Phosphine, triphenyl-: Human health tier II assessment

01 July 2016

CAS Number: 603-35-0

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.

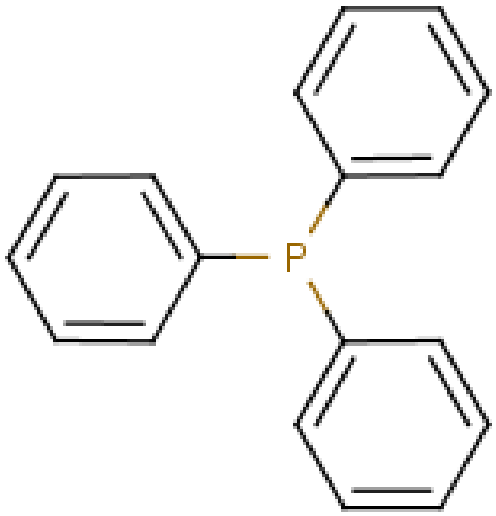
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	triphenylphosphine triphenylphosphide triphenylphosphorus triphenylphosphane TPP
Structural Formula	
Molecular Formula	C18H15P
Appearance and Odour (where available)	white odourless crystalline solid
SMILES	c1(P(c2ccccc2)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, 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; and
- the Substances and Preparations in Nordic countries (SPIN) database.

The chemical has reported domestic/commercial use in paints, lacquers and varnishes.

The chemical has reported commercial use in construction materials.

The chemical has reported site-limited uses, including as:

- a chemical intermediate for the synthesis of complexing agents, reducing agents and process regulators;
- a catalyst ligand in hydroformylation reactions of olefins (oxosynthesis);
- an additive in the synthesis of polyurethanes; and
- a polymerisation initiator.

The chemical has reported non-industrial use in the synthesis of vitamins and pharmaceuticals.

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 limits were identified for the chemical (Galleria Chemica):

- Temporary Emergency Exposure Limits (TEELs) defined by United States (US) Department of Energy (DOE):

TEEL-1 = 1.1

TEEL -2 = 1.2

TEEL-3 = 540

- German exposure limit for the chemical is 5 mL/m³ for pregnancy risk group classification Category C.
- Switzerland occupational exposure limits of 5 mg/m³ and short term limit value of 10 mg/m³.

Health Hazard Information

Toxicokinetics

There are no available toxicokinetic studies for the chemical. Based on the results obtained from animal studies, it can be assumed that the chemical may be absorbed following oral and inhalation exposures (See **Acute toxicity - Oral and Inhalation; Repeat Dose Toxicity - Oral and Inhalation**).

Absorption is assumed to be dependent on the vehicle used and the particle size of the chemical at the time of exposure (OECD, 2006).

Acute Toxicity

Oral

The chemical has low to moderate acute toxicity based on results from animal tests following acute oral exposure. In rats, the median lethal dose (LD50) by the oral route ranged from approximately 700 mg/kg bw (in olive oil) to > 6400 mg/kg bw (in aqueous suspension), depending on the vehicle used. In mice, the oral LD50 (in olive oil) was 1000 mg/kg bw. The available data suggest that the acute oral toxicity of the chemical is dependent on the vehicle used.

An acute oral toxicity study was conducted in albino rats (10/dose; unspecified sex) at doses of 250, 500, 1000 and 2000 mg/kg bw (in olive oil). No mortality was reported at 250 mg/kg bw; however, one animal died at 500 mg/kg bw and all animals died at 1000 mg/kg bw. Toxic effects observed within 1-4 days of exposure included dyspnoea, apathy and impaired balance. The LD50 was reported to be approximately 700 mg/kg bw (OECD, 2006; REACH).

In a study conducted in Sprague Dawley (SD) rats (five animals/sex/dose), the chemical was administered by oral gavage at doses of 200, 1600, 3200, 6400 mg/kg bw (in approximately 30% aqueous suspension with tragacanth). At the 14-day observation period, one male died at 6400 mg/kg bw and another male died at 3200 mg/kg bw. The animals showed transient dyspnoea and apathy. The LD50 was determined to be > 6400 mg/kg bw (OECD, 2006; REACH).

