Naphthalene: Human health tier II assessment

24 April 2015

CAS Number: 91-20-3

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.

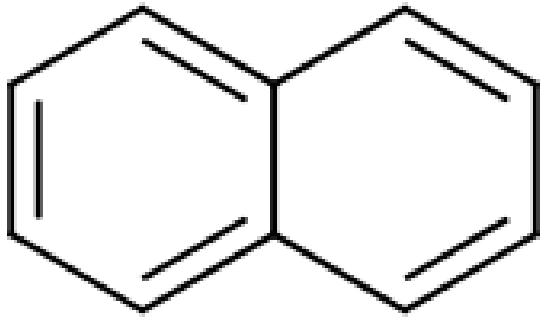
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	None
Structural Formula	
Molecular Formula	C10H8
Molecular Weight (g/mol)	128.173
Appearance and Odour (where available)	Colourless to brown solid with a characteristic odour
SMILES	c12c(cccc1)cccc2

Import, Manufacture and Use

Australian

The following Australian industrial uses were reported under previous voluntary calls for information.

The chemical has reported commercial use as a fuel and lubricant additive and in commercial surface coatings.

The chemical has reported site-limited uses including:

- as a dye dispersant and blocking agent in textile processing;
- as a water reducer in plasterboard manufacture; and
- in fragrance formulation.

The following non-industrial uses have been identified in Australia (APVMA, 2011):

- as an insecticide and fumigant in moth repellents;
- in veterinary products; and

- for plant growth regulation.

International

The following international uses have been identified through the European Union (EU) Registration, Evaluation, Authorization and Restrictions 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) National Library of Medicine's Hazardous Substances Data Bank (HSDB); and various international assessments including from the International Agency on Research on Cancer (IARC) and the National Toxicology Program (NTP).

The chemical has reported domestic use in a range of domestic products including home maintenance products such as paints and coatings up to a concentration of 2 % and auto products up to a concentration of 42 % (US Household Products Database).

The chemical has reported domestic uses in the SPIN database including (but not limited to):

- adhesive and binding agents;
- cleaning agents;
- colouring agents;
- corrosion inhibitors;
- fillers; and
- paints, lacquers and varnishes.

However, it should be noted that SPIN does not distinguish between direct use of the chemical, or use of the materials that are produced from chemical reactions involving the chemical.

The chemical has reported site-limited uses including:

- as an intermediate in producing various chemicals and pharmaceuticals including phthalic anhydride, dyestuff (azo dyes) and naphthalene derivatives used as solvents and fuel additives;
- in pyrotechnic manufacture; and
- as an artificial pore former in manufacturing grinding wheels.

Moth repellents and insecticides have been identified as international non-industrial uses.

Restrictions

Australian

This chemical is listed in the *Poisons Standard—the Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP) in Schedule 6 (SUSMP, 2015) as 'NAPHTHALENE (excluding its derivatives) except in liquid hydrocarbons as an impurity'.

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

International

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

- EU Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain; and
- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient ‘Hotlist’).

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):

- Xn; R22 (acute toxicity)
- R40 Carc. Cat 3 (carcinogenicity)

Exposure Standards

Australian

The chemical has an exposure standard of 52 mg/m^3 (10 ppm) time weighted average (TWA) and 79 mg/m^3 (15 ppm) short-term exposure limit (STEL) (Safe Work Australia).

International

The chemical has an exposure standard of 50 mg/m^3 (10 ppm) TWA and 75 mg/m^3 (15 ppm) STEL in many countries including China, Egypt, Estonia, France, Germany, Greece, India and Ireland (Galleria Chemica).

Health Hazard Information

The toxicity of the chemical has been shown to vary depending on the animal species and consequent metabolite formation (EPA, 1998).

Toxicokinetics

Following oral administration in rats, the chemical is reported to be readily and completely absorbed from the gastrointestinal (GI) tract. Extensive metabolism occurs, and the chemical is rapidly eliminated in the urine after enterohepatic circulation (EU RAR, 2003). In humans, the chemical is expected to be readily absorbed irrespective of the route of exposure and excreted primarily in the urine (SCOEL, 2010).

