at ≥10000 ppm), and induction of liver microsomal cytochrome P450 2B isomer (≥2500 ppm for males and ≥1250 ppm for females). Increased kidney weights were associated with various renal lesions including tubule dilatation (≥2500 ppm for males and ≥10000 ppm for females), tubule epithelial regeneration (all treated groups), mineralisation and necrosis in the renal papillae (≥10000 ppm for males and ≥20000 ppm for females). Based on these observations, a no observed adverse effect level (NOAEL) could not be determined (NTP, 2006; REACH).

In a repeated dose toxicity study (OECD TG 408), mice were administered the chemical in the diet at the same concentrations as in the rat study for 14 weeks. Equivalent doses were 200–3300 mg/kg bw/day in males and 270–4200 mg/kg bw/day in females. At the highest dose, all males and four females died or were sacrificed moribund. Reduced body weights were observed at 10000 ppm for males and ≥5000 ppm for females. Compared to rats, mice were less sensitive to the toxicity of the chemical. Significant observations were limited to the liver. Increased liver weights in all exposed groups were attributed to significantly increased incidences of centrilobular hypertrophy and/or cytoplasmic vacuolisation of hepatocytes. Male mice exhibited significantly greater liver cytochrome P450 in all treated groups, decreased testes and epididymis weights at 10000 ppm, and anaemia at ≥5000 ppm. A NOAEL could not be determined (NTP, 2006; REACH).

In another repeated dose oral study, SD rats were administered (in diet) the chemical at 20 mg/kg bw/day for 90 days, or 100 or 500 mg/kg bw/day for 28 days. After four weeks of treatment, significantly increased mean absolute and relative liver weights and increased absolute and relative kidney weights (statistically not significant) were observed at ≥100 mg/kg bw/day. Histopathological examination of the liver revealed hepatocellular enlargement with associated clumping of cytoplasmic basophilic material around the central vein. Other treatment-related effects observed were significant changes in erythrocyte counts, haemoglobin, haematocrit, total protein and albumin concentrations at ≥100 mg/kg bw/day. The NOAEL was established as 20 mg/kg bw/day for 90 days (REACH).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

Based on the data available, the chemical is not considered to be genotoxic.

The chemical gave negative results in several in vitro assays listed below (NTP, 2006; REACH):

- bacterial reverse mutation assays in several strains of *Salmonella typhimurium* at concentrations up to 1000 µg/plate, with or without metabolic activation;
- bacterial reverse mutation assay which is used for the detection of oxidative mutagenicity (WP2 Multoxitest,) in several *Escherichia coli* strains;
- unscheduled DNA synthesis (UDS) in *E. coli* strains W3110 (pol A+) and P3478 (pol A-); and
- mammalian gene mutation assay using mouse lymphoma L5178Y cells.

The chemical and its metabolites did not induce DNA damage measured by *umu* gene expression in *S. typhimurium* strain TA1535 in the absence or presence of rat, mouse or human liver microsomes. However, *umu* gene expression was elicited with the addition of various recombinant human cytochrome P450 preparations (NTP, 2006).

Two in vivo micronucleus assays gave negative results with the chemical (IARC, 2012; REACH):

- a bone marrow micronucleus test in two strains of mice injected intraperitoneally (i.p.) with the chemical (at 500, 1000 or 2000 mg/kg bw in CBA mice and at 100, 250, 400 or 600 mg/kg bw in NMR1 mice); and
- a bone marrow micronucleus test in male B6C3F1 mice administered the chemical (i.p.) at doses up to 500 mg/kg bw.

Carcinogenicity

Based on the available data, the chemical is potentially carcinogenic and warrants hazard classification.

After evaluating the available data in animals, IARC (2012) classified the chemical as '*possibly carcinogenic to humans (Group 2B)*' (IARC, 2012). IARC concluded that 'There is *sufficient evidence* in experimental animals for the carcinogenicity of benzophenone'.

