Australian Government
Department of Health and Ageing
NICNAS

INVENTORY MULTI-TIERED ASSESSMENT AND PRIORITISATION (IMAP)

HUMAN HEALTH TIER II ASSESSMENT FOR

Oxirane, (phenoxymethyl)-

CAS Registry Number: 122-60-1

PREFACE

As part of the reform regarding assessment of Existing Chemicals, the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) is implementing a new framework to address the human health and environmental impacts of industrial chemicals, not yet assessed, on the Australian Inventory of Chemical Substances (AICS).

The framework provides a more rapid, flexible and transparent approach for the assessment of existing chemicals.

The Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework was developed, with significant input from stakeholders, and will be applied in stages.

Stage One of this program, which will take three years, started 1 July 2012 and is examining 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This includes chemicals for which NICNAS already holds exposure information, chemicals identified as a concern or for which regulatory action has been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

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.

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

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

ACRONYMS & ABBREVIATIONS

ACToR	Aggregated Computational Toxicology Resource (US)
AICS	Australian Inventory of Chemical Substances
ASTDR	Agency for Toxic Substances and Disease Registry (US)
bw	bodyweight
CAS	Chemical Abstracts Service
CFR	Code of Federal Regulations (US)
CHO	Chinese hamster ovary
CosIng	Cosmetic Ingredients and Substances database (EU)
d	day
DNA	Deoxyribonucleic acid
EC	European Commission
EC3	Estimated concentration three
ECHA	European Chemicals Agency
ESIS	European Chemical Substances Information System
EU	European Union
EU RAR	European Union Risk Assessment Report
FDA	Food and Drug Administration (US)
FSANZ	Food Standards Australia and New Zealand
g	gram
g/mol	grams per mole
GHS	Globally Harmonized System of Classification and Labelling of Chemicals*
GLP	Good Laboratory Practice
GMP	Good Manufacturing Practice
GPMT	Guinea Pig Maximisation Test
h	hour
HGPRT	hypoxanthine guanine phosphoribosyltransferase
HPV	high production volume
HSDB	Hazardous Substances Data Bank
HSIS	Hazardous Substances Information System
HVICL	High Volume Industrial Chemicals List
IARC	International Agency for Research on Cancer
INCHEM	International Programme on Chemical Safety (also known as IPCS)
INCI	International Nomenclature of Cosmetic Ingredients
ip	intraperitoneal
IRIS	Integrated Risk Information System (US)
IUCLID	International Uniform Chemical Information Database
iv	intravenous
kg	kilogram
L	litre
LC50	median lethal concentration
LD50	median lethal dose
LCLo	lowest published lethal concentration
LLNA	local lymph node assay
LOAEL	lowest observed adverse effect level
LOEL	lowest observed effect level
m^3	cubic metre
mg	milligram
mg/cm^3	milligrams per cubic centimetre
mg/kg bw/d	milligrams per kilogram bodyweight per day
min	minute
mL	millilitre
µg	microgram
µL	microlitre
(m)SDS	(material) Safety Data Sheet

NIOSH	National Institute for Occupational Safety and Health (US)
NOAEC	no observed adverse effect concentration
NOAEL	no observed adverse effect level
NOEC	no observed effect concentration
NOEL	no observed effect level
NOHSC	National Occupational Health and Safety Commission
NTP	National Toxicology Program (US)
OECD	Organisation for Economic Cooperation and Development
OEL	occupational exposure limit
PCBU	person conducting a business or undertaking
PEL	permissible exposure limit
PND	postnatal day
ppb	parts per billion
PPE	personal protective equipment
ppm	parts per million
REACH	Registration Evaluation Authorisation of Chemicals (ECHA)
SD	Sprague Dawley
SIAP	SIDS Initial Assessment Profile (OECD)
SIAR	SIDS Initial Assessment Report (OECD)
SIDS	Screening Information Data Set (OECD)
SMILES	simplified molecular-input line-entry system
SPIN	Substances in Preparations In the Nordic countries
STEL	short-term exposure limits
STV	short-term value
SUSMP	Standard for the Uniform Scheduling of Medicines and Poisons (The Poisons Standard**)
TCLo	lowest published toxic concentration
TEEL	temporary emergency exposure limits
TSCA	Toxic Substances Control Act (US EPA)
TG	test guideline
TGA	Therapeutic Goods Administration
TLV	threshold limit values
TWA	time weighted average
UN	United Nations
US	United States of America
US EPA	United States Environmental Protection Agency
WHS	Work, Health and Safety
wt	weight
w/w	weight per weight

