

Benzene, (1-methylethenyl)-: Human health tier II assessment

01 September 2015



CAS Number: 98-83-9

- Preface
- Chemical Identity
- Import, Manufacture and Use
- Restrictions
- Existing Work Health and Safety Controls
- Health Hazard Information
- Risk Characterisation
- NICNAS Recommendation
- References

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.

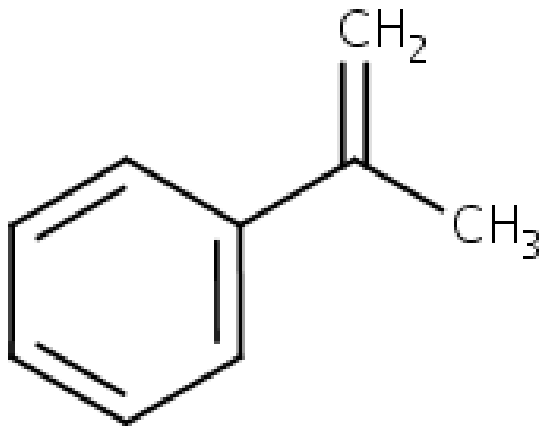
For more detail on this program please visit: www.nicnas.gov.au

Disclaimer

NICNAS has made every effort to assure the quality of information available in this report. However, before relying on it for a specific purpose, users should obtain advice relevant to their particular circumstances. This report has been prepared by NICNAS using a range of sources, including information from databases maintained by third parties, which include data supplied by industry. NICNAS has not verified and cannot guarantee the correctness of all information obtained from those databases. Reproduction or further distribution of this information may be subject to copyright protection. Use of this information without obtaining the permission from the owner(s) of the respective information might violate the rights of the owner. NICNAS does not take any responsibility whatsoever for any copyright or other infringements that may be caused by using this information.

Acronyms & Abbreviations

Chemical Identity

Synonyms	alpha-methylstyrene (AMS) 2-phenylpropene styrene, .alpha.-methyl- isopropenylbenzene
Structural Formula	
Molecular Formula	C ₉ H ₁₀
Molecular Weight (g/mol)	118.1
Appearance and Odour (where available)	colourless liquid with a characteristic odour
SMILES	<chem>C(=C)(C)c1ccccc1</chem>

Import, Manufacture and Use

Australian

The chemical is listed under 'Aromatic Hydrocarbons' on the 2006 High Volume Industrial Chemicals List (HVICL) with a total reported volume between 10,000–99,999 tonnes.

The chemical has reported commercial uses in fuels and oils.

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 OECD High Production Volume chemical program (OECD HPV); the US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR); the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and various international assessments including National Toxicology Program (NTP, 2007); the organisation for Economic Co-operation and Development Screening information dataset International Assessment Report (OECD SIAR) and International Agency for Research on Cancer (IARC, 2013).

The chemical has reported site-limited uses, as an intermediate in:

- manufacturing and processing of polymers;
- production and processing of rubber;
- paints, varnishes and coatings;
- construction materials;
- plastic production;
- corrosion inhibitors;
- printing inks; and
- surface-active agents.

Restrictions

Australian

This chemical is listed in the *Poisons Standard—the Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP)—under '**Hydrocarbons, Liquid**' in Schedule 5 (SUSMP, 2015).

Schedule 5:

'**Hydrocarbons, Liquid**, including kerosene, diesel (distillate), mineral turpentine, white petroleum spirit, toluene, xylene and light mineral and paraffin oils (but excluding their derivatives), except:

- (a) toluene and xylene when included in Schedule 6;
- (b) benzene and liquid aromatic hydrocarbons when included in Schedule 7;
- (c) food grade and pharmaceutical grade white mineral oils;

(d) in solid or semi-solid preparations;

(e) in preparations containing 25 per cent or less of designated solvents;

(f) in preparations packed in pressurised spray packs;

(g) in adhesives packed in containers each containing 50 g or less of adhesive;

(h) in writing correction fluids and thinners for writing corrections fluids packed in containers having a capacity of 20 mL or less;
or

(i) in other preparations when packed in containers with a capacity of 2 mL or less.'

Schedule 5 chemicals are described as 'Substances with a low potential for causing harm, the extent of which can be reduced through the use of appropriate packaging with simple warnings and safety directions on the label.'-Schedule 5 chemicals are labelled with 'Caution.' (SUSMP, 2015).

International

The chemical is restricted under Annex XVII to EU REACH Regulation (EC) No 1907/2006. The chemical cannot be used in substances and preparations placed on the market for sale to the general public in individual concentrations $\geq 0.1\%$.