In mice, the LD50 was determined to be 1000 mg/kg bw. Laboured and rapid breathing as well as tonic-clonic seizures were reported in animals that died following oral exposure to the chemical administered in olive oil (OECD, 2006).

Oral administration of the chemical in rabbits at doses of 1000, 2000 or 4000 mg/kg bw in olive oil or aqueous carboxymethylcellulose suspension resulted in hyperexcitability, followed by atony and transient ataxia. The effects were more pronounced in animals treated with the chemical in olive oil. At 2000 mg/kg bw in aqueous suspension, the rabbits survived; however, three out of four animals died at the same dose of chemical in olive oil. These observations sugggest higher systemic bioavailability of the chemical in olive oil solution compared with the aqueous carboxymethylcellulose suspension (OECD, 2006).

In beagle dogs, the chemical was administered at doses of 300, 600, 1200 mg/kg bw in an aqueous suspension with tragacanth or in olive oil solution. No mortality was reported in two beagle dogs following a single dose of the chemical in aqueous gum tragacanth at 1200 mg/kg bw. Transient diarrhoea or soft faeces were reported. No mortalities were reported in four beagle dogs administered with the chemical at 1200 mg/kg bw in olive oil solution. However, one animal died following oral exposure to 600 mg/kg bw in olive oil solution. Transient trembling, clonic-tonic convulsions, lateral position, panting with foam expulsion were reported at doses of 600 and 1200 mg/kg bw. At 300 mg/kg bw in olive oil, vomiting, associated with diarrhoea and loss of appetite were observed (OECD, 2006; REACH).

Dermal

The chemical has low acute toxicity based on results from animals studies following acute dermal exposure (as a 50% suspension in ethanol). The LD50 was > 2500 mg/kg bw in SD rats, and > 4000 mg/kg bw in Vienna white rabbits. No systemic toxicity or local irritation was reported (OECD, 2006; REACH).

Inhalation

The chemical has low acute toxicity based on an acute inhalation study in Chr-CD rats. The LC50 was 12,500 mg/m^3 following a whole body exposure for 4 hours. Clinical signs reported were indicative of respiratory irritation (OECD, 2006; REACH).

Corrosion / Irritation

Respiratory Irritation

No data are available.

Skin Irritation

The chemical is reported to slightly irritate the skin in animal studies. The irritation effects were not sufficient to warrant hazard classification.

In a skin irritation study, the chemical (as a 50% suspension in ethanol) was applied onto the shaved back of two Vienna white rabbits for 15 minutes under occlusive patch. The treated area was washed and observed for up to 7 days. Mild erythema was observed in one animal but was fully reversed within 7 days (OECD, 2006; REACH).

Eye Irritation

The chemical is reported to be a slight eye irritant in animal studies. The irritation effects were not sufficient to warrant hazard classification.

In an eye irritation study, the chemical (50 mg solid) was instilled in the eye of two Vienna white rabbits. Slight to marked redness of the conjunctivae at 1 and 24 hours after instillation was reported. Slight oedema and slight corneal opacity were seen in one animal after 24 hours and at day 8, respectively (OECD, 2006; REACH).

In another eye irritation study in two rabbits (unspecified strain), a drop of the chemical (10 % solution in olive oil) caused transient redness,10 minutes after instillation, and oedema after 3 hours. All effects were reversible within 24 hours after treatment (OECD, 2006; REACH).

Sensitisation

Skin Sensitisation

The chemical is considered to be a skin sensitiser based on positive results seen in a single guinea pig maximisation test (GPMT).

In a guinea pig maximization test conducted according to Directive 84/449/EEC, B.6, the chemical was applied to the skin of Pirbright-Hartley female guinea pigs (10 test animals and 5 control animals). During induction exposures, the animals were treated with the chemical at 5 % in olive oil solution (intradermal induction) and 10 % in olive oil solution (topical induction). The chemical caused very slight to well-defined erythema in eight out of 10 animals after a chemical challenge at 5 % in olive oil solution. Animals in the control group had negative responses (OECD, 2006; REACH).