Glossary

NICNAS uses the IPCS Risk Assessment Terminology (IPCS, 2004) glossary, which includes:
Part 1: IPCS/OECD Key Generic Terms used in Chemical Hazard/Risk Assessment; and
Part 2: IPCS Glossary of Key Exposure Assessment Terminology.
The IPCS Risk Assessment Terminology can be accessed at:
http://www.who.int/ipcs/methods/harmonization/areas/ipcsterminologyparts1and2.pdf

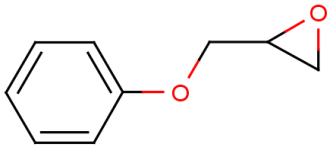
*****Globally Harmonised System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third edition. Can be accessed at: http://www.unece.org/trans/danger/publi/ghs/ghs_rev03/03files_e.html

******The Poisons Standard (the SUSMP) can be accessed at: http://www.tga.gov.au/industry/scheduling-poisons-standard.htm

Oxirane, (phenoxymethyl)-

CAS No: 122-60-1

Chemical Identity

Synonyms	Phenyl glycidyl ether 2,3-Epoxypropylphenylether PGE Phenoxymethyl oxirane
Structural Formula	
Molecular Formula	C9H10O2
Molecular Weight (g/mol)	150.2
Appearance and Odour (where available)	Colourless liquid with characteristic odour
SMILES	c1(OCC2CO2)ccccc1

Import, Manufacture and Use

Australian

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

International

The following international uses have been identified via Galleria Chemica, the Substances and Preparations In the Nordic countries (SPIN) and eChemPortal (the Aggregated Computer Toxicology Resources (ACTor) and Hazardous Substances Data Bank (HSDB)).

The chemical has reported domestic use including:
- in paints, lacquers and varnishes; and
- in adhesives and binding agents.

The chemical has reported commercial and site-limited use including:
- as a reactant in the production of industrial grade epoxy resins;
- component of epoxy resins/plasticiser for epoxy resins where it is an active diluent in uncured epoxy resins;
- in construction materials/specialised construction activities;
- as an intermediate (effective stabiliser for halogenated compounds due to it being an acid acceptor; and
- as a monomer for photoreactive polymers.

Restrictions

Australian

No known restrictions have been identified.

International

Use in cosmetics:
EU regulation (EC) No 1223/2009 Annex II: List of substances which must not form part of of the composition of cosmetic products. Classified as a category 1B carcinogen (Galleria Chemica); and

New Zealand Cosmetic Products Group Standard − Schedule 4: Components cosmetic products must not contain (Galleria Chemica).

Existing Worker Health And Safety Controls

Hazard classification

The chemical is currently classified on the Hazardous Substances Information System (HSIS) with the following (Safe Work Australia):
Xn; R20 (Acute toxicity);
Xi; R37/38 (Irritation);
Xi; R43 (Sensitisation);
T; R45 (Carcinogen Cat.2); and
Xn; R68 (Mutagen Cat.3).

Exposure standards

Australian

TWA (8 hours): 6.1 mg/m³ (1 ppm)

International

TWA: 60 mg/m³ (10 ppm) [Canada (Yukon), Argentina, US (OSHA)]
TWA: 60 mg/m³ (0.1 ppm) [Belgium, Argentina, US (ACGIH)]
STEL: 90 mg/m³ (15 ppm) [Canada (Yukon), Argentina, US (OSHA)]
Ceiling: 6 mg/m³ (1 ppm) [US (NIOSH RELs)]

Health Hazard Information

Toxicokinetics

Absorption, Distribution & Excretion:
Percutaneous absorption rates for the chemical were 13.5 mg/sq cm/hr in rats and 4.2 mg/sq cm/hr for rabbits (Czajkowska and Stekiewicz, 1972).

