1,2-Propanediol, 3-chloro-: Human health tier II assessment

13 February 2015

CAS Number: 96-24-2

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.

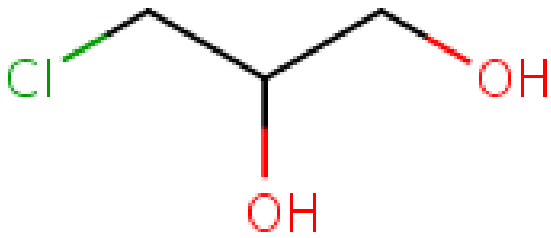
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	alpha-chlorohydrin 3-chloro-1,2-dihydroxypropane 3-chloropropane-1,2-diol 2,3-dihydroxypropyl chloride 3-MCPD
Structural Formula	
Molecular Formula	C3H7ClO2
Molecular Weight (g/mol)	110.54
Appearance and Odour (where available)	Clear pale yellow liquid with a slight odour
SMILES	C(O)(CO)CCl

Import, Manufacture and Use

Australian

No Australian use, import, or manufacturing information has been identified.

The chemical has been identified by Food Standards Australia New Zealand (FSANZ) as a chloropropanol formed in foods. Chloropropanols are considered to be contaminants present in foods, formed during the manufacturing process (FSANZ, 2003).

International

The following international uses have been identified through the European Union (EU) Registration, Evaluation and Authorisation of Chemicals (REACH) dossiers; Galleria Chemica; the Substances and Preparations in the Nordic countries (SPIN) database; the US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR); the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and International Agency for Research on Cancer (IARC) monographs.

The chemical has reported domestic uses (SPIN):

- in adhesives, binding agents and fillers; and
- in paints, lacquers and varnishes.

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

The chemical has reported commercial uses in construction materials.

The chemical has reported site-limited uses including:

- in manufacturing pulp paper and paper products;
- in manufacturing dye intermediates;
- as an intermediate in producing other chemicals; and
- as an ingredient in dynamite, to lower the freezing point.

The chemical has reported non-industrial uses as a:

- rodent chemosterilant (pesticide); and
- raw material or intermediate to synthesise pharmaceutical products.

Restrictions

Australian

The chemical alpha-chlorohydrin (3-MCPD, CAS No. 96-24-2) is listed in Schedule 6 of the *Poisons Standard*—the *Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP).

Schedule 6 chemicals are described as 'Substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label'. Schedule 6 chemicals are labelled with 'Poison' (SUSMP, 2014).

The chemical is listed on the Australia New Zealand Food Standards Code, Contaminants and Natural Toxicants—Maximum levels of non-metal contaminants in food (maximum level of 0.2 mg/kg in soy and oyster sauces, calculated based on a 40 % dry matter content).

International

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

- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient ‘Hotlist’); and
- EU Regulation (EC) No 1881/2006 Maximum levels for certain contaminants in foodstuffs (tolerable daily intake (TDI) is 2 µg/kg body weight).

Existing Work Health and Safety Controls

Hazard Classification

The chemical is not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

The following exposure standards are identified for the chemical (Galleria Chemica):

- an exposure limit of 0.023 mg/m^3 (0.005 ppm) time weighted average (TWA) in Germany; and
- 0.18 mg/m^3 (0.04 ppm) short-term exposure limit (STEL) in Switzerland.

Health Hazard Information

Toxicokinetics

Following intravenous injection, the chemical is widely distributed in body fluids, as it crosses the blood-brain barrier and blood-testes barrier. Metabolism of the chemical in mammals occurs primarily via conjugation with glutathione, followed by oxidation to form β-chlorolactate and oxalic acid. In bacterial systems, the chemical can be metabolised to glycidol. The major pathways of excretion include exhalation as carbon dioxide (approximately 30 %), excretion in the urine unchanged (approximately 8.5 %) or as the metabolite, β-chlorolactate (approximately 23 %) (IARC, 2012).

Acute Toxicity

Oral

Based on the available data, the chemical is considered to have high acute oral toxicity, warranting hazard classification (see **Recommendation** section).

The median lethal dose (LD50) was reported to be 150 mg/kg bw in rats (WHO, 2002).

Dermal

No data are available.

Inhalation

Based on the available data, the chemical is considered to have high acute inhalation toxicity, warranting hazard classification (see **Recommendation** section).