Repeated Dose Toxicity

Oral

Based on the available data, the chemical is not considered to cause serious damage to health from repeated oral exposure. However, exposure to high doses of the chemical indicates that the target organs for toxicity are the liver and the kidneys.

In a sub-chronic study in Wistar rats (10/sex/group) conducted in accordance with OECD Test Guideline (TG) 408, the chemical in 0.5 % aqueous carboxymethylcellulose suspension was administered daily by gavage at 0, 6, 60 or 120 mg/kg bw/day, 7 days/week for 90 days. The neurotoxic potential and reproductive effects of the chemical were also investigated in the study. At 6 mg/kg bw/day, a dose-dependent decrease in aspartate aminotransferase in females, which was considered of no toxicological significance, was reported. At 60 mg/kg bw/day, decrease in plasma prothrombin time and statistically significant increase in absolute liver weights in females were reported. Increased centrilobular hepatocyte hypertrophy in both sexes was reported. At 120 mg/kg bw/day, liver weights (absolute and relative weights) were statistically significantly increased in both sexes. The changes in clinical chemistry and liver weights were indicative of liver enzyme induction. Centrilobular hypertrophy of hepatocytes in both sexes, and statistically significant increase in kidney weights in females were also reported. The no observed adverse effect level (NOAEL) and lowest observed adverse effect level (LOAEL) were determined to be 6 mg/kg bw/day and 60 mg/kg bw/day, respectively (OECD, 2006; REACH).

In a study conducted in beagle dogs (1/sex/dose), the chemical (94.9 % purity) in corn oil was administered by gavage at 0, 1, 5, 10 or 20 mg/kg bw/day (2-5 days/week, total of 20 doses) for five weeks or by two oral administrations of 0, 1, 5 or 20 mg/kg bw. The study was terminated one day after the last exposure to 20 doses, and 30 days after the last exposure to 2 doses. No mortalities and no gross pathological changes were reported at necropsy. At doses 5 mg/kg bw/day and above, male dogs showed dose-related signs of neurological impairment (See **Other Effects - Neurotoxicity**). The NOAEL of this study was determined to be 1 mg/kg bw/day (OECD, 2006; REACH)).

In a "non-Good Laboratory Practice (GLP) study" in mongrel rabbits, the chemical was administered by gavage once daily at doses of 100, 200, 400 or 800 mg/kg bw/day (as 1, 2, 4 or 8 % suspensions in 0.5 % aqueous carboxymethylcellulose), or at doses of 100, 200 or 400 mg/kg bw/day (as 5, 10 or 20% in olive oil solution), 5 days/week for four weeks. Following treatment, the animals were observed for four weeks. After treatment with the chemical in olive oil solution, all animals died at 400 mg/kg bw/day, and 2/4 animals died at 200 mg/kg bw/day. Following treatment with the chemical in aqueous suspension, 3/4 animals at 800 mg/kg bw/day, 2/4 at 400 mg/kg bw/day, and 1/4 at 200 mg/kg bw/day died. All animals survived at 100 mg/kg bw/day for both vehicles. Clinical signs reported from treated animals included: reduced appetite, impaired gait, staggering, trembling, recumbent lateral position and atony. At necropsy, cardiac dilatation associated with venous hyperaemic congestion were reported at 200 mg/kg bw/day and above in olive oil solution, and at 400 mg/kg bw/day and above in aqueous suspension. Small necrotic foci and fat deposits in the liver were also reported for aqueous suspensions. Changes in haematology, and in blood urea and alanine aminotransferase levels were reported in all treated animals. These effects suggest impairment of the liver and kidney functions, which were consistent with the findings from macroscopic examinations of these organs. The LOAEL was determined to be 100 mg/kg bw/day (OECD, 2006; REACH).

Dermal

No data are available.

Inhalation

Based on the available data, the chemical is not considered to cause serious damage to health from repeated inhalation exposure.