Acute dermal treatment of rats and rabbits with the chemical PGE showed that the substance is well absorbed through the skin (HSDB).

Acute Toxicity

Oral

The chemical has low acute toxicity via the oral route.

Reported LD50 in rats is 3850 mg/kg bw. Clinical signs included behavioural symptoms, mainly somnolence (general depressed activity), changes in motor activity and ataxia (loss of control over some movement). Reported LD50 in mice is 1400 mg/kg bw. The mice showed somnolence (general depressed activity) and behavioural ataxia (Galleria Chemica).

Dermal

The reported cutaneous LD50 in rats is 2100 mg/kg bw, indicating low acute dermal toxicity (IARC, 1989).

Inhalation

The chemical is currently classified with the risk phrase 'Harmful by inhalation' (R20) (Safe Work Australia). Although the data available do not warrant a hazard classification, in the absence of more reliable data and considering the local effects seen in the repeat dose inhalation toxicity study, the existing hazard classification was not amended.

The reported LC50 values in rats (Long-Evans/male) and mice (Webster/male) are >100 ppm/4 hour. Clinical signs observed were dyspnoea (difficulty breathing), watering eyes, salivation, rhinitis and aerophagy (excessive swallowing of air). Pathology showed marked irritation of the lung accompanied by acute interstitial pneumonia. This was confirmed by histological examination (Hine et al., 1956).

Observation in humans

The lowest published toxic concentration (TCLo) observed when administered onto the human skin was 0.25 parts per hundred (pph). Skin and appendages showed an allergic dermatitis reaction (Gallera Chemica).

Corrosion / Irritation

Skin irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to skin' (R38) (Safe Work Australia). The data available support this classification.

The chemical is an irritant to skin (IARC, 1989).

Eye irritation

The chemical is an eye irritant.

Standard Draize tests in rabbits showed mild to severe irritation when administered into the eye (Galleria Chemica). Draize scores are not available, but as the chemical is reported to be a severe eye irritant, classification is warranted.

Respiratory irritation

The chemical is a respiratory irritant upon single or repeated inhalation exposure (HSDB). The data available support the current hazard classification.

Observation in humans

Short term acute exposure to the chemical can cause irritation of the eyes, nose, respiratory tract and skin. Long term (chronic) exposure can cause defatting and drying of the skin, dermatitis, blisters, oedema, rash and eczema (HSDB).

Sensitisation

Skin sensitisation

The chemical is classified as hazardous with the risk phrase 'May cause sensitisation by skin contact' (R43) (Safe Work Australia). The data available support this classification.

PGE was found to be a strong sensitiser in guinea pigs with 23/24 animals sensitised. No control animals had reactions to PGE at challenge ($P < 0.001$). Lowering the concentration for the challenge did not reduce the number of reacting animals. All 24 animals induced with PGE reacted when challenged with the lower concentration ($P< 0.001$) (Ponten et al. 2009).

In a subchronic study, the substance (5% in ethanol) was rubbed onto the skin of 10 guinea pigs daily for 34 days. A challenge was then carried out with a 1% solution of PGE in ethanol. PGE had a sensitising effect on guinea pig skin (HSDB).

In another study, the sensitisation potential of PGE was investigated in albino guinea pigs (Hartley strain) by means of the Buehler Patch Method. Twenty animals were used for the treatment group and 10 for the control. For induction, the guinea pigs were treated with a 5% PGE solution in 80% ethanol, three times at weekly intervals. After an interval of two weeks, a challenge was carried out with a 0.5% solution of PGE in acetone. Evaluations were made at 24 and 48 hours after the application. The results showed that PGE had a sensitising effect in guinea pigs (HSDB).

Cross sensitivity between different epoxy resins, including PGE, has been documented elsewhere (Geier et al., 2004).

Observation in humans

In humans, some cases of sensitisation from topical contact with the chemical have been described.