The National Toxicology Program (NTP) concluded that there were '*some evidence of carcinogenic activity*' in male rats, '*equivocal evidence of carcinogenic activity*' in female rats, and '*some evidence of carcinogenic activity*' in both male and female mice. Kidney tumours in male rats, histiocytic sarcomas in female mice and hepatoblastomas in male mice were considered rare spontaneous neoplasms (NTP, 2006).

In a two year carcinogenicity study (OECD TG 451), groups of B6C3F1 mice (n=50/sex/dose) were administered the chemical in the diet at concentrations of 0, 312, 625 or 1250 ppm (males: 0, 40, 80 or 160 mg/kg bw/day; females: 0, 35, 70 or 150 mg/kg bw/day). In males, a positive trend in the incidence of combined hepatocellular neoplasms (primarily adenomas) were observed in all exposure groups, and incidences at ≥625 ppm were significantly greater than the controls. In females, the incidence of hepatocellular adenoma was observed at ≥625 ppm but was not significantly different from controls. The incidence of histiocytic sarcoma in females was significantly increased at ≥625 ppm. Other observations in both sexes include significantly increased incidence of metaplasia of the olfactory epithelium (1250 ppm) and significantly increased hyperplasia of lymphoid follicles in the spleen of all treated males, and in females at 312 and 625 ppm (NTP, 2006; IARC, 2012; REACH).

In this study, F344/N rats (n=50/sex/dose) were fed diets containing the chemical at doses of 0, 312, 625 or 1250 ppm (males: 0, 15, 30 or 60 mg/kg bw/day; females: 0, 15, 30 or 65 mg/kg bw/day). In males, some evidence of carcinogenicity was observed based on significantly increased incidences of renal tubule adenoma (≥625 ppm) accompanied by increased incidences of renal tubule hyperplasia, increased pelvic transitional epithelium hyperplasia (all exposed groups) and mononuclear cell leukaemia (at 312 and 625 ppm). In females, there was equivocal evidence of carcinogenicity based on increased incidences of mononuclear cell leukaemia (all exposed groups) and rare histiocytic sarcomas in three rats (one at 625 ppm and two at 1250 ppm). The incidences of mononuclear cell leukemia in both sexes exceeded the range for historical controls from dietary studies. Non-neoplastic liver effects included significantly increased incidences of centrilobular hepatocellular hypertrophy (all exposed groups), cystic degeneration of hepatocytes (≥625 ppm males), and bile duct hyperplasia (all exposed females) (NTP, 2006; IARC, 2012).

The chemical gave negative results in dermal carcinogenicity studies at concentrations up to 50 % applied to the skin of Swiss mice and New Zealand White rabbits, up to 160 weeks of treatment (EFSA, 2009).

Reproductive and Developmental Toxicity

Based on the available data, the chemical is not considered to have reproductive or developmental toxicity. Some developmental effects were observed in SD rats and New Zealand White rabbits, secondary to maternal toxicity.

In a two-generation reproductive and developmental toxicity study (OECD TG 416), groups of SD rats (n=24/sex/dose) were administered the chemical in the diet at concentrations of 0, 100, 450 or 2000 ppm (~0, 6.4–8.8, 5.6–15.5, 130–179 mg/kg bw/day). The F0 generation was exposed at five weeks old, continuing for 10 weeks for males and until postnatal day (PND) 21 for females. The effects observed in F0 and F1 parental animals included reduced body weight gain and food consumption at ≥450 ppm, kidney effects at doses ≥450 ppm (increased renal weights, dilatation of the renal proximal tubules and regeneration of the proximal tubular epithelium), liver effects at ≥100 ppm (increased hepatic weight and centrilobular hepatocytic hypertrophy). No treatment-related effects in the oestrous cycle, reproductive capability, delivery and lactation, sperm parameters, and serum hormone levels were observed. For the offspring, reduced body weight gain in both F1 and F2 rats were observed at 2000 ppm. No treatment-related effects on litter size (F1 and F2 pups), viability, anogenital distance, and physical development were observed in the offspring. The maternal and developmental NOAELs were determined at <100 ppm and 450 ppm, respectively (REACH).