The median lethal concentration (LC50) was reported to be between 62 and 250 ppm (0.28 and 1.14 mg/L) in Sherman rats exposed to the chemical vapour (whole body) for four hours (REACH). Another study reported an LC50 value of >90 ppm (>0.41 mg/L) in male Long–Evans rats and male Webster mice, when exposed to the chemical vapour (whole body) for eight hours and four hours, respectively (Hine et al., 1956).

Observation in humans

A 34-year-old male died 10 days after cleaning a tank in which there were traces of the chemical; the cause of death was liver failure. A 27-year-old male exposed to the chemical in the same incident, but to a lesser extent, suffered from mild liver disorder (HSDB). No further details were available.

Corrosion / Irritation

Skin Irritation

Only limited data are available indicating the chemical to be a skin irritant. The available information is not sufficient to warrant hazard classification.

In an in vitro skin irritation test (in accordance with EU Method B.46), normal human skin cells were exposed to 30 µL of the chemical for 60 minutes. Skin irritation was reported 42 hours following exposure, measured by formazan (colour dye) production (20.2 % out of 100 %) (REACH).

Eye Irritation

Based on the available data, the chemical is considered to cause severe eye irritation, warranting hazard classification (see **Recommendation** section).

Although the test method used in the available study has not been validated by the Organisation for Economic Co-operation and Development (OECD), it was agreed at the 64th meeting of Competent Authorities (Copenhagen, November 2002; NOTIF/19/2002) 'that available evidence is sufficient to conclude that the methods are able to detect severe eye irritants' (ECB, 2006).

In an in vitro eye irritation study, using the hen's egg test on the chorioallantoic membrane (HET-CAM) assay (according to the protocol recommended by the National Institute of Environmental Health Sciences (NIEHS) Interagency Coordinating Committee on the Validation of Alternative Methods (ICCVAM)), six Lohmann Leghorn chicken egg membranes were exposed to 300 µL of the chemical for 300 seconds. The chemical was reported to be a severe eye irritant (score = 19.59/19.9) (REACH).

Sensitisation

Skin Sensitisation

Based on the available data, the chemical is not considered to cause skin sensitisation.

In a maximisation test (according to OECD Test Guideline (TG) 406), Himalayan white spotted guinea pigs (n = 10/sex) were injected intradermally with 0.1 mL of a 5 % concentration of the chemical to shaved skin, followed by topical (occlusive) application of a 2 x 2 cm piece of filter paper saturated with 100 % concentration of the chemical for 48 hours. Two weeks after induction, a 2 x 2 cm piece of filter paper saturated with 100 % concentration of the chemical was used for challenge by topical (occlusive) application for 24 hours. No differences were observed between the control and treated guinea pigs (REACH).

Repeated Dose Toxicity

Oral

Based on the available data, the chemical is considered to cause harmful effects from repeated oral exposure, warranting hazard classification (see **Recommendation** section).

In a 90-day oral gavage study, Wistar rats (n = 10/sex/dose), except for the female high dose group where n = 20) were administered the chemical at doses of 0, 1.84, 7.37 or 29.50 mg/kg bw/day. The no observed adverse effect level (NOAEL) was determined to be 7.37 mg/kg bw/day, based on significant histopathological signs of kidney toxicity and significantly increased liver weight in animals that were administered 29.5 mg/kg bw/day. Dose-dependent increases in kidney weight were also reported. One female rat died in the 7.37 mg/kg bw/day group (in the last week of the study), and 7/20 died in the 29.5 mg/kg bw/day group. This was attributed to acute renal failure (Barocelli et al., 2011).

In a 13-week study (according to OECD TG 408), B6C3F1 mice (n = 10/sex/dose) were exposed to the chemical via drinking water at concentrations of 0, 5, 25, 100, 200 or 400 ppm. Based on the average daily water intake, this was equivalent to doses of 0, 0.79, 3.94, 15.02, 30.23 and 61.34 mg/kg bw/day in female mice and 0, 0.94, 4.59, 18.05, 36.97 and 76.79 mg/kg bw/day in male mice. The NOAEL was reported to be 100 ppm (15 and 18 mg/kg bw/day for females and males, respectively), based on statistically significant increases in relative kidney weights at 200 ppm (30 and 37 mg/kg bw/day for females and males, respectively). No significant histopathological changes were reported in association with the increased kidney weights, but this observation was considered relevant toxicologically as it could be related to inhibition of glycolysis by metabolites of the chemical (REACH).

