# Benzene, 1-methyl-2-nitro-: Human health tier II assessment

22 March 2013

# CAS Number: 88-72-2

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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.

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

#### Disclaimer

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Acronyms & Abbreviations

# **Chemical Identity**

Synonyms	2-Nitrotoluene (2NT) o-Nitrotoluene 1-methyl-2-nitrobenzene 2-nitro-1-methylbenzol benzene, 1-methyl-2-nitro o-methylnitrobenzene o-nitrotoluene o-mononitrotoluene o-nitrotoluol toluene, o-nitro 2-methyl-1-nitrobenezene 2-nitro-1-methylbenzol	
Structural Formula		
Molecular Formula	C7H7NO2	
Molecular Weight (g/mol)	137.137	
Appearance and Odour (where available)	Light yellow liquid with characteristic odour	

c1(N(=O)=O)c(C)cccc1

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# Import, Manufacture and Use

### Australian

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

### International

The following International uses have been identified via the European Union Registration Evaluation Authorisation of Chemicals (EU REACH) Dossiers, Galleria Chemica, the Substances in Preparations in Nordic countries (SPIN) database, Canadian assessments (Challenge list batch 8) and through eChemPortal (the Organisation for Economic Cooperation and Development (OECD) High Production Volume (HPV) chemicals, the International Programme on Chemical Safety (INCHEM), the Aggregated Computer Toxicology Resource (ACToR), and the Hazardous Substances Data Bank (HSDB)).

The chemical has reported commercial use including:

- paints, lacquers and varnishes;
- odour agents;
- cleaning/washing agents;
- surface treatments; and
- in agriculture.

The chemical has reported site-limited use:

- as an organic chemical intermediate in manufacturing petrochemicals.
- as a colouring agent in the manufacture of:
  - toluidine, tolidine, fuchsin and other synthetic dyes;
  - azo & sulphur dyes;
  - dye for cotton, wool, silk, leather and paper; and
  - heat sensitive colourants.

# Restrictions

### Australian

No restrictions were identified.

## International

The Association of Southeast Asian Nations (ASEAN) Cosmetic Directive Annex II Part 1: list of substances which must not form part of the composition of cosmetic products.

Canadian list of prohibited and restricted cosmetic ingredients (the cosmetic ingredient 'Hotlist').

EU Cosmetic Directive 76/768/EEC Annex II: list of substances which must not form part of the composition of cosmetic products.

New Zealand Cosmetic Products Group Standard - Schedule 4: components cosmetic products must not contain - table 1.

# **Existing Work Health and Safety Controls**

## **Hazard Classification**

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

Carc. Cat. 2; R45

Muta. Cat. 2; R46

Repr. Cat. 3; R62

Xn; R22.

## **Exposure Standards**

#### Australian

TWA: 11 mg/m<sup>3</sup> (2 ppm).

#### International

The following are identified (Galleria Chemica):

TWA 11 mg/m³ (2 ppm). [Canada, Indonesia, Ireland, Malaysia, Poland, Spain, Switzerland, Taiwan, USA (NIOSH RELs)].

TWA 12 mg/m<sup>3</sup> (2 ppm). [Denmark and South Africa (OELs)].

STEL 11 mg/m3 (2 ppm) [Estonia, Sweden].

STEL 30 mg/m<sup>3</sup> (5 ppm). [Greece, Mexico, South Africa].

# **Health Hazard Information**

## **Toxicokinetics**

#### Absorption, distribution and excretion

2-Nitrotoluene is rapidly absorbed, extensively metabolised and rapidly excreted in rats and mice (OECD, 2008).

In a study conducted in male F344 rats, 72 hours after administration of a single oral dose of 200 mg/kg bw radiolabelled 2-nitrotoluene, excreta were collected. The radiolabel was excreted rapidly (86% within 24 hours). The major route of excretion was via urine; 70–85% of the dose was

excreted in the urine within 72 hours. Within that period, 5-13% and 0-0.1% of the dose was excreted in the faces and in exhaled air, respectively. The major metabolite excreted in the urine 72 hours after administration was 2-nitrobenzene (29% of the dose. Other metabolites identified included 2-nitrobenzyl glucuronide (14% of the dose) and *S*-(2-nitrobenzyl)-*N*-acetylcysteine (12% of the dose) (IARC, 2012).