Two case studies identifying positive allergic reactions to phenylglycidyl ether patch tests were documented, indicating that the patients were sensitised to this compound (Sasseville et al., 2000).

Twenty workers in an Italian aircraft factory suffered from an outbreak of contact dermatitis. Symptoms ranged from slight erythema to strong oedematous-vesicular lesions on the upper extremities and face but rarely on the genitalia and thighs. Patch testing was positive to the epoxy-resin material in 13/20 workers. Thin-layer chromatography identified PGE as one of the agents responsible for this outbreak of contact dermatitis (ACGIH, 2005).

In another case report, five out of 40 workers with dermatitis and occupationally exposed to epoxy resins, but not to phenoxymethyl oxirane, showed positive skin sensitivity reactions to phenoxymethyl oxirane. With long-term exposure, cross sensitisation with other glycidyl ethers can also occur. Among 58 dermatitis patients who had been exposed occupationally to PGE, nine primarily responded to PGE (HSDB).

Based on the above information the chemical is considered a skin sensitiser in humans.

Repeat Dose Toxicity

Oral

No data are available.

Dermal

No data are available.

Inhalation

In a sub chronic study, when rats inhaled aerosols of PGE at a concentration of 29 ppm for 4 hours/day, 5 days/week for two weeks, the animals exhibited weight loss, atrophic changes in the liver, kidneys, spleen, thymus, and testes, depletion of hepatic glycogen, and chronic catarrhal tracheitis. Local effects included respiratory irritation. The estimated NOAEC for rats exposed to vapours of PGE is 29 ppm (Terrill and Lee, 1977).

In a 90 day study, Sprague Dawley rats (32 per sex per dose level) were exposed to 1.3, 5, 12 ppm PGE by vapour inhalation for six hours each day, five days per week (for a total of 63 exposures). All rats appeared to be unaffected by treatment during and after exposure, except for significant hair loss, which occurred in both sexes at 5, and 12 ppm after about 45 days of exposure. At the end of the 90-day exposure period, 10% of the males and 25% of the females at the two highest dose levels exhibited areas of alopecia. In histopathological examinations, all tissues and organs from all test animals showed no significant changes when compared with controls at any sacrifice interval, except for the skin/hair of those rats at 5 and 12 ppm groups which exhibited alopecia. In the bald areas, some hair follicles were damaged. Also, these areas showed very slight acanthosis, parakeratosis, and inflammatory cellular infiltration of the dermis and hair follicles, and the hair shaft was disintegrated due to cellular infiltration and necrosis of the hair follicles. The estimated NOAEC for rats exposed to vapours of PGE is 12 ppm. However, local effects (respiratory irritation and alopecia) were observed at 1.3 and 5 ppm (Terrill and Lee, 1977).

The skin and hair effects, including in humans, were considered under hazard classification for skin irritation. As there were no other irreversible or serious effects reported in the repeat dose inhalation studies, the chemical is not classified for repeat dose toxicity.

Observation in humans

Long-term (chronic) exposure in humans has caused defatting and drying of the skin, dermatitis, blisters, oedema, rash and eczema (HSDB).

Genotoxicity

The chemical is classified as hazardous in the HSIS with the risk phrase 'Possible risk of irreversible effects' (R68) (Safe Work Australia). The data available support this classification.

In mammalian cells, the chemical did not induce chromosomal aberrations or gene mutations in cultured Chinese hamster ovary cells, but did induce transformation of hamster embryo cells in culture. No unscheduled DNA synthesis was induced in cultured rat hepatocytes (HSDB).

Escherichia coli strain WP2uvrA was used to investigate the mutagenic activity of phenoxymethyloxirane (20−10,000 µmol). No metabolising system was added. The chemical was mutagenic in this study. The chemical showed a dose-dependent mutagenic effect in a fluctuation test with *Klebsiella pneumoniae* at four concentrations (0.1−1 µmol/L) (HSDB).