Signs of systemic toxicity were reported in humans after ingesting or inhaling the chemical in mothballs, or after dermal contact with clothes impregnated with the chemical, which showed that the chemical was easily absorbed by all routes of exposure (EU RAR, 2003). Neonatal haemolytic anaemia was also reported following ingestion of the chemical by pregnant women, demonstrating the chemical or its metabolites can cross the placental barrier (EU RAR, 2003).

Susceptibility to the chemical depends on the animal species and exposure concentration (EPA, 1998):

- mice are more sensitive than rats to acute toxic effects (NTP, 2000);
- haemolytic anaemia, the main acute toxicological effect observed in humans, has not been observed in rats, mice or rabbits (SCOEL, 2010);
- naphthoquinones (assumed to cause cataracts) are produced in rats and rabbits only at exposure levels ≥700 mg/kg bw (EPA, 1998).

The two main stable metabolites formed by human hepatic microsomes are 1-naphthol and naphthalene 1,2-dihydrodiol (EPA, 1998). The metabolite 1-naphthol was detected in urine samples taken from workers exposed to the chemical and other hydrocarbons after an eight-hour shift. There was a linear relationship between the concentration of naphthalene in the air (0-6 mg/m^3) and the amount of 1-naphthol found in the urine (EU RAR, 2003). The same metabolite was also found in patients' urine treated with dermal applications of coal tar containing the chemical (total amount applied: 118 mg) for four weeks (EU RAR, 2003).

Acute Toxicity

Oral

The chemical is classified as hazardous with the risk phrase 'Harmful if swallowed' (Xn; R22) in the HSIS (Safe Work Australia). The available mouse data support this classification. However, low acute oral toxicity was found in rats, which showed that the sensitivity depended on the species exposed.

The median lethal dose (LD50) in Sprague Dawley (SD) rats was >2000 mg/kg bw in a series of well-conducted acute toxicity limit tests (EU RAR, 2003). There were only 2/30 rat mortalities when the chemical was administered at 2000 mg/kg bw. Diarrhoea was reported in 17/30 rats (EU RAR, 2003).

In CD-1 mice, LD50 values of 533 and 710 mg/kg bw were reported for males and females, respectively. Signs of toxicity included ataxia and depressed breathing, but not haemolytic anaemia (EU RAR, 2003).

Dermal

Based on the available data, the chemical has a low acute dermal toxicity.

Sherman rats (n = 40) administered the chemical dermally at 2500 mg/kg bw did not show any mortalities (EU RAR, 2003).

In another study, the chemical was applied at a dose of 16000 mg/kg bw to the skin of SD rats. No mortalities occured during the 13-day post-exposure observation period (REACH).

Inhalation

The overall information is inconclusive.

There were no mortalities in Wistar rats (n = 5/sex) exposed (whole-body) to vapours of the chemical at 77 ppm (ca. 0.4 mg/L) for four hours. Clinical signs of toxicity included lacrimation, closed eyes and breathing through the mouth. Although the study was conducted according to a standard guideline (equivalent to OECD Test Guideline (TG) 403), the toxicity category could not be determined as the highest achievable dose was 0.4 mg/L (REACH).

A median lethal concentration (LC50) of >0.34 mg/L/1hour for rats was indicated, but there were no details of the study (ChemIDPlus).

The EU Risk Assessment Report (RAR) noted that a four-hour exposure to doses up to 0.17 mg/m^3 resulted in slight pulmonary damage in mice (details not available), and also that mice exposed (nose-only) to 0.38 mg/m^3 of the chemical for four hours displayed bronchiolar damage. The EU RAR noted that these results were 'questionable' (EU RAR, 2003).

Observation in humans

Haemolytic anaemia and cataracts were the major toxic effects reported from accidental exposure to the chemical, either by inhalation or by ingestion (IARC, 2002). Typical symptoms of intoxication are dark urine, pallor, abdominal pain, fever, nausea, diarrhoea and vomiting (EU RAR, 2003). Combined inhalation and dermal exposure, or combined inhalation and oral exposure to the chemical can also lead to haemolytic anaemia in humans (EPA, 1998).