#### Metabolism

In an analysis of the urinary metabolites in workers who manufactured nitrotoluenes and trinitrotoluene in Liaoning Province, China, nitrobenzoic acids were found in 96% and 73% of the urine samples from workers exposed to 2-nitrotoluene, and 4-nitrotoluene respectively; and air concentrations of neither dinitrotoluenes nor 2-nitrotoluene correlated with the concentrations of nitrobenzoic acids. The air concentrations of 2-nitrotoluene and 4-nitrotoluene were  $759 \pm 836 \ \mu g/m^3$  and  $685 \pm 500 \ \mu g/m^3$  respectively; the concentrations ( $\mu$ mol/L) of the urinary metabolites were  $4.25 \pm 5.76$  2-nitrobenzoic acid,  $0.33 \pm 0.65$  2-nitrobenzyl alcohol,  $0.12 \pm 0.40$  4-nitrobenzoic acid and  $0.01 \pm 0.03$  4-nitrobenzyl alcohol. Thus, nitrobenzoic acids are the primary urinary metabolites in humans exposed to 2-nitrotoluene and 4-nitrotoluene (IARC, 2012).

## **Acute Toxicity**

#### Oral

The chemical is harmful via the oral route. Based on the data available, the current hazard classification for its acute oral toxicity is considered appropriate.

The acute oral toxicity of 2-nitrotoluene has been investigated in rats, mice and rabbits. The available studies on acute oral toxicity indicate the oral  $LD_{50}$  value range from 890 to 2546 mg/kg bw in rats, from 970 to 2462 mg/kg bw in mice and 1750 mg/kg bw in rabbits. Clinical signs of toxicity were related to methaemoglobin formation (no other details provided) (OECD, 2008).

#### Dermal

The chemical is of low acute toxicity via the dermal route.

In acute dermal toxicity studies, 2-nitrotoluene did not produce either mortality or toxicity following exposure to 5000 mg/kg bw in rats or 20000 mg/kg bw in rabbits.

#### Inhalation

Based on the data available, the chemical is of low acute inhalation toxicity.

At the following saturated vapour concentrations, rats and mice did not show mortalities, toxicity or gross lesions (up to 14 days) following exposure to 2-nitrotoluene:

- 190.8 ppm (1.086 mg/L) for eight hours or 320 ppm (1.795 mg/L) for four hours in rats;
- 354 ppm (1.986 mg/L) for four hours in mice.

Although the LC<sub>50</sub> between 1 and 5 mg/L warrants hazard classification, as the tested conditions are limited by saturated vapour concentrations, classification is not considered necessary.

## **Corrosion / Irritation**

#### **Respiratory Irritation**

The acute inhalation studies have not revealed any signs of respiratory irritation (OECD, 2008).

#### Skin Irritation

According to several studies, 2-nitrotoluene was considered non-irritating to intact skin.

On abraded skin, although erythema and eschar formation were observed in three rabbits (out of six) at 24 hours, it did not persist at the end of the observation time, i.e. at 72 hours (OECD, 2008).

#### Eye Irritation

Based on the data available, 2-nitrotoluene is not considered an eye irritant.

Six male albino rabbits were treated with undiluted 2-nitrotoluene (0.1 mL/rabbit) for  $\geq$ 24 hours to investigate ocular irritation. The eyes of each animal were examined at 24, 48 and 72 hours. The mean Draize scores for corneal opacity, iris lesion, redness of the conjunctivae and chemosis were all zero for each animal at the specified reading times. Therefore, 2-nitrotoluene was considered a non irritant to rabbit eyes (OECD, 2008).

### Sensitisation

#### Skin Sensitisation

Based on a study conducted in a structurally similar chemical (4-nitrotoluene), 2-nitrotoluene is not considered a skin sensitiser.

A skin sensitisation study was conducted in guinea pigs (n = 20) using 4-nitrotoluene (Buehler test–OECD TG 406 with deviations on GLP compliance and analytical purity not reported) did not reveal any skin sensitisation. Induction was performed by dermal application of a 50% solution in acetone and a 10% solution was used for the challenge. In the absence of experimental data on 2-nitrotoluene and similarity between the two compounds, it was reported that 2-nitrotoluene was non-sensitising based on the read-across from the 4-nitrotoluene with sufficient confidence (REACH, 2012).

### **Repeated Dose Toxicity**

#### Oral

The chemical is not considered to be toxic with short term repeated exposure up to 45 mg/kg bw/day in rats.

Several studies have investigated the toxicity of 2-nitrotoluene following repeated oral administration to rats and mice. The rat was more susceptible than mouse to 2-nitrotoluene in the repeat dose oral studies (OECD, 2008).