In an inhalation toxicity study, six Chr-CD male rats were exposed to the chemical (whole body exposure) at 2400 mg/m^3, 4 hours/day, 5 days/week for 12 days. The observation period was for two weeks after treatment. Mild respiratory irritation including salivation, lacrimation, dyspnoea and red ears were reported. The lowest observed adverse effect concentration (LOAEC) was determined to be 2400 mg/m^3 based on irritation (OECD, 2006; REACH).

In another inhalation study, beagle dogs (1/sex/dose) were exposed to the chemical (whole body exposure) at concentrations of 0, 0.5, 3.2, 9.7, 28 (as aerosol in xylene, aerosol particle size of ≤ 0.5 µm), 6 hours/day, 3-5 days/week for five weeks (total of 20 exposures) with observation period of one day after treatment, or exposed twice to doses of 0, 0.5, 3.2, 9.7, 28 mg/m^3 with observation period of 30 days after treatment. No mortalities or no gross pathological changes at necropsy were reported. At 28 mg/m^3 for 20 exposures, both dogs showed indications of neurological impairment. (See **Other Health Effects - Neurotoxicity**). The NOAEC in this study was determined to be 9.7 mg/m^3 (OECD, 2006; REACH).

Genotoxicity

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

The chemical gave negative results in the following in vitro assays:

- in several bacterial reverse mutation assays with or without metabolic activation using strains of *Salmonella typhimurium* (TA 1535, TA 1537, TA 98 and TA 100 up to 8200 µg/plate) (OECD, 2006; REACH), and in *Escherichia coli* strains WP2 and WP2uvrA (OECD, 2006);
- recombination assay in *Bacillus subtilis* (OECD, 2006);
- micronucleus assay in Chinese hamster lung cells without metabolic activation (OECD, 2006; REACH); and
- mammalian cell gene mutation assay in Chinese hamster ovary cells with or without metabolic activation (REACH).

The chemical gave negative results in an in vivo micronucleus assay in bone marrow cells of mice. Mice (5/sex) were treated intraperitoneally (i.p.) with doses corresponding to 10, 20, 40 and 80 % of the LD50 of the chemical (LD50 value chosen not reported) for four consecutive days (OECD 2006; REACH).

Carcinogenicity

No data are available.

Reproductive and Developmental Toxicity

The available data indicate that the chemical does not cause reproductive and developmental toxicity.

In a sub-chronic study conducted in accordance with the OECD TG 408, fertility effects were examined in rats dosed with 0, 6, 60 or 120 mg/kg bw/day by gavage for three months (See **Repeat Dose Toxicity - Oral**). There were no reported gross pathology, or histopathology changes in the reproductive organs. At 120 mg/kg bw/day, a slight increase in the number of spermatids/g testes was reported. The NOAEL for reproductive effects was considered to be 120 mg/kg bw/day (the highest dose tested) (OECD, 2006; REACH).

In a developmental study conducted in accordance with OECD TG 414, Wistar rats were dosed with 0, 10, 30 or 90 mg/kg bw/day by gavage on days 6–19 post coitum. Maternal toxicity (transient reduction in food consumption, decreased absolute body weight gain, and statistically significantly increased absolute and relative liver weights) was reported at 90 mg/kg bw/day. The chemical did not induce adverse effects on gestational parameters. Isolated occurrences of external and soft tissue malformations, and non-dose-related skeletal malformations in foetuses were reported. The statistically significant skeletal variations (delays in ossification process) reported in foetuses at 90 mg/kg bw/day were regarded as transient in nature and the authors considered the effects as secondary to maternal toxicity. The NOAEL for maternal toxicity was determined to be 30 mg/kg bw/day. The NOAEL for developmental toxicity was determined to be 90 mg/kg bw/day (the highest dose tested) (OECD, 2006; REACH).

Other Health Effects

Neurotoxicity

The available studies showed that the chemical may cause neurotoxic effects in dogs, rabbits and birds, but not in rats. Further studies are necessary to properly evaluate the neurotoxicity potential of the chemical.

