Oxirane, (chloromethyl)-: Human health tier II assessment

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CAS Number: 106-89-8

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	epichlorohydrin 1-Chloro-2,3-epoxypropane ?-chloropropylene oxide 2-(chloromethyl)oxirane a-Epichlorohydrin
Structural Formula	
Molecular Formula	C3H5ClO
Molecular Weight (g/mol)	92.53
Appearance and Odour (where available)	Colourless liquid with a chloroform-like odour
SMILES	C1(CCl)CO1

Import, Manufacture and Use

Australian

The following Australian industrial uses were reported under previous mandatory and/or voluntary calls for information.

The chemical has reported commercial use including as a solvent for resins, gums, cellulose, esters, paints, and lacquers.

The chemical has reported site-limited use including:

- as a stabiliser in chlorine-containing substances such as rubber; and
- manufacturing of elastomers, glycidyl ethers, and a variety of resins.

International

The following international uses have been identified through European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) dossiers; the Organisation for Economic Cooperation and Development Screening information data set International Assessment Report (OECD SIAR); Galleria Chemica; Substances and preparations in the Nordic countries (SPIN) database; the US Environmental Protection Agency's Aggregated Computer Toxicology Resource—ACToR, and the US National Library of Medicine's Hazardous Substances Data Bank—HSDB.

The chemical has reported commercial use including:

- as a solvent for resins, gums, cellulose, paints, and lacquers;
- in curing propylene-based rubbers; and
- as a starch-modifying agent (historically).

The chemical has reported site-limited use including:

- as an intermediate in the production of epoxy and phenoxy resins, synthetic glycerines and glycerols, glycidyl ethers and methacrylates, flame retardants, and quaternary amines;
- in the production of food contact materials;
- in the production of elastomers, surfactants, plasticisers, and dyestuffs.

Restrictions

Australian

This chemical is listed in The Poisons Standard (Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP, 2012)) in Schedule 7 and Appendix J.

Schedule 7 chemicals are labelled with 'Dangerous Poison'. These are substances with a high potential for causing harm at low exposure and which require special precautions during manufacture, handling or use. These poisons should be available only to specialised or authorised users who have the skills necessary to handle them safely. Special regulations restricting their availability, possession, storage or use may apply.

This chemical is not to be available except to authorised or licensed persons under Appendix J in the SUSMP.

This chemical is listed on the 2011 Australian Drinking Water Guidelines (ADWG) with a health guideline value of 0.0005 mg/L in the unlikely event of a contamination (NHMRC, 2011).

International

This chemical is listed in the following international directives/standards to restrict use in cosmetic products:

- ASEAN Cosmetic Directive Annex II Part 1: List of substances which must not form part of the composition of cosmetic products;
- EU Cosmetic Directive Annex II: List of substances which must not form part of the composition of cosmetic products;
- New Zealand Cosmetic Product Group Standard 2006 (as amended 17 Nov, 2011) Schedule 4: Components cosmetic products must not contain; and
- Canada list of prohibited and restricted cosmetic ingredients.

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):

R23/24/25: Toxic by inhalation, in contact with skin and if swallowed

R34: Causes burns

R43: May cause sensitisation by skin contact

R45: May cause cancer

Exposure Standards

Australian

The chemical has an exposure standard of 7.6 mg/m³ (2 ppm) time weighted average (TWA).

International

The following exposure standards are identified (Galleria Chemica):

An exposure limit (TWA) of 2–20 mg/m³ (0.5–5 ppm) in different countries such as USA (Hawaii, Minnesota), Canada (Yukon), Bulgaria, Germany, Greece, Norway and Switzerland.

Health Hazard Information

Toxicokinetics

The chemical was well absorbed following oral, dermal and inhalation exposure in animal studies (EHC, 1984). Rapid distribution of this chemical to various tissues was observed post-absorption. Following oral ingestion of the chemical, concentrations found in blood were exceeded by a factor of two or more in the stomach, intestine, kidneys, prostate and lacrimal glands, and the liver. Direct inhalation of this chemical showed increased concentrations in the nasal turbinates, lacrimal glands, kidneys, liver, and large intestines (EHC, 1984). The chemical is a reactive epoxide, which is metabolised through glutathione-binding and hydrolysis via epoxide hydrolase (IARC, 1999). Regardless of the exposure route, approximately 90 % of the absorbed chemical is excreted within 72 hours as either carbon dioxide through the lungs or as metabolites in the urine (EHC, 1984).

Acute Toxicity

Oral

The chemical is classified as hazardous with the risk phrase ‘Toxic if swallowed’ (T; R25) in HSIS (Safe Work Australia). The available data support this classification.

