Australian Government
Department of Health and Ageing
NICNAS

INVENTORY MULTI-TIERED ASSESSMENT AND PRIORITISATION (IMAP)

HUMAN HEALTH TIER II ASSESSMENT FOR

Propane, 2-nitro-

CAS Registry Number: 79-46-9

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

Propane, 2-nitro-

CAS No: 79-46-9

Chemical Identity

Synonyms	2-Nitropropane Dimethylnitromethane Isonitropropane Nitroisopropane Nipar S-20
Structural Formula	
Molecular Formula	C3H7NO2
Molecular Weight (g/mol)	89.0933
Appearance and Odour (where available)	Colourless liquid with a pleasant fruity odor.
SMILES	C(C)(C)N(=O)=O

Import, Manufacture and Use

Australian

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

International

The following international uses have been identified via Galleria Chemica, Substances and Preparations In the Nordic countries (SPIN), Registration Evaluation and Authorisation of Chemicals (REACH) Dossiers, Canadian Assessments (Challenge List Batch 8) and eChemPortal data sources including the Hazardous Substances Data Bank (HSDB), the Aggregated Computer Toxicology Resource (ACToR), European chemical Substances Information System (ESIS), the International Programme on Chemical Safety (INCHEM) and the Organisation for Economic Cooperation and Development (OECD).

The chemical has reported commercial use including:

- as an adhesive and binding agent in flexographic printing;
- as a solvent in paint and coating applications;
- reported use as coating/linings for beverage cans;
- in the manufacture of fats, waxes, gums, dyes and other organic compounds;
- as a rocket propellant/fuel additive; and
- as a component of explosives.

The chemical has reported site-limited use including:

- as a chemical intermediate in the manufacture of 2-nitro-2-methyl-1-propanol, 2-amino-2-methyl-1-propanol and 2-dimethylamino-2-methyl-1-propanol.

Restrictions

Australian

No known restrictions have been identified.

International

Cosmetics

EU Cosmetic Directive 76/768/EEC Annex II: List of Substances which must not form part of the Composition of Cosmetic Products.

New Zealand Cosmetic Products Group Standard - Schedule 4: Components Cosmetic Products Must Not Contain.

Food Packing

US FDA Indirect Food Additives: Adhesives and Components of Coatings - Substances for Use Only as Components of Adhesives.

US FDA List of "Indirect" Additives Used in Food Contact Substances: Substances that may come into contact with food as part of packaging or processing equipment, but are not intended to be added directly to food.

Existing Worker Health And Safety Controls

Hazard classification

The chemical is currently classified in Australia as hazardous with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

Carc. Cat. 2 ; R45 (May cause cancer)
Xn; R20/22 (Harmful by inhalation and if swallowed)

Exposure standards

Australian

Time Weighted Average (TWA): 10 ppm (36 mg/m³).

International

TWA: 5 ppm (18 mg/m³) [Denmark, Estonia, Germany, Ireland, South Africa]
TWA: 5 ppm (19 mg/m³) [Spain, United Kingdom]

TWA: 10 ppm (36 mg/m³) [Canada, Indonesia, Malaysia, Singapore, South Africa, Taiwan]
TWA: 10 ppm (35 mg/m³) [Greece,Norway, USA]

TWA: 25 ppm (90 mg/m³) [Phillipines]

Health Hazard Information

Toxicokinetics

The chemical is reported to have limited absorption through skin, following dermal exposure to female adult rhesus monkeys for 12 hours (REACH Dossier, 2011). The study reported a high loss of start material (98.2% of the total dose) with approximately 30% recovered from swabs used to wipe the skin after the 12-hour occluded exposure. Of the total dose, 0.7% was excreted within 72 hours, of which 96.6% was removed in the urine and 3.6% excreted through the faeces.