In rats exposed to the chemical at 30 mg/kg bw/day for four weeks or 9 mg/kg bw/day for 13 weeks, the kidney was reported as the target organ of toxicity based on increased relative weights. In rats and mice, the chemical at doses ≥25 mg/kg bw/day was associated with toxicity in the central nervous system, particularly brain stem lesions (WHO, 2002). No further details were available for these studies.

Dermal

No data are available.

Inhalation

Only limited data are available.

In a two-week repeated dose inhalation toxicity study (similar to OECD TG 412), Sprague Dawley (SD) rats (n = 10 males and five females/dose) were exposed (nose/head only) to the chemical vapour at doses of 0, 1.3, 4.8 or 18.1 ppm (0, 0.006, 0.022 or 0.082 mg/L) for six hours/day, five days/week. The no observed effect concentration (NOEC) was determined to be 4.8 ppm (0.022 mg/L) based on reduced food intake during the first week of treatment observed at 18.1 ppm (0.082 mg/L) (REACH).

Genotoxicity

The chemical is not considered to be genotoxic.

Several in vitro assays conducted with the chemical gave mixed results for mutagenicity and clastogenicity (IARC, 2012):

- positive results in bacterial reverse mutation assays in *Salmonella typhimurium* strains TA100 and TA1535, with or without metabolic activation;
- positive results in forward mutation assays in *S. typhimurium* strains TA98 and TM677 and *Schizosaccharomyces pombe*, without metabolic activation; but negative results with metabolic activation in *S. pombe*;
- negative results in a bacterial reverse mutation assay in *S. typhimurium* strain TA97 with metabolic activation, but not tested without metabolic activation;
- negative results in bacterial reverse mutation assays in *Escherichia coli* strains WP2, TM930 and TM1080, with or without metabolic activation;
- positive results in a gene transformation assay in mouse fibroblasts;
- positive results in a comet assay for DNA strand breaks in Chinese hamster ovary (CHO) cells, without metabolic activation (not tested with metabolic activation); and
- negative results in a gene mutation assay in HeLa cells, with or without metabolic activation.

All in vivo genotoxicity assays with the chemical gave negative results (IARC, 2012):

- a bone marrow micronucleus assay in male Han Wistar rats administered the chemical at a dose of 60 mg/kg bw/day for two days by oral gavage;
- an unscheduled DNA synthesis assay in hepatocytes of male Han Wistar rats administered the chemical once at 100 mg/kg bw by oral gavage;
- two comet assays in male SD rats (leukocytes, liver, kidney, testes and bone marrow) and male Fischer 344 (F344) rats (leukocytes and testes), where the animals were exposed to the chemical at a dose of 60 mg/kg bw/day for two days by oral gavage;
- a somatic mutation (wing-spot test) assay in *Drosophila melanogaster* exposed to the chemical at 1.1 mg/mL; and
- dominant lethal mutation assays in ICR/Ha Swiss male mice (exposed to the chemical at 125 mg/kg bw once by intraperitoneal (i.p.) injection or 20 mg/kg bw/day for five days by oral gavage); male rats (strain not specified; exposed to the chemical at 10 mg/kg bw/day for five days by oral gavage); and male Wistar rats (exposed to the chemical at 20 mg/kg bw/day for five days by oral gavage).

Carcinogenicity

Based on the available data, the chemical is considered to be carcinogenic, warranting hazard classification (see **Recommendation** section).

The IARC has classified the chemical as 'Possibly carcinogenic to humans' (Group 2B), based on inadequate evidence for carcinogenicity in humans, but sufficient evidence for carcinogenicity in animals (IARC, 2012).

In a two-year carcinogenicity study in F344 rats exposed to the chemical at 0, 20, 100 or 500 ppm (equiavlent to 0, 1.1, 5.2 or 28 and 0, 1.4, 7.0 or 35 mg/kg bw/day for males and females, respectively) in drinking water, there was increased incidence of kidney tumours in all treated rats, and increased incidence of Leydig cell (testes), mammary gland and preputial gland adenomas in treated male rats. Increased kidney weight and nephrotoxicity were observed at doses ≥20 ppm (1.1 or 1.4 mg/kg bw/day for males and females, respectively) (WHO, 1993; REACH).