In the 14 day studies, 2-nitrotoluene, administered up to 5000 ppm (mice) or 10000 ppm (rats), did not cause effects on either survival or clinical signs of toxicity, although mice at 5000 ppm showed decreases in body weight gains relative to controls. In addition, minimal oval cell hyperplasia in liver was observed only in 10000 ppm male rats. Therefore, 10000 ppm was selected as the high concentration for 13 week studies (OECD, 2008).

In a 13 week toxicity study in rats, relative liver weights were increased from 625 ppm in both sexes. However, at this dose level there was no treatment related histopathology. Non neoplastic lesions occurred at dose levels of 1250 ppm and above. Therefore, the NOAEL for sub-chronic toxicity was considered to be 625 ppm (45 mg/kg bw/day) based on capsular fibrosis observed in male rat spleens at 1250 ppm (89 mg/kg bw/day). There were no effects on survival. The clinical signs of toxicity were limited to decreases in mean body weight gain and feed consumption in all exposure groups (OECD, 2008).

In a 13 week study in mice, the only histopathological lesion observed was degeneration and metaplasia of the olfactory epithelium in both sexes from 1250 ppm (223 and 268 mg/kg bw/day for males and females, respectively).

#### Dermal

No data are available.

Inhalation

No data are available.

#### Observation in humans

Amongst workers in a factory that produced di- and trinitrotoluenes, a wide variety of haemoglobin adducts were found, one of which was a 2-methylaniline–haemoglobin adduct, which derives specifically and only from 2-nitrotoluene (IARC 2012).

## Genotoxicity

The chemical is currently classified as a Category 2 Mutagenic Substance with the risk phrase 'May cause heritable genetic damage' (R46) in Australia. The data summarised below support this classification.

Several in vitro and in vivo genotoxicity studies are described below. In the Ames test, 2-nitrotoluene is not mutagenic in any of several strains of *Salmonella typhimurium* with or without metabolic activation (S9). However, the addition of norharman (a substance found in tobacco tar) produced a positive result in the presence of S9 mix. On *Bacillus subtilis*, 2-nitrotoluene had a genotoxic effect. In cytogenetic tests on Chinese hamster ovary (CHO) cells, 2-nitrotoluene increased the sister chromatid exchange rate, this being more pronounced in the presence of S9 mix. In a chromosomal aberrations test in CHO cells, the result was negative with or without S9 mix. The chemical also induced chromosomal aberrations in human lymphocytes. There was an increase in polyploidy cells when 2-nitrotoluene was tested in cultures of Chinese hamster lung (CHL) cells in the absence of S9 mix (OECD, 2008).

No induction of unscheduled DNA synthesis was observed in isolated rat spermatids, spermatocytes or hepatocytes. 2-Nitrotoluene was found to be clastogenic when tested in larvae of mosquito Culex fatigans in vivo. However, a dominant lethal test in the same species was negative. 2-Nitrotoluene induced adducts in haemoglobin and hepatic DNA in male Wels-Fohm rats dosed chronically 5 days a week for 12 weeks. However, Wistar rats dosed with a single dose of 2-nitrotoluene by oral gavage did not show induced DNA adducts; nevertheless, they formed haemoglobin adducts (OECD, 2008).

2-Nitrotoluene did not induce a significant increase in the frequency of micronuclei in bone marrow polychromatic erythrocytes of male rats or male mice when administered by intraperitoneal injection up to doses of 2500 mg/kg in rats and 400 mg/kg in mice. Results of a peripheral blood micronucleus test were equivocal for male mice and negative for female mice administered o-nitrotoluene in feed for 13 weeks. Positive results were found in the unscheduled DNA synthesis (UDS) test for both male and female rats administered 2-nitrotoluene (OECD, 2008).

Males were more sensitive to genotoxicity from 2-nitrotoluene. A sex difference in biliary excretion may explain the sex difference in the genotoxicity of 2-nitrotoluene. In addition, the chemical did not induce DNA repair in germ-free animals, whereas DNA repair was induced in Charles River Altered Schaedler Flora-associated animals. Male and female F344 rats were shown to have similar populations of intestinal bacteria; however at the doses used, females were resistant to the genotoxic action of the chemical. These results indicate the obligatory role of intestinal bacteria in the metabolic activation of the chemical, showing that the genotoxic potential of the chemical is dependent upon the sex of the animal under study (OECD, 2008).