In a developmental toxicity study conducted by the NTP, SD rats (n=22-25/dose) were administered the chemical (gavage) at doses of 0, 100, 200 or 300 mg/kg bw/day from gestation day (GD) 6-19. No treatment-related maternal deaths were observed. Maternal toxicity was observed in all treated groups, and included neurotoxicity (lethargy and piloerection), weight loss, and increased maternal liver and kidney weights. At the highest dose, significantly reduced maternal body weight gain and food consumption were observed. The chemical did not affect prenatal viability or overall incidence of foetal malformations or variations. Developmental toxicity effects observed included increased incidences of unossified sternebrae (all doses), extra rib on lumbar I (≥200 mg/kg bw/day), and reduced foetal body weight per litter at the highest dose (NTP, 2006; REACH). The maternal NOAEL was determined to be <100 mg/kg bw/day. No NOAEL for developmental toxicity could be established and the authors stated that the observed effects ‘are limited to mild developmental delays with a high probability of recovery during early postnatal development’ (NTP, 2006).

In a separate NTP developmental toxicity study, New Zealand White rabbits (n=24/dose) were administered the chemical (gavage) at doses of 0, 5, 25 or 45 mg/kg bw/day from GD 6–29. Maternal toxicity was observed at ≥25 mg/kg bw/day, which included reduced body weight and food consumption, dose-related maternal mortality and early termination of pregnancy (abortion or early delivery). No changes were observed for the gravid uterus, liver or kidney weights. Developmental toxicity effects included significantly reduced average foetal weight per litter at the highest dose. No adverse effects on prenatal viability or incidences of foetal morphological anomalies among litters were observed. The authors stated that ‘developmental toxicity

was noted only in the presence of well-defined maternal toxicity', which is a similar finding to the rat studies (NTP, 2006). The maternal and developmental NOAELs were determined to be 5 mg/kg bw/day and 25 mg/kg bw/day, respectively (NTP, 2006; REACH).

The chemical is listed in the EC Endocrine Disruptors Priority List under Category 3b classification (i.e. no evidence of endocrine disrupting activity or no data available) (EC, 2015); and the US EPA's Universe of Chemicals list for potential endocrine disruptor screening and testing (US EPA, 2012).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (carcinogenicity). Based on the available data, the chemical was carcinogenic in animal studies following oral exposure but not with dermal exposure. No animal carcinogenicity data are available through inhalation exposure.

The potential of the chemical to cross react with other benzophenone derivatives to produce photoallergic skin reactions cannot be excluded based on available data (see **Skin sensitisation**).

Public Risk Characterisation

The European Food Safety Authority (EFSA) has evaluated the carcinogenicity data for benzophenone, due to its occurrence in food and food packaging, and concluded that exposure to food with benzophenone at current levels is a low risk (EFSA, 2009). The EU Standing Committee on the Food Chain and Animal Health stated that benzophenone is suitable for use in inks and coatings for food packaging provided an effective functional barrier is present which prevents migration (EC, 2009).

For the use of the chemical as a fragrance ingredient or light stabiliser in cosmetic products, dermal exposure from cosmetic use is expected to be low compared with its use in flavouring, as assessed by EFSA. Dermal carcinogenicity studies on the chemical gave negative results (see **Carcinogenicity**) and, therefore, the risk from this use is expected to be low.

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 systemic long-term 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 (see **Recommendation**).

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]
Carcinogenicity	Carc. Cat 3 - Limited evidence of a carcinogenic effect (Xn; R40)	Suspected of causing cancer - Cat. 2 (H351)

[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 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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Last update 01 September 2015

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