In a two-year carcinogenicity study (according to OECD TG 451), SD rats (n = 50/sex/dose) were exposed to the chemical at doses of 0, 25, 100 or 400 ppm (equivalent to 0, 1.97, 8.27 or 29.50 and 0, 2.68, 10.34 or 37.03 mg/kg bw/day for males and females, respectively) in drinking water. At the highest dose, there was an increased incidence of renal tubule neoplasms in male and female rats compared with concurrent and historical controls. In male rats, an increased incidence of Leydig cell tumours was observed at 400 ppm (REACH).

Two other studies in rats and mice (strain, dose or duration of exposure not indicated) reported no changes in the incidence of tumours in treated animals when administered the chemical via oral gavage doses, in drinking water or via subcutaneous injection (IARC, 2012).

Reproductive and Developmental Toxicity

Based on the available data, the chemical is considered to cause reproductive toxicity, warranting hazard classification (see **Recommendation** section).

In a 90-day oral gavage study, Wistar rats (n = 10/sex/dose), except for the female high dose group where n = 20) were exposed to the chemical at doses of 0, 1.84, 7.37 or 29.50 mg/kg bw/day. In male rats exposed to the chemical at 1.84 mg/kg bw/day, decreased spermatid density (9/10 animals) and slight atrophy of spermatogenic (3/10 animals) and supporting cells (4/10 animals) were reported. In male rats exposed to the chemical at 7.37 mg/kg bw/day, a mild decrease in spermatids and atrophy of seminiferous tubule spermatogenic and supporting cells were reported in 6/10 animals. In male rats exposed to the chemical at 29.50 mg/kg bw/day, total degeneration of the seminiferous tubules and inflammatory infiltration in the epididymis were reported in 9/10 animals (Barocelli et al., 2011).

In a 13-week study (according to OECD TG 408), B6C3F1 mice (n=10/sex/dose) were exposed to the chemical via drinking water at concentrations of 0, 5, 25, 100, 200 or 400 ppm. Based on the average daily water intake, this was equivalent to doses of 0, 0.79, 3.94, 15.02, 30.23 and 61.34 mg/kg

bw/day in female mice and 0, 0.94, 4.59, 18.05, 36.97 and 76.79 mg/kg bw/day in male mice. At 400 ppm, decreased sperm motility in males and delayed total oestrus cycle in females were reported. There was also increased incidence and greater severity of germinal epithelium degeneration in testes of males exposed to ≥200 ppm (REACH).

In a 2-week repeated dose inhalation toxicity study (similar to OECD TG 412), SD rats (n=10 males and 5 females/dose) were exposed (nose/head only) to the chemical vapour at doses of 0, 1.3, 4.8 or 18.1 ppm (0, 0.006, 0.022 or 0.082 mg/L) for six hours/day, five days/week. Two days, six to nine days and 27-30 days after exposure, males were mated with untreated females (n=30). Reduced fertility (0/6 and 4/6 pregnancies from males exposed at 0.082 mg/L and 0.022 mg/L, respectively, compared with 6/6 pregnancies from males in the control or 0.006 mg/L groups), without associated histopathological changes in the testes or epididymides, was reported as a consequence of chemical exposure; but this did not occur in the groups mated from day nine onwards (REACH).

In a non-guideline developmental toxicity study (focussed on testicular organogenesis), pregnant female SD rats (n = 5-6/dose) were exposed to the chemical by oral gavage at doses of 0, 5, 10 or 25 mg/kg bw/day from gestation days (GD) 11 to 19. Body weight gain was significantly lower in dams exposed to the chemical at ≥10 mg/kg bw/day. There were no effects of chemical exposure of the dams on the outcomes for male foetuses (GD 19) or three to five day old male neonates in the parameters of testicular morphology, germ cell proliferation and apoptosis, and testosterone levels in the testes or testicular gene expression (REACH).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- the systemic long-term effects of carcinogenicity and reproductive toxicity; and
- systemic acute effects from oral and inhalation exposure.

The chemical can also cause harmful effects following repeated oral exposure and eye irritation.

Public Risk Characterisation

Given the uses identified for the chemical, it is unlikely that the public will be exposed. Although the public could come into contact with articles/coated surfaces containing the chemical, it is expected that the chemical will be bound within the article/coated surface and hence will not be bioavailable. Therefore, the chemical is not considered to pose an unreasonable risk to public health. However, there is a Schedule 6 entry for the chemical in the SUSMP (2014) for its use as a pesticide (see **Restrictions** section).