Between 1949 and 1959, 12 cases of haemolytic anaemia were reported in children who had sucked or swallowed mothballs containing the chemical. Haemolytic anaemia was also associated with ingesting anointing oils containing the chemical (IARC). Very severe haemolytic anaemia (almost lethal) was reported in a 16-year-old female who ingested a single dose of 6 g of the chemical (EU RAR, 2003).

Based on several case studies, individuals with a glucose-6-phosphate dehydrogenase (G6PD) deficiency were more sensitive to the chemical's haemolytic effects (IARC).

An in vitro study on human blood samples mixed with either the chemical, 1-naphthol or 2-naphthol, showed that the metabolite 1-naphthol was responsible for most of the haemolytic effect observed, while the unmetabolised chemical did not induce any haemolytic activity (EU RAR, 2003).

Corrosion / Irritation

Respiratory Irritation

The chemical is considered to cause respiratory tract irritation, warranting hazard classification (see **Repeat dose toxicity: Inhalation**).

Data on acute inhalation toxicity are limited. In the single study available (see **Acute toxicity: Inhalation**), no respiratory tract effects were reported in rats apart from breathing through the mouth.

Repeated dose toxicity studies in rats indicated minimal damage to the nasal olfactory epithelium after exposure to vapours of the chemical starting from 1 ppm in a 13-week study and 2 ppm in a four-week study (see **Repeat dose toxicity: Inhalation**). The damage was more pronounced at higher exposure concentrations (SCOEL, 2010).

After long-term exposure (see **Carcinogenicity**), nasal and lung inflammation were observed in rats and mice at all doses of 10 ppm (50 mg/m^3) and above.

Skin Irritation

The chemical is considered to be a slight skin irritant. The reported effects were not sufficient to warrant hazard classification.

In a well-conducted study (as reported by the EU RAR, 2003), six rabbits were exposed to a single dose of 500 mg of the chemical for four hours and observed for six days. Slight to well defined erythema was seen in three rabbits, appearing 30 minutes after exposure, with a slight fissuring of the skin appearing 72 hours after exposure. No oedema was reported and all signs had cleared within six days (EU RAR, 2003).

In another study (reported as similar to OECD TG 404), the chemical was applied in occlusive patches, at a dose of 500 mg, to the skin of six New Zealand White rabbits for 24 hours. Mean erythema and oedema scores were <2 for individual animals. Effects had not completely cleared within 48 hours (REACH).

Eye Irritation

The chemical is a slight eye irritant. The reported effects were not sufficient to warrant hazard classification.

In an eye irritation study (reported as similar to OECD TG 405), six rabbits were exposed to the chemical (0.1 mg in one eye of each rabbit) for 24 hours and then observed for seven days. Only minor effects were reported. One rabbit had an iris reaction on day two after dosing (grade 1), five rabbits showed conjunctival redness (grade 1) over a period of two days and slight chemosis (grade 1) was noted in one rabbit on day one after dosing. All effects had cleared by day three after dosing (REACH).

Sensitisation

Skin Sensitisation

The chemical is not a skin sensitiser.

In a guinea pig maximisation test reported to be OECD TG 406-compliant (REACH), male Hartley guinea pigs (n = 12/group) were exposed to an intradermal (i.d.) dose of 1 % followed seven days later by a 48-hour topical application of 10 % of the chemical in acetone (induction concentrations). On day 21, animals were dermally exposed for 24 hours to 0.1 % and 1 % of the chemical in acetone (challenge concentrations). The chemical did not cause any sensitisation reactions, compared with the positive control (2-naphthol), which induced 100 % positive reactions in animals tested (REACH).

In a Buehler test in guinea pigs (guideline not indicated), 20 animals were exposed dermally to 400 mg of the chemical for three six-hour periods (induction phase), and 14 days later were exposed again to 400 mg (challenge phase). No skin reactions were reported (EU RAR, 2003).