In a study using rats exposed either by inhalation of 20 or 154 ppm (6 hour whole body chamber

exposure), or a single intravenous injection of 13 to 148 mg/kg of the chemical, 40% of the dose was reported to be absorbed. The chemical was distributed to excretory organs (liver, kidney and lungs), with excretion occurring mainly via the lungs. Urine and faeces were reported as minor routes. The half life was estimated in rats to be less than 2 hours. A low bioaccumulation potential based on study results was reported.

Acute Toxicity

Oral

The chemical is currently classified in Australia with the risk phrase 'Harmful if swallowed (R22)' (Safe Work Australia 2012). The data available support this classification.

The chemical was reported to cause acute toxicity via the oral route (median lethal dose (LD50) in rats = 725 ± 160 mg/kg bw) (REACH Dossier, 2011). Reported signs of intoxication included progressive unsteadiness, weakness and incoordination ending in ataxia. Necropsy revealed lung haemorrhage and dark areas in the liver, spleen and kidneys.

Dermal

The chemical was reported to cause low acute toxicity via the dermal route in a non-guideline study (LD50 > 2000 mg/kg) (REACH Dossier, 2011). Abrasion tests were carried out on 10 rabbits exposed for 24 hours with 2000 mg/kg bw of the chemical. None of the rabbits exhibited signs of toxicity.

Inhalation

The chemical is currently classified in Australia with the risk phrase 'Harmful by inhalation (R20)' (Safe Work Australia, 2012). The data available support this classification.

The Government of Canada (2010) reported the lowest median lethal concentration (LC50) = 1460 mg/m³ (male rats). Rat LC50s have also been reported as 400 ± 38 ppm (males) and 720 ± 46 ppm (females) (REACH Dossier, 2011). Mouse LC50 = 560 ppm for both sexes (REACH Dossier, 2011).

Reported signs of toxicity included slight depression, hyperventilation and cyanosis (a bluish discolouration of the skin caused by a lack of oxygen in the blood). Necropsies were unremarkable.

Observation in humans

Acute toxicity was reported in humans exposed to the chemical via daily inhalation of 20 to 45 ppm (Government of Canada, 2010). Signs of toxicity included severe headaches, nausea, aversion to food, vomiting and diarrhoea.

Two separate work place related fatalities have been reported following exposure to a surface coating product containing the chemical, which was being applied in confined spaces. Four workers succumbed to fulminant hepatic failure within 6 to 10 days after exposure. In one of the occurrences, the man who died had serum concentrations of 13 mg/L of the chemical. A second man, who recovered but had persistently elevated serum aminotransferase activity, was reported to have 8.5 mg/L of the chemical detected in serum. It is noted that concurrent exposure to other solvents in the product may have contributed.

Corrosion / Irritation

Skin irritation

The chemical is reported as not irritating to the skin following exposure to the skin of rabbits for periods of 24 hours and 48 hours (REACH Dossier, 2011). No erythema or oedema were observed.

Eye irritation

The chemical is reported as non-irritating to eyes in a non-guideline animal study (REACH Dossier, 2011). The chemical was administered at 0.1 mL to the conjunctival sac of one eye to six New Zealand White rabbits, and observed at 24, 48 and 72 hours after exposure. The mean eye irritation score was 1.0 ± 1.7 at 24 hours and 0.3 ± 0.8 at 48 hours. Effects reported are insufficient to warrant a hazard classification.

Sensitisation

Skin sensitisation

It is reported that the chemical is not a skin sensitiser in guinea pigs following intradermal injection twice in 48 hours with 0.05 mL of a 5% test material in saline, followed by a further eight 0.1 ml injections over a one week period (REACH Dossier, 2011).

Repeat Dose Toxicity

Oral

The lowest observed adverse effect level (LOAEL) was reported to be 26 mg/kg bw/day (male F344 rats) (Government of Canada, 2010).