Gene mutations in rats and mice (B6C3F1) exposed to 2-nitrotoluene suggest that 2-nitrotoluene is metabolised to mutagenic intermediates and that could be the reason why most of the in vitro genotoxicity tests were negative. 2-Nitrotoluene has a number of potentially active metabolites that could account for the mutation profile observed in the tumours of mice (OECD, 2008).

In conclusion, 2-nitrotoluene is mutagenic in somatic cells and a hazard classification for genotoxicity/mutagenicity is warranted. In addition, it reaches the germ cells (at 353 mg/kg bw/day) since toxicity was observed in testis and epididymis of rats in a 13-week dietary study. The OECD report (2008) states: *It is reasonable to assume that a somatic cell mutagen also has the potential to cause mutations in germ cells. The likelihood of mutagenicity occurring in the germ cells in vivo is determined by the toxicokinetics of the substance and its ability to reach the target tissues in* 

sufficient amounts to elicit the effect. Although there is no direct evidence relating to mutagenicity in the germ cells, in view of the above, the current HSIS classification is supported.

# Carcinogenicity

The chemical is currently classified as a Category 2 carcinogen with the risk phrase 'May cause cancer' (R45) in Australia. The data summarised below support this classification.

There was clear evidence of carcinogenic activity of 2-nitrotoluene in rats, based on increased incidences of: malignant mesothelioma; subcutaneous skin neoplasms; mammary gland fibroadenoma and liver neoplasms in males; subcutaneous skin neoplasms and mammary gland fibroadenoma in females. The increased incidences of lung neoplasms in males and of hepatocellular adenoma in females were also considered to be exposure related. Malignant mesotheliomas occurred with incidences of 33%, 48% and 73% in the 625, 1250 and 2000 ppm core study male rat groups, respectively. The incidences of malignant mesotheliomas were 73% and 90% in the 2000 and 5000 ppm stop-exposure male rat groups. The incidence of mesothelioma was higher in the 2000 ppm stop-exposure group than in the 625 ppm even though the latter group received approximately 50% more total exposure to 2-nitrotoluene. The incidences of mesotheliomas were similar in the 2000 ppm core study and stop-exposure groups of male rats. Thus, critical events leading to mesothelioma occurred early in the study, and this damage was irreversible. The molecular pathogenesis of mesotheliomas is not well understood. Decreased incidences of mononuclear cell leukaemia and testicular interstitial cell adenoma in exposed groups were related to splenic and testicular toxicity, respectively (OECD, 2008; IARC, 2012).

There was clear evidence of carcinogenic activity of 2-nitrotoluene in male and female mice based on increased incidences of haemangiosarcoma, carcinoma of the large intestine (caecum), and hepatocellular neoplasms (females only because males died early due to the development of haemangiosarcomas). The occurrence of gene mutations (p53 or  $\beta$ -catenin) in 2-nitrotoluene induced haemangiosarcomas, but not in spontaneous haemangiosarcomas, suggest that the pathways leading to 2-nitrotoluene-induced cancer differ from the pathways in spontaneous haemangiosarcomas (OECD, 2008).

No 2-nitrotoluene epidemiology studies on carcinogenesis have been reported in the literature. However, excess cancers have been found in workers exposed to a related chemical, o-toluidine (OECD, 2008).

In summary, there is good evidence of an increase in tumour incidence at multiple sites in both rats and mice. There is also evidence that time to onset effects, following exposure, is very short. These observations are consistent with genotoxic aetiology, which is consistent with the findings from the genotoxicity studies. The LOAEL for chronic toxicity was considered to be 625 ppm in rats (25 and 30 mg/kg bw in males and females, respectively) based on lesions observed in liver, bone marrow, spleen and lung for both sexes and in mammary gland and mandibular lymph node only for females.

There is sufficient evidence in experimental animals for the carcinogenicity of 2-nitrotoluene based on the occurrence of mesotheliomas and mesothelial hyperplasia in treated animals.

## **Reproductive and Developmental Toxicity**

The chemical is currently classified as a Category 3 reproductive toxin with the risk phrase 'Possible risk of impaired fertility' (R62) in Australia. The data summarised below support this classification. The information available is limited and cannot prove that the impaired fertility effects occurred in the absence of other toxic effects. Therefore, it is not possible to rule out that impaired fertility is not a secondary non-specific consequence of other toxic effects.