Observation in humans

In spite of the widespread use of the chemical in products involving dermal contact, no cases of skin or respiratory sensitisation were reported (EU RAR, 2003).

Repeated Dose Toxicity

Oral

Based on the treatment-related effects reported in rat and mice studies, repeated oral exposure to the chemical is not considered to cause serious damage to health.

In a well-conducted 13-week study (as reported by the EU RAR, 2003), groups of Fischer 344 (F344) rats (n = 10/sex/dose) were administered the chemical at oral gavage doses of 0, 25, 50, 100, 200 or 400 mg/kg bw, five days/week (duration-adjusted 0, 17.9, 35.7, 71.4, 142.9, and 285.7 mg/kg bw/day). In the highest dose group, two male rats died and rats of both sexes had diarrhoea and displayed lethargy, hunched posture, and rough coats at intermittent intervals throughout the study. Haematological parameters were affected only at the highest dose. Male rats exhibited focal cortical lymphocytic infiltration or focal tubular regeneration in kidneys at 200 mg/kg bw (adjusted dose = 142.9 mg/kg bw/day) (2/10) and diffuse renal tubular degeneration at the highest dose (1/10); female rats exhibited lymphoid depletion of the thymus at the highest dose (2/10). Based on the significantly decreased mean body weight at the two highest doses, a lowest observed adverse effect level (LOAEL) of 200 mg/kg bw was determined (adjusted dose = 142.9 mg/kg bw/day) (EPA, 1998).

In a chronic study, BD rats were treated with 10–20 mg of the chemical in the diet per day, six days/week, for 100 weeks. No signs of toxicity (including damage to the eye) were recorded (EU RAR, 2003).

In another 13-week study, B6C3F1 mice (n = 10/sex/dose) were administered the chemical by gavage at 0, 12.5, 25, 50, 100 or 200 mg/kg bw, five days/week. At the highest dose, transient signs of toxicity were reported including lethargy, rough hair coats and decreased food consumption. No exposure-related lesions were observed in any organs. Changes in haematological parameters observed at the highest dose were not considered 'biologically significant' by the authors; therefore, the highest dose was reported as the LOAEL in mice (EPA, 1998).

In a subchronic study in mice (CD-1 albino strain), the chemical was orally administered at 0, 5.3, 53 or 133 mg/kg bw/day for 90 days. A no observed adverse effect level (NOAEL) of 53 mg/kg bw/day was established based on significantly decreased organ weights (brain, liver and spleen) in females at

the highest dose (EPA, 1998).

The occurrence of cataracts was specifically investigated in other studies. These studies indicated that the repeated administration of the chemical at very high doses (1000 mg/kg bw) can cause cataracts in rats, mice and rabbits (EPA, 1998).

As the main animal species used (rats, mice and rabbits) in toxicological studies did not show haemolytic anaemia (the main toxicological effect observed in humans) after exposure to the chemical (SCOEL, 2010), no NOAEL or dose-response could be identified for haemolytic anaemia (EU RAR, 2003).

Dermal

Considering the results from a well conducted rat study (as reported in the EU RAR, 2003), repeated dermal exposure to the chemical is not considered to cause serious damage to health.

SD rats (n = 10/sex/dose) were treated with dermal occlusive patches of the chemical alone at 0, 100, 300 or 1000 mg/kg bw/day for 13 weeks (following OECD TG 411). Apart from irritated skin and papules in the treatment area that were observed in both treated and non-treated animals, no signs of systemic toxicity were reported, establishing a NOAEL of 1000 mg/kg bw/day in rats (EU RAR, 2003; REACH).

Inhalation

Considering the results from two well-conducted rat studies (as reported in EU RAR, 2003), repeated inhalation exposure to the chemical is not considered to cause serious systemic toxicity. Local irritation effects were report in both studies (see **Irritation: Respiratory**).