The chemical was tested for genetic activity in bacterial and mammalian cells. It was active in the *Salmonella* microsome mutagenicity test. Concentration-dependent mutagenicity was demonstrated in *Salmonella typhimurium* strains TA 1535 and TA 100 with and without rat S9, but not in strains TA 98, TA 1537 or TA 1538. Phenoxymethyloxirane is apparently a direct-acting mutagen causing base substitutions. The chemical did not induce 6-thioguanine resistant mutants of Chinese hamster ovary (CHO) cells, with or without rat S9, and with or without serum in the medium. Dose-dependent enhancement of SA7 virus transformation of primary hamster embryo cells was observed at concentrations of 1.6 µg/mL and higher. This compound was able to chemically transform secondary hamster embryo cells at concentrations of 6.2 µg/mL and higher. At a dose of 2500 mg/kg, phenoxymethyloxirane was active in the host-mediated assay using C57B1/6 and *Salmonella typhimurium* strain TA 1535. This activity represented a positive response in two of five animals tested (HSDB).

A dominant lethal assay in rats inhaling PGE at concentrations of 0, 2, 6 and 11 ppm for 19 days (6 hours/day) was also negative (HSDB). Male rats were treated by inhalation of 0, 1, 5, or 12 ppm of the chemical for 6 hours/day on 19 consecutive days. No increases in the incidence of chromosomal gaps, breaks, or rearrangements were observed (ACGIH, 2005). The chemical was also negative in the mouse micronucleus assay following an oral dose of 1 g/kg (HSDB).

The chemical has been reported to alkylate nucleic acid bases in vitro; however it did not bind to DNA in *Escherichia coli* with or without metabolic activation (HSDB).

Alkylation activity was compared with mutagenicity of the chemicals to *Escherichia coli* WP2 UVRA without metabolic activation. All epoxide-containing compounds including glycidyl ethers elicited alkylation activity and mutagenic potency. There was no correlation between rate of alkylation and mutagenic potency (HSDB).

No effects on DNA synthesis in mouse testes were observed after oral administration of 0.5 g/kg PGE. There are no in vitro studies conducted in germ cells showing positive results.

Carcinogenicity

The chemical is classified with the risk phrase 'May cause cancer' (R45 Category 2 carcinogen) in HSIS (SWA). There are no long term carcinogenicity studies available using the oral or dermal route of exposure to eliminate those routes of exposure for carcinogenicity. The data available support this clssification.

Classified by the International Agency for Research on Cancer (IARC) as Group 2B (possibly carcinogenic to humans) based on sufficient evidence of carcinogenicity in experimental animals (IARC 1989).

Classified by American Conference of Governmental Industrial Hygienists (ACGIH) as A3 carcinogen (confirmed animal carcinogen with unknown relevance to humans).

In a chronic inhalation carcinogenicty bioassay, 100 rats were exposed to the chemical for 6 hours/day, 5 days/week for 24 months at 0, 1, or 12 ppm. After 621 exposure days, malignant nasal tumors were found in 9/85 (11%) of the males and in 4/89 (4.4%) of the females exposed at 12 ppm (average latent period was 688 days). No nasal tumors were found in the rats exposed at 1 ppm (even up to 24 months). A nasal

tumor was found in 1 of 89 male controls, while none were found in the female controls. Nasal tumors were primarily epidermoid carcinomas sharply limited to the anterior nasal cavity. Tumors were derived from respiratory epithelium and nasal glands (both of which revealed squamous metaplasia or dysplasia). Squamous metaplasia was seen in 72% of the 12 ppm exposure group, 4.7% of the 1 ppm group, and 3.4% for controls; rhinitis was observed in 78% at 12 ppm, 22% at 1 ppm, and 19% for controls.

There is sufficient evidence in experimental animals for the carcinogenicity of the chemical.

Reproductive and developmental toxicity

Based on the data available, the chemical is not considered a reproductive or developmental toxin.

In a two-generation rat reproduction study of PGE, four groups of eight male rats (F0) were exposed 6 hours/day for 19 consecutive days at 0, 1, 5, or 12 ppm. For six consecutive weeks, three female rats (F0) were placed in the cage with each treated male. The F1 generation was paired within treatment groups for mating. Body weight gain was unaffected. There was no increase in parental mortality. At the highest concentration, reduced fertility in the males was observed. Histopathological examination revealed focal degeneration of the seminiferous tubules. No other adverse effects on reproduction were found (ACGIH, 2005).