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):

- Xi; R36/37 (irritation)

Exposure Standards

Australian

The chemical has an exposure standard of 242 mg/m^3 (50 ppm) time weighted average (TWA) and 483 mg/m^3 (100 ppm) short-term exposure limit (STEL).

International

The following exposure standards are identified (Galleria Chemica).

An exposure limit of $48\text{--}242 \text{ mg/m}^3$ (10 ppm) TWA and $240\text{--}485 \text{ mg/m}^3$ (50-100 ppm) STEL/MAK/occupational exposure limit (OEL) in various countries such as the United States of America (USA) (Alaska, Hawaii), Abu Dhabi, Austria, Belgium, Canada (Yukon), Norway and Switzerland.

The American Conference of Governmental Industrial Hygienists (ACGIH, 2005) recommends a threshold limit value (TLV) of 50 ppm TWA and 100 ppm STEL.

Health Hazard Information

Toxicokinetics

The metabolism of the chemical, hereafter referred to as α -methylstyrene (AMS), involves non-stereoselective epoxidation followed by hydrolysis to form 2-phenyl-1,2-propanediol, which is further oxidised to atrolactic acid or conjugates with glucuronide and with glutathione which then undergoes cleavage to form mercapturate. The mercapturate is further oxidised to yield 2-phenylpropionic acid (OECD, 1998; De Costa et al, 2001; NTP, 2007; REACH).

Radio-labelled AMS was administered in male Fischer 344/N (F344/N) rats via intravenous or nose-only inhalation exposure. Following inhalation, the highest concentration of AMS-derived radioactivity was found in the adipose tissue, urinary bladder, liver, kidney and skin. Following intravenous exposure, the highest concentration of AMS-derived radioactivity was found in the kidney, heart, lung, liver, urinary bladder and spleen. Approximately 90 % of the administered chemical was eliminated in urine within 72 hours and 1-3 % was excreted in the faeces or as volatile breath. In an inhalation study, the major metabolites found in blood were 2-phenyl-1,2- propanediol and 2-phenylpropanoic acid. The elimination half-life of AMS, in an inhalation study, was calculated to be 3 to 5 hours. The major urinary metabolites for AMS in both studies, were the glucuronide conjugate of 2-phenyl-1,2-propanediol and atrolactic acid (OECD, 1998; De Costa et al, 2001; NTP, 2007; REACH).

Acute Toxicity

Oral

The chemical AMS has low acute toxicity based on results from animal tests following oral exposure. The median lethal dose (LD50) in rats is >2000 mg/kg bw. Observed sub-lethal effects included lethargy, increasing weakness, reduced appetite, salivation, collapse and death.

In an acute toxicity study, AMS was administered once via gavage to male Wistar rats (20 animals/dose) and observations were made for 14 days. The LD50 for the chemical was calculated to be 4900 mg/kg bw (OECD, 1998; REACH).

In another study in male Wistar-derived rats (five animals/dose), AMS was administered via gavage (in doses equivalent to 3640-14560 mg/kg bw) once. Clinical signs observed included rubbing the mouth on the bottom of the cage, sluggishness, unsteady gait and prostrate position until death. The LD50 was reported to be 5915 mg/kg bw. Gross pathology revealed slight petechial haemorrhages in the lungs; mottled livers and spleens; distended stomach filled with liquid, gas and pylorus; gas and liquid filled yellow coloured intestines; slightly congested kidneys and full urinary bladders. The surviving animals also had mottled livers (REACH).

The chemical AMS was administered in Sprague Dawley (SD) rats (mixed groups of five males and five females) once by gavage at 7940, 10000, 12600 or 15800 mg/kg bw. All animals showed reduced appetite and activity, increasing weakness, collapse and in many cases progressing to death. Gross pathology examination showed haemorrhagic lungs, discolouration of the liver and inflammation of the gastro-intestinal tract. The oral LD50 was reported to be \leq 10000 mg/kg bw. Some surviving animals had areas of lung congestion (REACH).

Dermal

The chemical has low acute toxicity based on results from animal tests following dermal exposure. The median lethal dose (LD50) in rats is >2000 mg/kg bw. Observed sub-lethal effects included dermal erythema, hiccup-like spasms, and scaly and leathery skin.