In experiments that were conducted in Sprague Dawley (SD) and Fischer 344 rats (five animals/sex/dose), the chemical was orally administered at 25, 50, 100, 200, 398, and 795 mg/kg bw. Additionally, Fischer 344 rats were administered the chemical at 210, 225, and 252 mg/kg bw. The animals were observed for 14 days; lethargy was the main clinical sign that was observed. Mortality in SD rats was 1/5, 4/5 and 5/5 for males and 2/5, 5/5, and 5/5 for females at 200, 398 and 795 mg/kg, respectively. No deaths were noted for lower doses. In Fischer 344 rats, all males and females in every dose group died, except for the 210 mg/kg bw group, where 2/5 females survived. The LD50 value was found to be 175–282 mg/kg bw (OECD, 2009; REACH).

Dermal

The chemical is classified as hazardous with the risk phrase ‘Toxic in contact with skin’ (T; R24) in HSIS (Safe Work Australia). While the available data (indicating moderate acute dermal toxicity) do not support this classification, in the absence of more comprehensive information, there is insufficient evidence to support a recommendation to amend this classification.

In an experiment conducted in New Zealand White rabbits (two animals/sex/dose), the chemical was administered at doses of 100, 200, 465, or 795 mg/kg bw for 24 hours. Mortalities were observed in the 465 mg/kg bw (males) and in the 795 mg/kg bw dose groups (males and females). Severe redness, swelling, and slight necrosis were noted after the 24-hour exposure period. The acute dermal LD50 was found to be 515 mg/kg bw (OECD, 2009; REACH).

Inhalation

The chemical is classified as hazardous with the risk phrase ‘Toxic by inhalation’ (T; R23) in HSIS (Safe Work Australia) and in GHS (H331) (EU CLP). While the available data do not support this classification, in the absence of more comprehensive information, there is insufficient evidence to support a recommendation to amend this classification.

Fischer 344 rats (six animals/sex/dose) were exposed to TWA analytical concentrations of 552, 1008, 1970, or 3995 ppm (2.08, 3.81, 7.45 or 15.11 mg/L, respectively) for one hour. Two additional groups, consisting of six male rats each, were also exposed to 2865 or 3275 ppm of the chemical (10.83 or 12.39 mg/L, respectively). All of the male animals in the 3995 ppm dose groups died within four days with no deaths occurring at other concentrations. Mortalities in the females were observed in the 1970 ppm and 3995 ppm dose groups. Clinical signs, which includes eye and nasal irritation, difficulty in respiration, and secretions around the facial area, were noted from the 1970 ppm to the 3995 ppm dose groups. It was determined that the one hour LC50 of this chemical in male rats was 3617 ppm. The one hour LC50 of this chemical in female rats was determined, by the moving average method of analysis, to be 2165 ppm (OECD, 2009; REACH).

Corrosion / Irritation

Corrosivity

The chemical is classified as hazardous with the risk phrase ‘Causes burns’ (C; R34) in HSIS (Safe Work Australia). The available data support this classification.

The chemical was applied in an occlusive patch on the skin of rabbits at 0.1 to 0.2 mL, for two hours, or 0.5 mL, for 24 hours. Application of the chemical at 0.5 mL for 24 hours showed lesions with a central zone of necrosis surrounded by hard swelling. Erythema of varying intensity was also observed around the periphery of the lesions. Smaller lesions were observed with the lower doses (0.1 mL to 0.2 mL for two hours) of the chemical. Eschar formation around the necrotic area was persistent for more than 30

days post-application, although healing proceeded normally by macroscopic observation. It was concluded that this chemical was corrosive to the skin of rabbits (OECD, 2009; REACH).

Respiratory Irritation

Evidence that the chemical causes respiratory irritation has been reported (OECD, 2009; REACH). In an inhalation study conducted in rats, nasal irritation and difficulty in respiration were noted as clinical signs post application of the chemical. In a chronic toxicity study conducted on rats, effects on the nasal turbinates of the animals were reported as treatment related effects (OECD, 2009; REACH).

Eye Irritation

Corrosive chemicals are considered to cause irreversible effects in the eyes. The data available support this conclusion. In an experiment conducted in rabbits, 0.001 mL of the chemical, undiluted or as a 0.1 % or 0.01 % solution in water, was applied to the eyes with observation for 24 hours. Ocular necrosis was observed on animals who were administered both the undiluted solution and the 0.1 % solution of the chemical (REACH). In an inhalation study conducted in rats, it was noted that bilateral corneal cloudiness was the main exposure-related lesion observed (OECD, 2009; REACH). Furthermore, eye and nasal irritation was also observed in this study.

Observation in humans

It was reported that workers experienced burning of the eyes and nasal mucosa when exposed to the chemical at a vapour concentration of 76 mg/m^3. Throat irritation, which lasts up to 48 hours, occurs at concentrations of 151 mg/m^3 (EHC, 1984). In the case of a 39 -year-old man who inhaled vapours of the chemical, slight irritation to the eyes and throat were reported along with headaches, nausea, and vomiting (EHC, 1984).