In a well-conducted 13-week study (as reported by the EU RAR, 2003), groups of SD rats (n = 10/sex/dose) were exposed (nose-only) to vapours of the chemical at 0, 2, 10 or 58 ppm (approximately 0, 10, 50 or 300 mg/m^3), six hours/day, five days/week. Significantly decreased body weight gain (by 43 % and 34 % in male and female rats, respectively) associated with decreased food consumption was reported at the highest dose. No other significant clinical or haematological changes were reported. Only the nasal epithelium showed dose-related effects. At the highest dose, the chemical induced erosion of the olfactory epithelium, hyperplasia of basal cells in the olfactory epithelium and loss of Bowman's glands. At the lowest dose, effects included minimal atrophy, rosette formation in the nasal epithelium, occasional degenerate cells, loss of Bowman's glands and minimal hyperplasia. No NOAEL was identified in this study (EU RAR, 2003; REACH).

Similar results were obtained in a four-week rat study (n = 5/sex/dose) using vapours of the chemical at 0, 1, 3, 10, 29 or 71 ppm (approximately 0, 5, 15, 50, 150 and 370 mg/m^3), with no systemic toxicity but local effects observed at all dose levels on the nasal epithelium (EU RAR, 2003).

Observation in humans

After being exposed for up to five years by inhalation and direct eye contact to vapours of the chemical in a manufacturing plant (exposure levels not indicated), 21 workers were examined for eye problems. Among them, eight workers had developed multiple pin-point lens opacities not related to age, which were considered a consequence of the long-term exposure to the chemical (ATSDR, 2005).

Genotoxicity

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

Negative results were obtained in bacterial gene mutation tests on diverse strains of *Salmonella typhimurium* and *Escherichia coli*, even at cytotoxic concentrations (not available). The chemical was reported as 'clearly not genotoxic in bacterial test systems' (EU RAR, 2003).

The chemical gave positive results in a chromosome aberration test in Chinese hamster ovary (CHO) cells. A statistically significant dose-related increase of metaphase aberrations was observed in CHO cells, but only with metabolic activation at 30–67.5 µg/mL (EU RAR, 2003; REACH). Negative results were reported for other in vitro studies including:

- an unscheduled DNA synthesis (UDS) assay that used rat hepatocytes with doses up to 5000 µg/mL (EU RAR, 2003);
- sister chromatid exchange (SCE) assays that used CHO cells or human peripheral lymphocytes (doses not stated) (EU RAR, 2003); and
- no mutations at the *hprt* and *tk* loci in human lymphoblastoid cells exposed to 40 µg/mL of the chemical (REACH).

Two in vivo genotoxicity assays with the chemical gave negative results including (EU RAR, 2003; REACH):

- a micronucleus test in which there was no increase in the frequency of micronuclei in the polychromatic erythrocytes of Swiss mice orally administered a single dose of the chemical at 50, 250 or 500 mg/kg bw or CD-1 mice given an intraperitoneal (i.p.) injection of the chemical at 250 mg/kg bw; and
- a UDS assay in SD rats orally administered the chemical at 100, 600 or 1600 mg/kg bw.

Carcinogenicity

The chemical is classified as a Category 3 carcinogenic substance with the risk phrase 'Limited evidence of carcinogenic effect' (Xn; R40) in the HSIS (Safe Work Australia). The available data support this classification.

The IARC has classified the chemical as 'Possibly carcinogenic to humans' (Group 2B), based on inadequate evidence for carcinogenicity in humans, but sufficient evidence for carcinogenicity in animals. According to the IARC, the mechanism of carcinogenicity relies on the rates of metabolism of the chemical, depending on the tissue exposed and animal species involved. Higher rates of metabolism in mice led to cytotoxic metabolites in the lungs, causing lung tumours, while high rates of metabolism in rat nasal epithelium similarly led to nasal tissue damage and nasal tumours (IARC, 2002). A recent study on *Caenorhabditis elegans* suggested that the chemical, known as a non-genotoxic carcinogen, could inhibit apoptosis, promoting the survival and proliferation of latent tumour cells (Kokel et al., 2006).