In a dermal toxicity study, AMS was applied (approximately 7280 mg/kg bw in two rabbits and 14560 mg/kg bw in seven rabbits) to clipped intact skin of male albino rabbits under occlusive conditions for a 24 hour contact period. The LD50 value was determined to be >14560 mg/kg bw. Pathological examination revealed congested spleen and kidneys (OECD, 1998; REACH).

In another dermal toxicity study, AMS was applied under semiocclusive conditions to clipped intact skin of New Zealand White rabbits (one animal/sex/dose) at 501, 794, 2010, 3160, 5010 or 7940 mg/kg bw for up to a 24 hour exposure period. Reduced appetite and activity was observed during days 2-5 days in all animals. Female rabbits showed greater weight loss as compared to male rabbits. The LD50 was determined to be > 7940 mg/kg bw (REACH).

Inhalation

The chemical has low acute toxicity based on results from animal tests following inhalation exposure. The median lethal concentration (LC50) in rats is 22.85 mg/L. Observed sub-lethal effects included lethargy, slight ocular discharge, changes to respiration, increasing weakness, changes in motor activity, collapse and death.

In an acute inhalation toxicity study, male SD rats (six animals/dose) were exposed to AMS via a whole body vapour inhalation exposure at a concentration of 22.85 mg/L for 6 hours. Clinical observations during exposure included slight discomfort, slow reflexes, slight ocular discharge, lethargy, initial rapid and shallow respiration, collapse, roughened fur and wet bodies. Three animals died 48 hours after exposure. All survivors appeared to be normal 10 days after exposure. Necropsy examination revealed haemorrhagic lungs, slight liver discolouration and acute gastro-intestinal inflammation. The median lethal concentration (LC50) was calculated to be 22.85 mg/L air for a 6 hour exposure period (REACH).

Corrosion / Irritation

Respiratory Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to respiratory system' (Xi; R37) in the Hazardous Substances Information System (HSIS) (Safe Work Australia). Available human data to support this classification.

Skin Irritation

The chemical is reported to be slightly irritating to skin, particularly following repeated exposure. The irritation effects were not sufficient to warrant a hazard classification.

In a skin irritation study, AMS (0.5 mL; undiluted) was applied under semi-occlusive patch to the clipped, intact skin of New Zealand White rabbits (three male and three females) for four hours. Slight erythema and slight oedema were observed at 72 hours, but these were completely reversed at 120 and 168 hours of observation (REACH).

In another skin irritation study, AMS (0.01 mL; undiluted) was applied to shaved skins of five rabbits (strain specified) for 24 hours. All five animals had marked capillary injection at 24 hours of observation and the chemical was reported to be moderately irritating (REACH).

Eye Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to eyes' (Xi; R36) in HSIS (Safe Work Australia). The available data support this classification.

In an eye irritation study, AMS (two drops, 99.1 % purity) was instilled into the right eye of rabbits (number of animals not known). Conjunctival irritation which persisted up to seven days after exposure was reported (REACH).

In another eye irritation study in rabbits, the chemical was found to be slightly irritating with conjunctivitis observed up to 7 days after application (REACH).

Observation in humans

Humans volunteers exposed to AMS at concentrations between 10-600 ppm showed eye and nasal irritation at concentration ≥ 600 ppm (approximately 2900 mg/m³) (REACH).

Sensitisation

Skin Sensitisation

No data are available.

Repeated Dose Toxicity

Oral

In a 43-day combined repeated dose and reproductive toxicity oral gavage study conducted in accordance with OECD Test Guideline (TG) 422, Crj: CD(SD) rats (both sexes) were administered AMS at doses of 40, 200 or 1000 mg/kg/day. Males in the 1000 mg/kg/day showed decrease in body weight gain and food consumption. One male died due to ischuria (urinary retention) with urinary calculi. Decrease in body weight gain was also observed in females at 1000 mg/kg/day during late gestation period.

Changes observed at 1000 mg/kg/day in both sexes of the rats included increased liver and kidney weights, acidophilic change of the hepatocytes, increase of fatty droplets in the fascicular zone of the adrenals, increased renal hyaline droplets, and basophilic changes in the renal tubular epithelium. Changes observed in males included formation of urinary calculi and hyperplasia of the mucosal epithelium in the urinary bladder. Increases in alanine transaminase (ALT), urea, nitrogen and potassium and a decrease in triglyceride levels were reported in clinical biochemical examination in males. Effects observed in females included vacuolation and infiltration of lymphocytes in the renal tubular epithelium and atrophy of the thymus.