In a teratogenicity study, 25 pregnant female rats per group were exposed 6 hours/day at 0, 1, 5, or 12 ppm of PGE on days 4 to 15 of gestation, then sacrificed on day 20. No signs of maternal intoxication were observed in these animals. The numbers of implantations, embryonic deaths, and live foetuses, as well as mean foetal body weight and length, were similar in all groups. Foetuses were normal on gross and internal examination (ACGIH, 2005).

Risk Characterisation

Critical Health Effects

The main critical effects to human health are carcinogenicity following long-term occupational exposure and sensitisation from dermal contact. The chemical also possesses other hazardous properties such as skin, eye and respiratory tract irritation.

Public Risk Characterisation

The general population may be exposed to phenyl glycidyl ether if it is used in domestic products such as paints and varnishes. The exposure may be limited to dermal and inhalation routes. Characterised hazards have the potential to pose an unreasonable risk under the uses identified.

The EU and New Zealand have banned the use of this chemical in cosmetics. There are no restrictions in Australia to prevent this chemical being used in cosmetics or domestic products. However, no cosmetic use for this chemical has been identified in Australia. Considering the possibility of using this chemical in domestic products, risk management is required through scheduling restrictions.

Occupational Risk Characterisation

Occupational exposure to phenyl glycidyl ether may occur through dermal, eye or inhalation contact with the chemical at workplaces where phenyl glycidyl ether is produced or used.

The existing hazard classification of the chemical needs updating to include the risk phrase for eye irritation. Given the critical health effects, the risk to workers from this chemical is considered high unless adequate control measures to minimise occupational exposure to the chemical are implemented.

NICNAS Recommendation

Sufficient information is available to recommend the chemical to be adequately risk managed for public and worker safety. The chemical is considered to be fully assessed at the Tier II level with recommendations for scheduling and an update to the current hazard classification.

Regulatory Control

Public Health

It is recommended that the use of this chemical in domestic products such as paints and varnishes be

restricted through scheduling. Important matters for consideration for scheduling are skin sensitisation, carcinogenicity and genotoxicity of the chemical. The chemical is also an irritant to the eyes, skin and respiratory system and is harmful if inhaled. There were adverse skin effects including sensitisation reported in humans from long-term exposure to the chemical.

Occupational Health and Safety

The chemical is recommended for classification and labelling under the current Approved Criteria and adopted GHS as below. This does not consider classification of physical hazards and environmetal hazards.

	Approved Criteria (HSIS)[a]	***GHS Classification***
Acute Toxicity	Harmful by inhalation (Xn; R20)*	Harmful if inhaled - Cat. 4 (H332)
Irritation / Corrosivity	Irritating to eyes (Xi; R36) Irritating to skin (R38)* Irritating to respiratory system (Xi; R37)*	Causes serious eye irritation - Cat. 2A (H319) Causes skin irritation - Cat. 2 (H315) May cause respiratory irritation - Specific target organ tox, single exp Cat. 3 (H335)
Sensitisation	May cause sensitisation by skin contact (R43)*	May cause an allergic skin reaction -Cat. 1 (H317)
Genotoxicity	Muta. Cat 3 - Possible risk of irreversible effects (Xn; R68)*	Suspected of causing genetic defects - Cat. 2 (H341)
Carcinogenicity	Carc. Cat 2 - May cause cancer (T; R45)*	May cause cancer - Cat. 1B (H350)

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

* Existing Hazard Classification. No change recommended to this classification.

Advice for consumers

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

Advice for industry

Control measures

Control measures to minimise the risk from dermal/ocular/inhalation exposure to the chemical should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include: substitution, isolation and engineering controls. Measures required to eliminate or minimise risk arising from storage, handling and use of a hazardous chemical are dependent on the physical form and the manner in which the chemical is used. Examples of control measures which may minimise the risk include, but are not limited to:

- use of closed systems or isolation of operations;
- use of 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;
- minimisation of manual processes and work tasks through automation of processes;
- work procedures that minimise splashes and spills;
- regular cleaning of equipment and work areas; and
- use of protective equipment that is designed, constructed, and operated to ensure that, the worker does not come into contact with the chemical.