In a 104-week carcinogenicity study, groups of F344 rats (n = 49/sex/dose) were exposed (whole body) to vapours of the chemical at 0, 10, 30 or 60 ppm, six hours/day, five days/week. The NTP concluded there was 'clear evidence of carcinogenic activity' based on treatment-related increased incidences of respiratory epithelial adenoma and olfactory epithelial neuroblastoma of the nose in male and female rats (NTP, 2000).

In another 104-week carcinogenicity study, groups of B6C3F1 mice (n = 75/sex/dose) were exposed (whole body) to vapours of the chemical at doses of 0, 10 or 30 ppm (equivalent to 0, 50 and 150 mg/m^3), six hours/day, five days/week. The NTP concluded there was 'some evidence of carcinogenic activity' in female mice but not in male mice, based on the statistically significant increase in the incidence of pulmonary alveolar/bronchiolar adenomas in the high-dosed females (22 % compared with 7 % in the control group) (NTP, 1992).

Reproductive and Developmental Toxicity

Based on the available data, the chemical is not considered to have reproductive or developmental toxicity. Due to effective absorption through the placental barrier, the chemical can cause haemolytic anaemia in the human foetus following ingestion by the mother. Apart from this effect, the chemical has not shown specific developmental toxicity.

No reliable animal data on fertility were available. However, in a carcinogenicity study in mice (see **Carcinogenicity**), no histopathological changes in the epididymis, prostate, seminal vesicle, testes or ovaries were observed following inhalation of the chemical at 30 ppm (NTP, 1992).

The case of a newborn baby suffering from haemolytic anaemia was reported following the ingestion of the chemical (in mothballs) by the mother during pregnancy. The mother was G6PD-deficient and also suffered from haemolytic anaemia (EU RAR, 2003).

A similar case was reported in which a 26-year-old woman ingested an undetermined amount of the chemical during the last trimester of pregnancy, giving birth to a boy suffering from haemolytic anaemia (EU RAR, 2003).

Groups of rabbits were orally administered the chemical at doses of 0, 40, 200 or 400 mg/kg bw/day during gestation days (GD) 6-18. Although maternal toxicity was observed at the two highest doses (dyspnoea, cyanosis, salivation and abortions), no developmental effects were reported at the doses tested (EU RAR, 2003).

Female SD rats (n=25/dose) were orally administered the chemical at 0, 50, 150 or 450 mg/kg bw/day during GD 6–15. Maternal toxicity was reported at the two highest doses, including decreased maternal body weight gains, lethargy and slow respiration rates. At the highest dose, there was a two-fold increase in the number of resorptions per litter, a slight increase in the number of litters with visceral malformations and slight dose-related increases in the percentage of foetuses per litter with visceral malformations. However, these developmental changes were not statistically significant. The EU report (2003) concluded that the observed foetotoxicity was secondary to maternal toxicity (EU RAR, 2003).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- systemic acute effects from oral exposure;
- local effects on the respiratory tract following repeated inhalation exposure; and
- systemic long-term effects (carcinogenicity).

Public Risk Characterisation

Given the uses identified for the chemical, it is unlikely that the public will be exposed to the chemical from its industrial uses.

The chemical is listed on Schedule 6 of the SUSMP for 'NAPHTHALENE (excluding its derivatives) except in liquid hydrocarbons as an impurity'. The current controls are considered adequate to minimise the risk to the public from any domestic uses of the chemical. Therefore, the chemical is not

considered to pose an unreasonable risk to public health.

Occupational Risk Characterisation

During product formulation, oral and inhalation exposure might occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemical at lower concentrations could also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

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

The data available support an amendment to the hazard classification in the (HSIS (Safe Work Australia) (refer to **Recommendation** section).

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, 2015).

Work Health and Safety

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

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)*	Harmful if swallowed - Cat. 4 (H302)
Irritation / Corrosivity	Irritating to respiratory system (Xi; R37)	May cause respiratory irritation - Specific target organ tox, single exp Cat. 3 (H335)
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 oral and 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 storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical is used. Examples of control measures which could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- 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 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

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