Effects observed at 200 mg/kg bw/day included histopathological changes in the liver and kidney in both sexes of the rats, increase in GPT levels in males and changes in the thymus in females. A lowest observed adverse effect level (LOAEL) of 200 mg/kg bw/day was reported (OECD, 1998; REACH).

Dermal

No data are available.

Inhalation

In a 14-week inhalation study similar to OECD TG 413, F344/N rats (10/sex/dose) were exposed by whole-body inhalation exposure to AMS at concentrations of 0, 75, 150, 300, 600 or 1000 ppm for 6 hours/day, 5 days/week for 14 weeks. Significant increases in absolute kidney weights were observed in the 1000 ppm dose group in both sexes and in females in 600 dose group. Significant increases in liver weights were observed in females at ≥ 600 ppm and in males at ≥ 150 ppm. Accumulation of hyaline droplets in the renal proximal tubules was observed in males of all treated groups, with a dose dependent increase in irregularity in shapes of the hyaline droplets. At concentrations of 150 ppm and higher, increased renal cell proliferation was reported. A no observed adverse effect concentration (NOAEC) of 150 ppm was reported based on the liver and kidney effects in rats in both sexes (NTP, 2007; REACH).

Genotoxicity

Based on the weight of evidence from the available in vitro and in vivo genotoxicity studies, the chemical is not considered to be genotoxic. Available in vitro (bacterial gene mutation assays; sister chromatid exchange assays and chromosomal aberration tests) and in vivo (micronucleus assay) tests for gene mutation and clastogenicity were negative.

In vitro studies

In a bacterial assay similar to OECD TG471, AMS at concentrations up to 400 µg/plate did not induce mutagenic activity in *Salmonella typhimurium* TA98, TA100, TA1535 and TA 1537 with and without metabolic activation (OECD, 1998; HSDB; REACH).

In a chromosomal aberration test (OECD TG473), AMS was tested in Chinese hamster lung cells at concentrations up to 0.23 mg/mL. The chemical did not induce chromosomal aberration in mammalian cells (OECD, 1998; HSDB; REACH).

In vivo studies

In a micronucleus assay (OECD TG 474), B6C3F1 (10/sex/dose) mice were exposed to AMS via whole body inhalation exposure at concentrations of 0, 75, 150, 300, 600 or 1000 ppm, 6 hours/day for 5 day/week for 14 weeks. The chemical did not induce increases in the number of micronucleated polychromatic erythrocytes in this assay (OECD, 1998; HSDB; REACH).

Carcinogenicity

Based on the available information, a hazard classification for carcinogenicity is warranted. The International Agency for Research on Cancer (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.

In a two-year study similar to OECD TG 451, F344/N rats (groups of 50/sex) were exposed by whole-body inhalation exposure to AMS at concentrations of 0, 100, 300 or 1000 ppm for 6 hours/day, 5 days/week for 105 weeks. Renal tubule carcinomas were observed in two males in the 1000 ppm dose group and a male administered 300 ppm. One male in the 300 ppm group had renal tubule adenoma. Significantly greater incidences of renal tubule adenomas and carcinomas along with increased incidences of mineralisation of the renal papilla were observed in males at 1000 ppm dose. However, based on the observations of hyaline droplets consistent with the alpha-globulin nephrotoxicity (which is specific to male rats) in repeat dose toxicity studies, the renal toxicity rate in male rats is likely to increase from this mechanism, which is not considered relevant to humans. Increased incidence of mononuclear cell leukaemia was also reported. At 100 ppm, increased incidence of basal cell hyperplasia in the nose and increased incidence of degeneration of the olfactory epithelium were reported in both sexes. The lowest observed adverse effect concentration (LOAEC) was determined to be 100 ppm (NTP, 2007; REACH).

A two-year study conducted according to OECD TG 451, B6C3F1 mice (groups of 50/sex/dose) were exposed by inhalation exposure to AMS at concentrations of 0, 100, 300 or 600 ppm for 6 hours/day, 5 days/week for 105 weeks. After week 13, decreases in the mean body weights were reported at 600 ppm in both sexes. Significantly higher incidences of eosinophilic foci of the liver are also reported in high dose females. High dose females had increased incidence and severity of nephropathy. Increased incidences of hepatocellular adenoma and/or carcinomas were reported in all treated animals, except for males in the mid-dose group. Males in the mid and high dose groups also showed significantly increased atrophy of the olfactory epithelium. All treated animals showed increased incidences of olfactory epithelial metaplasia and hyperplasia of the glands overlying the olfactory epithelium. A LOAEC was 100 ppm was reported for this study (NTP, 2007; REACH).