Sensitisation

Skin Sensitisation

The chemical is classified as hazardous with the risk phrase ‘May cause sensitisation by skin contact’ (R43) in HSIS (Safe Work Australia). The positive results reported in several guinea pig tests support this classification (OECD, 2009; REACH).

Observation in humans

It was reported that workers at an epoxy resin plant who were exposed to the chemical developed contact dermatitis (OECD, 2009). In one volunteer, the chemical, in 0.1–1.0 % solution in ethanol, was administered in an occluded patch of two days. Late reactions developed after 8–11 days. Erythema was immediately observed upon application of 0.01 % solution of the chemical at a challenge exposure after two days (EHC, 1984).

Repeated Dose Toxicity

Oral

Observed adverse effects following oral exposure to the chemical are principally related to local toxicity (irritation) at the site of contact.

In a 90-day chronic toxicity study conducted in SD rats (10 animals/sex/dose), the chemical was administered daily by oral gavage at doses of 0, 1, 5, or 25 mg/kg bw/day. No mortalities were observed in any of the dose groups throughout the study. Dose-related changes in the liver and kidneys, a decrease in red blood cell (RBC) parameters (including RBC counts, haemoglobin, and haematocrit levels) and thickening of the mucosal lining of the forestomach were observed in the 5 and 25 mg/kg bw/day dose groups. Histological

examination found that hyperkeratosis and hyperplasia (acanthosis) in the forestomach were the only treatment-related microscopic changes that were observed in the 5 and 25 mg/kg bw/day dose groups. The NOAEL for this 90-day study was determined to be 1 mg/kg bw/day (OECD, 2009; REACH).

Dermal

There are no reliable data available.

Inhalation

Observed adverse effects following oral exposure to the chemical are principally related to local toxicity (irritation) at the site of contact.

In an inhalation toxicity experiment conducted in rats (Fischer 344 and SD) and mice (B6C3F1) (20 animals/sex/dose/strain), the animals were exposed to vapours of the chemical at 0, 5, 25, and 50 ppm (or 1, 18.9, 94.5, and 189 mg/m³, respectively) for six hours a day, five days a week, for a duration of 12 weeks. After 30 days of testing, 10 animals/sex/group were sacrificed. No significant toxicological effects were observed in the 5 ppm dose groups. However, the following treatment-related effects were reported for the 25 and 50 ppm dose groups: adverse histopathological changes in the nasal turbinate epithelium; slight pathological changes in the kidney (observed in rats only), liver (rats and mice), adrenal glands (males of both species), and decreased body weight gain. The no observed adverse effect concentration (NOAEC) and lowest observed adverse effect concentration (LOAEC) for both species was determined to be 5 ppm and 25 ppm, respectively (OECD, 2009; REACH).

Although the NOAEC is sufficiently low to allow classification, as the effects were due to the irritant nature of the chemical and there is no significant evidence of systemic toxicity observed, no hazard classification for repeated dose inhalation toxicity is recommended. However, a classification for respiratory irritation is warranted.

Genotoxicity

The chemical is a direct-acting alkylating agent. The chemical is positive in a large number of in vitro and in vivo genotoxicity studies. The chemical has been reported to: bind to the DNA of mice and rats treated in vivo; induce sister chromatid exchanges in the bone marrow of partially hepatectomised CBA/J mice; induce chromosomal aberrations in mouse bone marrow; and cause sperm head abnormalities in rats, but not mice. The chemical does not appear to induce micronuclei or dominant lethal mutations in mice in vivo. Chromosomal aberrations have also been observed, in four separate studies, in workers occupationally exposed to the chemical; however, these observations may be confounded by the presence of other chemicals (Giri, 1997; IARC, 1999; OECD, 2009; REACH).

Based on the sperm head abnormalities in rats, the data available support classification for germ cell mutagenicity (see **Recommendation section**).

Carcinogenicity

The chemical is classified as hazardous—Category 2 carcinogenic substance—with the risk phrase ‘May cause cancer’ (T; R45) in HSIS (Safe Work Australia). The available data support this classification.

The International Agency for Research on Cancer (IARC) has classified the chemical as ‘Probably carcinogenic to humans’ (Group 2A), based on inadequate evidence for carcinogenicity in humans, but sufficient evidence for carcinogenicity based on animal testing (IARC, 1999).

The chemical is also listed by the US National Toxicology Program Report on Carcinogens as 'Reasonably anticipated to be a human carcinogen' based on sufficient evidence of carcinogenicity from studies in experimental animals (NTP, 2011) and by the American Conference of Governmental Industrial Hygienists (ACGIH) as an A3 carcinogen (confirmed animal carcinogen with unknown relevance to humans) (ACGIH, 2011).