Guidance on managing risks from hazardous chemicals are provided in the *Managing Risks of Hazardous Chemicals in the Workplace—Code of Practice* available on the Safe Work Australia website.

Personal protective equipment should not be relied upon on its own to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selection of personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to assist with meeting obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((m)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of hazardous chemical are prepared; and
- management of risks arising from storage, handling and use of a hazardous chemical.

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

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

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

References

ACGIH (American Conference of Governmental Industrial Hygienists). Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. CD-ROM Cincinnati, OH 45240-1634 2005, p. 2.

Approved Criteria for Classifying Hazardous Substances [NOHSC: 1008(2004)] Third edition. Accessed at http://www.nohsc.gov.au/pdf/Standards/approved_criteriaNOHSC1008_2004.pdf.

Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982, p. 2215.

Czajkowska T, Stetkiewicz J; Med PR 1972 23 (4): 363.

eChemPortal, Oxirane, (phenoxymethyl)- (122-60-1) . Accessed September 2012 at http://www.echemportal.org/echemportal/substancesearch/substancesearchresult.action?queryTicket=SUBQo99&view=grouped&pageID=9.

Galleria Chemica. Accessed June 2012 at http://jr.chemwatch.net/galleria/.

Geier J, Lessmann H, Hillen U, Jappe U, Dickel H, Koch P, Frosch PJ, Schnuch A, Uter W. (2004) An attempt to improve diagnostics of contact allergy due to epoxy resin systems. First results of the multicentre study EPOX 2002. Contact Dermatitis. Nov-Dec; 51(5-6): 263-72.

Globally Harmonised System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third edition. Accessed at http://www.unece.org/trans/danger/publi/ghs/ghs_rev03/03files_e.html.

Hazardous Substances Data Bank (HSDB). National Library of Medicine. Accessed October 2012 at http://toxnet.nlm.nih.gov.

Hine, C.H., Kodama, J.K., Wellington, J.S., Dunlap, M.K. and Anderson, H.H. (1956) The toxicology of glycidol and some glycidyl ethers. Archived of Industrial Health, 14: 250-264.

IARC (1989). Volume 47- Some Organic Solvents, Resin Monomers and Related Compounds, Pigments and Occupational Exposures in Paint Manufacture and Painting. Accessed at http://monographs.iarc.fr/ENG/Monographs/vol47/mono47.pdf.

James S.P., Pheasant A.E., Solheim E., (1978) Xenobiotica. April; 8(4): 219-28.

Ponten, A., Zimerson, E. and Bruze, M. (2009) Sensitizing capacity and cross-reactivity of phenylglycidyl ether studied in the guinea-pig maximization test. Contact Dermatitis, 60: 79-84.

Safe Work Australia (SWA). Hazardous Substances Information System (HSIS). Accessed at http://hsis.safeworkaustralia.gov.au/HazardousSubstance in October 2012.

Sasseville, D., Moreau, L., Brassard, J. and Leclerc, G. (2000) Allergic contact dermatitis to epoxy resin in microscopy immersion oil: cases from Canada. American Journal of Contact Dermatitis, 11(2): 99-103.

Substances in Preparations in Nordic Countries (SPIN). Oxirane, (phenoxymethyl)- (122-60-1). Accessed September 2012 at http://188.183.47.5/fmi/xsl/spin/SPIN/spinNACE.xsl?-db=SPINstof&-skip=0&-max=1&casnr.op=eq&casnr=122-60-1&SPINnavn%3a%3anavn.op=eq&SPINnavn%3a%3anavn=&ec_nr.op=eq&ec_nr=&-lay=SpinIndustUse&-find.

Terrill, J.B. and Lee, K.P. (1977) The inhalation toxicity of phenylglycidyl ether. I. 90-day inhalation study. Toxicology and Applied Pharmacology, 42: 263-269.