In a study conducted in accordance with the United States Environment Protection Agency (US EPA) guideline 163 ("Acute delayed neurotoxicity study"), *Gallus gallus* domesticus hen (ten animals/dose) were dosed once by gavage with the chemical at doses of 0, 1500, 3000, 6000 or 9000 mg/kg bw in corn oil followed by a 14- or 21-day observation period. No mortalities were reported. Treatment-related neuropathological changes in the spinal cord were found even in animals treated at the lowest dose of 1500 mg/kg bw (REACH).

In a 4-week neurotoxicity screening study, female rats (10 animals) were dosed by gavage with the chemical at 0, 700 or 2100 mg/kg bw/day in 0.5 % aqueous carboxymethylcellulose. At 2100 mg/kg bw/day, four animals were sacrificed a few days after treatment due to their moribund state. At necropsy, animals showed stomach erosion/ulcers and hyperaemic intestines. Mortality was reported in both doses. Isolated occurrences of clinical observations including extended abdomen, hypothermia, red crusts on the nose, unsteady gait, urine smears in the abdominal region, piloerection and skin paleness were reported. Results from the functional observation battery tests were similar to those for the control group. At 700 mg/kg bw/day, serum cholinesterase activities were statistically significantly decreased, by 37 % and 34 % on days 15 and 29, respectively. Statistically significantly increase in erythrocyte cholinesterase activity was also reported on both days. No effect was seen on brain cholinesterase activity. There were no neuropathological changes in the central and peripheral nervous systems. A NOAEL was not determined from the study (OECD, 2006; REACH).

In a 4-week rabbit study (See **Repeat Dose Toxicity - Oral**), axonal degeneration was reported at 120 mg/kg bw/day (highest dose tested), but the incidence was regarded as spontaneous in nature and not considered to be treatment-related (OECD, 2006).

In a study in beagle dogs (See **Repeat Dose Toxicity - Oral**), oral doses of 5 mg/kg bw/day and above, 2-5 days/week for five weeks, induced dose-related signs of neurological impairment including: failure in the extensor postural thrust reflex with a tendency of knuckling under or knuckling over of paws, unsteadiness and weakness in the hind limbs, and failure of the patellar reflexes in males. Similar effects without dose-response relationship were seen in females at doses 1 and 5 mg/kg bw/day, for 20 or two exposures, respectively. Histopathology showed treatment related increase in the incidence of axonal degeneration in the spinal cord of both sexes after 20 doses of 10 or 20 mg/kg bw/day. The NOAEL of this study was determined to be 1 mg/kg bw/day (OECD, 2006; REACH).

In a sub-chronic study conducted in accordance with the OECD TG 408, neurotoxic effects were also examined in rats dosed with 0, 6, 60 or 120 mg/kg bw/day by gavage for three months (See **Repeat Dose Toxicity - Oral**). No effects in the functional observation battery tests or motor activity assessment were reported (OECD, 2006; REACH).

Risk Characterisation

Critical Health Effects

The critical local health effect for risk characterisation is skin sensitisation. The chemical can also cause liver and kidney effects following repeated exposure at high concentrations. Neurotoxic effects were also reported in some experimental animals following exposure to the chemical.

Public Risk Characterisation

There are no known uses of the chemical in Australia. However, the chemical has been reported overseas having domestic uses in paints, lacquers and varnishes. The chemical is not listed in the US Household Products Database (US HPD); therefore, the identified domestic uses of the chemical are not expected to be widespread.

Occupational Risk Characterisation

During product formulation, dermal 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 local health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise dermal exposure is 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 an amendment to the hazard classification in the HSIS (Safe Work Australia) (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

Public Health

Products containing the chemical should be labelled in accordance with state and territory legislation (SUSMP, 2016).

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]
Sensitisation	May cause sensitisation by skin contact (Xi; R43)	May cause an allergic skin reaction - Cat. 1 (H317)

[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 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:

- 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.

References

Approved Criteria for Classifying Hazardous Substances [NOHSC: 1008(2004)] Third edition. Accessed at http://www.safeworkaustralia.gov.au/sites/SWA/about/Publications/Documents/258/ApprovedCriteria_Classifying_Hazardous_Substances_NOHSC1008-2004_PDF.pdf

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