In a short term repeat dose oral toxicity study, five male F344 rats were exposed to the chemical by gavage, given 6 times over 2 weeks, at doses from 26 to 47 mg/kg bw/day (Government of Canada, 2010). The LOAEL of 26 mg/kg bw/day was based on a significant increase in hepatic lipid peroxidation. At 47 mg/kg bw/day, significant elevation of serum aspartate aminotransferase was reported, as well as a dose related increase in oxidative DNA damage and cell proliferation in the liver.

In another study, rats (Wistar) were exposed to the chemical by gavage at 0, 20, 200 or 400 mg/kg bw/day for 28 days (WHO, 1990). At 400 mg/kg bw/day, all male rats died within one day and all female rats within ten days. At 200 mg/kg bw/day, all male rats died within seven days while all females survived. No mortalities were reported for the 20 mg/kg bw/day group. There was no change in blood biochemistry. While multifocal centrilobular hepatocellular hypertrophy was reported, these were reported to be adaptive changes. Reduced body weight gain, higher alanine aminotransferase, aspartate aminotransferase and total bilirubin, and lower serum total protein and albumin levels were also reported in females at the higher doses. A LOAEL of 200 mg/kg bw/day was reported for this study (Government of Canada, 2010).

The minor and likely reversible effects reported in the liver at low doses, do not meet the classification criteria of being clear functional disturbances or morphological changes that are toxicologically significant.

Dermal

No data available.

Inhalation

In a repeat dose inhalation toxicity study, a lowest observed adverse effect concentration (LOAEC) value of 0.365 mg/L in Sprague Dawley (SD) rats was reported following whole body inhalation exposure to the chemical for 7 hours per day, for 4 days (Government of Canada, 2010). Enhanced total glutathione and increased activities of glutathione S-transferase and uridine 5'-diphosphate-glucuronosyltransferase in the liver were observed.

In another subchronic inhalation study, SD rats (125 of each sex) were exposed to 130 mg/m³ of the chemical (reported as a duration-adjusted dose) for 7 hours/day, 5 days/week for up to 6 months (Government of Canada, 2010). Groups of 10 rats per sex were sacrificed at 10 days, 1 month, 3 months

and 6 months. Vacuolisation was observed after 10 days exposure, necrosis of hepatocytes after 1 month exposure, elevation in liver weights from 3 to 6 months and an increase in alanine aminotransferase was reported after 6 months.

Observation in humans

The Government of Canada (2010) highlighted findings from a health examination of employees exposed to the chemical in a chemical plant. A small group of 18 workers from a large plant was recognised as potentially exposed to the chemical. They had served the company for 16 to 35 years. Their age group was between 45 and 64 years. Personal time weighted average exposure levels were below 25 ppm (91 mg/m^3). The examination included medical history questionnaires, clinical tests such as haematology and urinalysis, with body systems evaluated including lungs, liver, kidney, blood, skin and cardiovascular. No indications of cancer or liver dysfunction could be attributed to the workplace. There was no significant difference between the group of 18 employees believed to have been in potential contact with the chemical, compared with employees who worked at the plant but were not considered to be potentially exposed to the chemical.

Genotoxicity

The chemical was positive in several in vitro and in vivo genotoxicity studies (IARC, 1987, REACH Dossier, 2011).

In vitro

Bacterial reverse mutation assay (Ames test) studies in *S. typhimurium* were positive with or without S9 metabolic activation (REACH Dossier, 2011, IARC, 1987). Unscheduled DNA synthesis was reported in mouse and rat primary hepatocytes without exogenous metabolic activation. Gene mutation assays in Chinese hamster lung and rat hepatoma cells were also positive without exogenous metabolic activation. A negative result was found for sister chromatid exchange in Chinese hamster ovary cells, with or without metabolic activation.

Unscheduled DNA synthesis was induced by the chemical in hepatocytes from three of six humans (IARC, 1987). The chemical also induced sister chromatid exchange and chromosomal aberration in the presence of an exogenous metabolic system in human peripheral lymphocyte cells.