Reproductive and Developmental Toxicity

Based on the available information, the chemical does not show specific reproductive or developmental toxicity.

In a reproductive toxicity study conducted according to OECD TG 422, AMS was administered to SD (Crj:CD) rats (10 /sex/dose) at doses of 0, 40, 200 or 1000 mg/kg bw/day for 43 days by oral administration. Males were treated from 14 days prior to mating and continued for a few days after mating. Females were treated from gestation period to delivery and 3 days post-partum. No effects on the mating index, fertility index, gestation period, number of corpora lutea, implantation index, gestation index, delivery index or parturition were observed. Decrease in body weights and a slight decrease in the viability index were observed in the neonates at 1000 mg/kg bw/day on day 4 post-partum. No significant changes were reported in the numbers of live offsprings at birth, sex ratio, the live birth index, body weight gain after birth and necropsy of the offspring. The

no observed effect levels (NOELs) were 1000 mg/kg bw/day for paternal, 200 mg/kg bw/day for maternal and offspring (OECD, 1998; REACH).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effect (carcinogenicity). The chemical can also cause harmful effects following repeated oral and inhalation, as well as eye irritation.

Public Risk Characterisation

Given the uses identified for the chemical, the main routes of public exposure are expected to be through the skin, eye and inhalation from products containing the chemical. The chemical is listed in Schedule 5 of the SUSMP. A number of warning statements, first aid instructions and safety directions apply. These controls are considered adequate to minimise the risk to public health posed by the use of products containing the chemical. Therefore, the chemical is not considered to pose an unreasonable risk to the public.

Occupational Risk Characterisation

During product formulation, dermal, ocular and inhalation 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 and local health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise dermal, ocular and inhalation 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.

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
--------	---------------------------------------	--

Hazard	Approved Criteria (HSIS) ^a	GHS Classification (HCIS) ^b
Irritation / Corrosivity	Irritating to eyes (Xi; R36)* Irritating to respiratory system (Xi; R37)*	Causes serious eye irritation - Cat. 2A (H319) 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 industry

Control measures

Control measures to minimise the risk from ocular 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 that 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

De Costa KS, Black SR, Thomas BF, Burgess JP and Mathews JM (2001). Metabolism and disposition of alpha-methylstyrene in rats. *Drug Metabolism and Disposition* 2001 FEB;29 (2): 166-71.

Hazardous Substances Data Bank (HSDB). US National Library of Medicine. Accessed July 2015 at <http://toxnet.nlm.nih.gov>

International Agency for Research on Cancer (IARC) 2013. alpha-methylstyrene, IARC Monographs Volume 101. Accessed July 2015 at http://monographs.iarc.fr/ENG/Classification/vol1_113.php

IPCS Environmental Health Criteria (EHC) 0732 (2005). Accessed July 2015 at <http://www.inchem.org/documents/icsc/icsc/eics0732.htm>

National Toxicology Program (NTP) 2007. US Department of Health and Human Services. Technical Report on the Toxicology and Carcinogenesis Studies of Alpha-methylstyrene (CAS No. 98-83-9) in F344/N rats and B6C3F1 mice (Inhalation Studies). Accessed July 2015 at http://ntp.niehs.nih.gov/ntp/htdocs/lt_rpts/tr543.pdf

Organisation for Economic Cooperation and Development (OECD) 1998. SIDS Initial Assessment Report (SIAR) on (1-methylethenyl)benzene (CAS No. 98-83-9). Accessed July 2015 at <http://webnet.oecd.org/Hpv/UI/handler.axd?id=3aba311a-5963-4ed1-991b-84e48a582731>

Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) Dossier. 98-83-9. Accessed July 2015 at http://apps.echa.europa.eu/registered/data/dossiers/DISS-9d9a4808-4c5f-28db-e044-00144f67d249/DISS-9d9a4808-4c5f-28db-e044-00144f67d249_DISS-9d9a4808-4c5f-28db-e044-00144f67d249.html

Safe Work Australia (SWA). Hazardous Substances Information system (HSIS). Accessed July 2015 at <http://hsis.safeworkaustralia.gov.au/HazardousSubstance>

SPIN (Substances in Preparations in Nordic Countries) Database. Accessed July 2015 at <http://195.215.202.233/DotNetNuke/default.aspx>

US Environmental Protection Agency's (EPA) Aggregated Computational Toxicology Resource (ACToR). Accessed July 2015 at <http://actor.epa.gov/actor/faces/ACToRHome.jsp>

Last update 01 September 2015

Share this page