Occupational Risk Characterisation

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

NICNAS Recommendation

Assessment of the chemical is considered to be sufficient, provided that the recommended 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]
Acute Toxicity	Toxic if swallowed (T; R25) Toxic by inhalation (T; R23)	Toxic if swallowed - Cat. 3 (H301) Fatal if inhaled - Cat. 2 (H330)

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Irritation / Corrosivity	Risk of serious eye damage (Xi; R41)	Causes serious eye damage - Cat. 1 (H318)
Repeat Dose Toxicity	Harmful: Danger of serious damage to health by prolonged exposure if swallowed (Xn; R48/22)	May cause damage to organs (kidney) through prolonged or repeated exposure - Cat. 2 (H373)
Carcinogenicity	Carc. Cat 2 - May cause cancer (T; R45)	May cause cancer - Cat. 1B (H350)
Reproductive and Developmental Toxicity	Repro. Cat 2 - May impair fertility (T; R60)	May damage fertility - Cat. 1B (H360F)

[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 oral, 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 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;
- 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

Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)]. Third edition [NOHSC:1008 (2004)]. Accessed at http://www.safeworkaustralia.gov.au/sites/swa/about/publications/Documents/258/ApprovedCriteria_Classifying_Hazardous_Substances_NOHSC1008-2004_PDF.pdf

Barocelli E, Corradi A, Mutti A and Petronini PG 2011. Scientific Report submitted to European Food Say Authority (EFSA) on Comparison between 3-MCPD and its palmitic esters in a 90-day toxicological study. Accessed at http://www.efsa.europa.eu/en/supporting/doc/187e.pdf

European Chemicals Bureau (ECB) 2006. MANUAL OF DECISIONS FOR IMPLEMENTATION OF THE SIXTH AND SEVENTH AMENDMENTS TO DIRECTIVE 67/548/EEC ON DANGEROUS SUBSTANCES (DIRECTIVES 79/831/EEC AND 92/32/EEC). Accessed at http://tsar.jrc.ec.europa.eu/documents/Manual_of_decisions.pdf

Food Standards Australia and New Zealand (FSANZ) October 2003. Chloropropanols in food - An analysis of public health risk. Accessed at http://www.foodstandards.gov.au/publications/Documents/Technical Report Chloropropanol Report 11 Sep 03.doc

Galleria Chemica. Accessed January 2015 at http://jr.chemwatch.net/galleria/

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

Hine CH, Kodama JK, Wellington JS, Dunlap MK& Anderson HH 1956. The toxicology of glycidol and some glycidyl ethers. AMA Archives of Industrial Health 14(3) pp. 250-264.

International Agency for Research on Cancer (IARC) 2012. Some Chemicals Present in Industrial and Consumer Products, Food and Drinking-water, IARC Monographs Volume 101. Accessed at http://monographs.iarc.fr/ENG/Monographs/vol101/index.php.

Registration, Evaluation and Authorisation of Chemicals (REACH) Dossier. 96-24-2. Accessed January 2015 at http://echa.europa.eu/web/guest/information-on-chemicals/registered-substances

Safe Work Australia (SWA). Hazardous Substances Information System (HSIS). Accessed January 2015 at http://hsis.safeworkaustralia.gov.au/HazardousSubstance

Substances in Preparations in Nordic Countries (SPIN). Accessed January 2015 at http://188.183.47.4/dotnetnuke/Home/tabid/58/Default.aspx

Therapeutic Goods Administration–Department of Health and Ageing 2014. Standard for the Uniform Scheduling of Medicines and Poisons No. 5 (the SUSMP 5). Accessed at http://www.comlaw.gov.au/Details/F2014L01343

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

US National Library of Medicine's Hazardous Substances Data Bank (HSDB). 96-24-2. Accessed January 2015 at http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

World Health Organisation (WHO) 1993. Technical Report Series 837. Evaluation of certain food additives and contaminants. Forty-first report of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). Accessed at http://whqlibdoc.who.int/trs/WHO_TRS_837.pdf

World Health Organisation (WHO) 2002. Technical Report Series 909. Evaluation of certain food additives and contaminants. Forty-first report of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). Accessed at http://whqlibdoc.who.int/trs/WHO_TRS_909.pdf

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