In vivo

Gene mutation studies in male SD rats, administered via oral gavage with a single dose of 0, 50, 100 or 300 mg/kg (bone marrow micronucleus test), or 0, 25, 50 or 75 mg/kg (liver micronucleus test) showed that the chemical did not increase micronuclei frequency in the bone marrow. However, the result was positive in hepatocyte cells (REACH Dossier, 2011). The chemical induced DNA modifications (8-hydroxydeoxyguanosine and 8-aminodeoxyguanosine) in the bone marrow and liver but not in the kidney. DNA strand breaks were induced in SD rat livers, but not in lung, kidney, bone marrow or brain of rats treated in vivo. The chemical also induced micronuclei in the hepatocytes of SD rats but was negative in CD-1 mouse, F1 mouse and SD rat bone-marrow cells (IARC, 1987).

There is sufficient evidence to classify the chemical as possibly causing mutagenic effects.

Carcinogenicity

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

The chemical is also listed by the International Agency for Research on Cancer as a 'Group 2B carcinogen: possibly carcinogenic to humans' (IARC 1987) and 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 (NTPRoC 2011).

The chemical was tested for carcinogenicity in male SD rats by oral gavage at 0 or 40 mg/kg-bw per day, for 16 weeks to 22 animals (The Government of Canada 2010). Malignant liver tumours were found in all treatment group animals. No malignant tumours were found in the control animals; a benign tumour was reported in one animal from this group.

In an inhalation exposure study, rats were administered the chemical at 0, 98 or 755 mg/m^3 for 7 hours /day, for 2 days, 10 days, 1, 3 or 6 months. All rats exposed to 755 mg/m^3 for 6 months developed multiple hepatocellular carcinomas, whereas at 3 months, while no tumours were observed, hyperplastic changes in the liver were reported. No tumours were reported in the group that received 98 mg/m^3 (NOAEL).

Reproductive and developmental toxicity

In an IARC reported study, SD rats were treated intraperitoneally with 170 mg/kg bw/day of the chemical on days 1 to 15 of gestation (IARC 1987). While reduced pre- and post-implantation survival and reduced foetal body weight or length were reported, no signs of maternal toxicity or teratogenicity were reported to be observed.

Risk Characterisation

Critical Health Effects

The critical effects for risk characterisation are carcinogenicity and potential mutagenic effects. The chemical is expected to have acute oral and inhalation toxicity.

Public Risk Characterisation

The European Union and New Zealand have restricted the use of this chemical in cosmetics, while the US has restricted its use as a component of coatings in food packaging. There are no reported cosmetic or domestic uses of this chemical.

The Government of Canada (2010) has reported that a significant source of exposure to the chemical is from cigarette smoke. Exposure to the chemical in this way is not due to its use as an industrial chemical, and is therefore outside the scope of this risk assessment.

Occupational Risk Characterisation

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. Notably, as the liquid is highly volatile, the risks of inhalation and acute toxicity via inhalation would be considered high, if current occupational exposure standards for the chemical are not met. The chemical should be appropriately classified and labelled to ensure that a person conducting a business, or an employee at a workplace, has adequate information to determine appropriate controls.

NICNAS Recommendation

Current risk management measures are considered adequate for the protection of public and workers' health and safety, provided that all requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory. No further assessment is required.

Regulatory Control

Public Health

Considering the available information to indicate low public exposure from this chemical, no regulatory controls are recommended.

Occupational Health and Safety

adopted GHS as below. This does not consider classification of physical hazards and environmental hazards.

	Approved Criteria (HSIS)[a]	*GHS Classification*
Acute Toxicity	Harmful if swallowed (Xn; R22)* Harmful by inhalation (Xn; R20)*	Harmful if swallowed - Cat. 4 (H302) Harmful if inhaled - Cat. 4 (H332)
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

Products containing the chemical should be used according to label instructions.

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 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 and physicochemical (physical) hazards) of chemicals 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.

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