Investigations on four human populations found a weak association between lung cancer and exposure to the chemical (IARC, 1999). A relationship between the central nervous system tumours and exposure to the chemical was observed in 11 cases (OECD, 2009). However, extrapolation from these data was limited due to the small cohort size and confounding factors. Animals studies have shown

that administration of the chemical through the oral and inhalation route produces papillomas (in the forestomach and nasal cavity) and carcinomas (in the nasal cavity) (IARC, 1999).

Reproductive and Developmental Toxicity

The chemical had effects on male fertility in experiments conducted in rats following oral and inhalation exposure. There is no evidence of reproductive toxicity in females and the observed developmental effects were only secondary to maternal toxicity (OECD, 2009; REACH).

In an inhalation study conducted in rats, a NOAEL for male reproduction of 5 ppm (18.9 mg/m³) was established based on a decreased male fertility index (8–16 % of the males bred successfully) compared with the control index (100%) at 50 ppm. In addition, a decrease in the number of implantations in unexposed females mated with rats exposed to the two highest doses was also reported. These effects were reversible after 10 weeks of no exposure. No effects were observed in a similar study in rabbits (OECD, 2009; REACH).

Male infertility and effects on sperm have been observed in a number of oral studies in rats. A lowest observed adverse effect level (LOAEL) of 3.3 mg/kg bw/day was established from these studies. The reversibility of effects was not consistently observed, although this appeared to be influenced by dose and frequency of exposure. Morphologically abnormal sperm has been reported following limited exposure (< 5 times) to doses of 50 mg/kg bw/day and higher, and histopathological changes of the testes (at 10 and 30 mg/kg bw/day) and epididymis (at 10 mg/kg bw/day), have been reported in a one-generation study (OECD, 2009; REACH).

Reproductive effects in humans have not been established based on studies in workers exposed to the chemical (OECD, 2009).

Developmental toxicity was only evident at doses where maternal toxicity was evident. A decrease in foetal body weight was reported in mice who were given oral doses of 120 mg/kg bw/d of the chemical. No other treatment-related developmental effects were reported. The maternal and developmental NOAEL was 25 ppm (inhalation studies) and 80 mg/kg bw/day (oral studies) (OECD, 2009; REACH).

The data available support classification for toxicity to fertility (see **Recommendation section**).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (carcinogenicity and reproductive toxicity), systemic acute effects (acute toxicity by oral, dermal and inhalation exposure) and local effects (corrosivity, skin sensitisation and respiratory irritation).

A genotoxic mode of action for carcinogenicity cannot be precluded.

Public Risk Characterisation

Given the uses identified for the chemical, public exposure is expected to be minimal. The chemical is used in the manufacture of consumer products and food contact materials; however, it is expected that any residual traces of the chemical would only be present in minimal amounts. Hence, the public risk from this chemical is not considered to be unreasonable.

Occupational Risk Characterisation

During product formulation, dermal, ocular and inhalation exposure of workers to the chemical may occur, particularly where manual or open processes are used. These may include transfer and blending activities, quality control analysis, and cleaning and maintenance of equipment. Worker exposure to the chemical at lower concentrations may 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, systemic acute and local health effects, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise dermal, ocular and inhalation exposure to the chemical are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking at a workplace (such as an employer), has adequate information to determine appropriate controls. The data available support an amendment to the hazard classification in HSIS (see **Recommendation section**).

Although the Australian exposure standard (TWA 7.6 mg/m³ —2 ppm) lies within the range of identified international exposure standards (2–20 mg/m³ —0.5–5 ppm), based on the critical effects identified, exposures should be kept as low as possible. Therefore, the current exposure standard may not be adequate to mitigate the risk of adverse effects.

NICNAS Recommendation

The chemical is recommended for Tier III assessment to examine the adequacy of the current exposure standard.

All other aspects have been sufficiently assessed at the Tier II level 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 hazards and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Toxic if swallowed (T; R25)* Toxic in contact with skin (T; R24)* Toxic by inhalation (T; R23)*	Toxic if swallowed - Cat. 3 (H301) Toxic in contact with skin - Cat. 3 (H311) Toxic if inhaled - Cat. 3 (H331)
Irritation / Corrosivity	Irritating to respiratory system (Xi; R37) Causes burns (C; R34)*	May cause respiratory irritation - Specific target organ tox, single exp Cat. 3 (H335) Causes severe skin burns and eye damage - Cat. 1 (H314)
Sensitisation	May cause sensitisation by skin contact (Xi; R43)*	May cause an allergic skin reaction - Cat. 1 (H317)
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)

[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 dermal, 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 which may 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 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 the chemical are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (m)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of Safety Data Sheets for Hazardous Chemicals— Code of Practice* and *Labelling of Workplace Hazardous Chemicals—Code of Practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of the chemical has not been undertaken as part of this assessment.

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