2-Nitrotoluene was administered in feed to F344/N rats at doses 0, 45, 89, 179, 353 or 694 mg/kg bw/day for 13 weeks. At 353 mg/kg bw/day and above, damage to the testes and the epididymis with a simultaneous reduction in the sperm count and the motility of the sperm in males, and a prolongation of the menstrual cycle among the females were observed. The NOAEL for reproductive toxicity in rats was 179 mg/kg bw/day. Reduced sperm motility was also observed at 1536 mg/kg bw/day (the highest dose level tested) for mice (OECD, 2008).

Two non-standard reproduction studies reported some developmental toxicity effects in rats. In one study with CD rats (0, 50, 150 or 450 mg/kg bw/day), the only effect considered as indicative of developmental toxicity was the retardation in pup growth, at all doses. However, details are not available on its severity. Transfer of the substance through milk contributing to this effect cannot be ruled out. In the other study carried out in Wistar rats (0, 200 mg/kg bw/day), mortality, vitality and behaviour of pups from both treated and untreated animals were the same and no histopathological changes in organs occurred among the young animals, regardless of treatment. Furthermore, there were no toxic effects from transfer of the substance through milk. Differences in results between the two studies could be due to differences in sensitivity between strains (OECD, 2008).

Based on the above, the chemical is not considered a developmental toxin.

# **Risk Characterisation**

## **Critical Health Effects**

The main critical effects to human health are genotoxicity, carcinogenicity and potential reproductive/fertility effects. The chemical may also cause harmful effects if ingested.

## **Public Risk Characterisation**

The characterised hazards have the potential to pose an unreasonable risk to public under the international uses identified. Although use in cosmetics or domestic products in Australia is not known, the chemical is reported to be used in cleaning/washing agents, paints, lacquers and varnishes overseas. Also, there are international restrictions on this chemical for use in cosmetics. Hence, there is a concern for potential use of this chemical in domestic products resulting in public exposure. Therefore, regulatory controls are necessary to restrict its use in cosmetics and domestic products through scheduling.

## **Occupational Risk Characterisation**

The most probable routes of worker exposure to 2-nitrotoluene is inhalation and dermal contact during manufacture and use of this chemical (e.g. end product formulations and application of paints). Oral exposure is assumed to be prevented by good hygiene practices.

Given the critical health effects, the risk to workers from this chemical is considered high unless adequate control measures to minimise occupational exposure to the chemical are implemented. The existing hazard classification is considered adequate for determining appropriate controls to protect workers handling the chemical and products/mixtures containing it.

# **NICNAS Recommendation**

Further risk management is required for public safety. Sufficient information is available to recommend the chemical to be risk managed for public safety from the potential use in domestic products through scheduling.

The chemical is sufficiently assessed and risk managed for worker safety provided the recommendation for classification and labelling is followed.

# **Regulatory Control**

**Public Health** 

The chemical is recommended for scheduling to prohibit its sale, supply and use in domestic products. This will help in mitigating the risk to the public.

Matters to be taken into consideration include carcinogenicity, mutagenicity and reproductive toxicity of the chemical and its possible use in paints, varnishes, lacquers and cleaning/washing agents.

#### Work Health and Safety

The chemical is recommended for classification and labelling under the current approved criteria and adopted GHS as below. This does not consider classification of physical hazards and environmental hazards.

Hazard	Approved Criteria (HSIS)ª	GHS Classification (HCIS) <sup>♭</sup>
Acute Toxicity	Harmful if swallowed (Xn; R22)*	Harmful if swallowed - Cat. 4 (H302)
Genotoxicity	Muta. Cat 2 - May cause heritable genetic damage (T; R46)*	May cause genetic defects - Cat. 1B (H340)
Carcinogenicity	Carc. Cat 2 - May cause cancer (T; R45)*	May cause cancer - Cat. 1B (H350)
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of impaired fertility (Xn; R62)*	Suspected of damaging fertility - Cat. 2 (H361f)

<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

Any domestic product containing the chemical should be used according to label instructions.

## Advice for industry

#### **Control measures**

Control measures to minimise the risk from dermal/ocular/inhalation 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 storage, handling and use of a hazardous chemical are dependent 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:

- use of closed systems or isolation of 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;
- minimisation of manual processes and work tasks through automation of processes;
- work procedures that minimise splashes and spills;
- regular cleaning of equipment and work areas; and
- use of 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 be relied upon on its own to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selection of 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 hazards and physicochemical (physical) hazards) of hazardous chemical are prepared; and
- management of risks arising from storage, handling and use of 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 physical hazards of the chemical has not been undertaken as part of this assessment.

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